Adams & Stashak's Lameness in Horses

SEVENTH 7 EDITION

EDITED BY

Gary M. Baxter







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To my family for their understanding and encouragement

and

To all of the faculty, residents, interns, students, staff, and referring veterinarians that I have worked with through the years at both Colorado State University and the University of Georgia

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PREFACE TO THE SEVENTH EDITION

Welcome to the seventh edition of *Adams and Stashak's Lameness in Horses*. The seventh edition is a full-color edition, and therefore a major effort was made to include new color images and illustrations. The goal was to have every image meet the standard of digital quality and clearly illustrate what the author had intended. Another major goal was to update existing material and add new information without expanding the size of the book. This required reorganization, consolidation, and addition and subtraction of material throughout the text. You will notice that the previous Chapters 2 and 3 were consolidated into a single chapter to permit the addition of more material on objective lameness diagnosis by Dr. Kevin Keegan. The large Chapter 5 in the sixth edition was condensed and divided into two individual chapters that focused on lameness problems of the distal vs. proximal aspects of the limbs. Additionally, the references throughout the book were grouped at the end of each major subheading instead of listed after each topic to prevent repetition of the references within chapters.

Also available to the reader are short "how to" video clips that demonstrate a variety of different physical examination (palpation, hoof testing, flexion tests) and perineural and intrasynovial injection techniques. The clips will be available on a companion website intended to complement the text within the book. The goal is for the reader to use the video clips to better interpret and understand the text by being able to clearly see the procedure being performed on a live horse. The select perineural and intrasynovial injection video clips contain extensive anatomic details inserted directly into the live demonstrations to better illustrate the techniques. Important anatomic landmarks are clearly labeled on the videos for further clarity.

I wish to thank all of the authors that have contributed to the seventh edition as well as those that have contributed in the past and for the medical illustrators that helped create the new images and videos for the book. I would also like to thank all of the horses, clients, and veterinarians that have provided the case material, knowledge, and experiences that have been included within this text. I hope that the seventh edition continues to exemplify the excellent tradition that has been characteristic of the previous editions of Adams and Stashak's Lameness in Horses.

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PREFACE TO THE SIXTH EDITION

Welcome to the sixth edition of *Adams and Stashak's Lameness in Horses*. When Dr. Stashak approached me about being an editor for the new edition, I failed to realize the complexity of the endeavor. However, I have tried to modify the book with the specific goal of providing the most current information as concisely as possible. You will notice that Dr. Stashak's name has been added to the book title to reflect his numerous contributions to this text over the last few editions.

The primary objectives of the sixth edition were to update existing information and add new information without expanding the size of the book. This required reorganization, consolidation, and deletion of existing material in some cases. Expansive text on surgical procedures was condensed or eliminated in the sixth edition to focus on lameness and not surgery in horses.

You will notice that only Chapters 1–5 and 12 are similar in content to previous chapters in the fifth edition. However, Chapter 4 (Diagnostic Procedures) has been expanded considerably to reflect the advances that have been made in this important area over the last several years. Chapters 6–11 are new, although much of the information from the fifth edition has been reorganized into a different format within these chapters. Chapter 6 was added because of the growing importance of the axial skeleton in lameness and poor performance, especially in certain occupations. Chapters 7 and 8 focus on the principles of musculoskeletal diseases and treatments, respectively, and hopefully permit the reader to better understand these basic disease processes as well as the multitude of treatment options that are available for the numerous disease conditions covered elsewhere in the text. Chapter 9 contains a wealth of information from experienced equine veterinarians regarding lameness conditions unique to a horse's specific sport. Knowing these specific occupation-related conditions can be extremely helpful in lameness diagnosis. Chapter 10 is a "catch all" for many conditions and situations that do not fit neatly within another chapter yet are important aspects of the musculoskeletal system in the horse. Examples include prepurchase examinations, saddle fit, headshaking, and assessment of the neurologic horse. Chapter 11 discusses the unique features of the musculoskeletal system in the growing horse and serves to remind us of the numerous differences between the immature and mature horse with respect to lameness.

A major effort was also made to include as many new color images and illustrations within the sixth edition as possible. Several older anatomical illustrations were converted to color, but many of the black and white illustrations were retained because they remain excellent examples. Conventional radiographs were replaced with digital images whenever possible due to their improved quality and reproducibility. The goal was to have every image clearly illustrate what the author had intended.

An instructional DVD titled The "How to" Guide for Equine Lameness Evaluation complements the sixth edition. Its primary purpose is to demonstrate physical examination procedures, manipulative tests, and other diagnostic techniques that are somewhat unique to the horse. Perineural and intrasynovial anesthetic techniques are illustrated both with still images and live demonstrations. Examples of lameness cases were included, so the observer could translate written text to the live horse regarding what to look for when evaluating a lame horse. Specific examples of uncommon musculoskeletal problems were also included with the idea that once you see one, you will never forget it. Finally, an example of how to evaluate lameness using objective data was included to make readers aware of the possible future of lameness diagnosis in the horse.

I wish to thank all who contributed to the text in any way, including the numerous horses, clients, and veterinarians who have provided me with the case material, knowledge, and experiences that have been included within this text. I hope that the sixth edition continues in the rich tradition of excellence that has been provided by previous editions of *Adams' Lameness in Horses*. However, as the specialty of equine lameness continues to evolve, ideas to further improve the text are always welcomed. Thank you.

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PREFACE TO THE FIFTH EDITION

First and foremost, I want to extend my sincere thanks to the veterinary profession, veterinary students, students in related equine science programs, paraprofessionals in the equine industry, and horse owners throughout the world for their wide acceptance of the fourth edition of *Adams' Lameness in Horses*. The many favorable comments I received throughout the years have, to a large degree, provided me with the impetus to embark on the much-needed revision of the fourth edition. That being said, it pleases me to provide the veterinary profession and persons in equine-related fields with the extensively revised fifth edition of *Adams' Lameness in Horses*. As with the fourth edition, the changes are substantial, including the addition of new authors, the reorganization of material, and the reduction in the number of Chapters from 14 to 9. As with the other editions, the fifth edition is designed to appeal to a wide audience in equine-related fields.

Chapter 1 has been revised to provide the reader with an updated version of the functional anatomy of the equine locomotor system. The latest information regarding the dermal microcirculation of the foot and the anatomy of various joint capsules and their distribution has been added with detailed illustrations to support the discussion. As usual, Dr. Kainer's attention to detail provides a complete reference for the various regions of the musculoskeletal system. I would like to thank Dr. Robert Bowker for his contributions to this chapter.

Chapter 2 has changed considerably and covers a discussion of conformation and locomotion. The part on conformation has been extensively revised and updated with as much reference material as possible in hopes of providing objective data from which to draw conclusions. Additionally, the discussion of normal movement, movement abnormalities, and factors that affect movement, which expands on the material from Chapter 13, "Natural and Artificial Gaits," from the fourth edition, has also been included. Cherry Hill's coauthorship has provided much needed insight from a certified (carded by the U.S. breed associations) equine judge's standpoint. Cherry's background as a professional horse trainer and instructor has also added a practical perspective that I believe will appeal to veterinarians and horsemen alike.

Chapter 3 is presented in the same format as in the previous edition, with the addition of new material to make it as current as possible. Most of the anecdotal material has been removed except where personal experience was interjected to provide another perspective. Many new illustrations have been added to facilitate the discussion.

Chapter 4, the imaging chapter, has been completely updated and includes two new parts, one on ultrasound and one on nuclear medicine. The discussion of these two imaging modalities, used extensively for lameness diagnosis, has greatly increased the amount of material presented. Chapter 4 is divided into three parts. Part I, authored by Dr. Richard Park, provides an updated discussion of radiography in the diagnosis of equine lameness. This is followed by Part II, a comprehensive discussion by Dr. Robert Wrigley on the usefulness of ultrasound in lameness diagnosis. This part's many illustrations provide a useful and clear understanding of the anatomy being imaged. In Part III, Dr. Phillip Steyn provides a comprehensive discussion and presentation of illustrations on the value of nuclear medicine in the diagnosis of equine lameness. I would like to thank Dr. Richard Park for his leadership role in the development of this chapter.

Chapter 5 has also been completely updated with the addition of a new first author, Dr. Kate Savage, with Dr. Lewis acting as second author. This chapter provides the most current information regarding the role that nutrition plays in musculoskeletal development and disease.

Chapter 6 has also been completely revised and updated. With the departure of my colleague, Dr. Simon Turner, from the clinical arena to research, Dr. Gary Baxter has taken over as the first author of this chapter, with Dr. Turner serving as second author. A significant addition to this chapter is a comprehensive and practical discussion of the emergency ("first aid") management of equine fracture patients for transport and/or treatment. Many illustrations have been added to support the discussion.

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Chapter 7 has been extensively revised by Dr. Wayne McIlwraith. The addition of much research material to this chapter provides the reader with the most current information on the etiopathogenesis, diagnosis, and treatment of the various causes of joint disease and related structures. Many new illustrations have been added to augment the discussion of these various entities.

Chapter 8 has been extensively revised and greatly expanded, with the addition of new diseases. Dr. Alicia Bertone has updated discussion on the diseases associated with the fetlock region, including the metacarpus and carpus. Dr. Ken Sullins has updated discussion on the diseases of the hindlimb up to the coxofemeral joint. Dr. Dean Hendrickson has revised discussion on the diseases associated with the pelvis, back, and axial skeleton. The addition of these authors has greatly improved my ability to provide the reader with the most comprehensive and current discussion of the various diseases that cause lameness. As with the fourth edition, Chapter 8 concludes with discussion of "wobbler syndrome" and the various diseases of the spinal cord that can produce locomotor disorders that appear similar clinically. Dr. Alan Nixon has completely revised this section and, of note, has added a comprehensive discussion of the most current information on the diagnosis and treatment of equine protozoal myeloencephalitis (EPM).

Chapter 9 has been completely reorganized and updated and is presented in an entirely different format from that presented in the fourth edition. It incorporates information from Chapters 10 to 12 of the fourth edition. The addition of Cherry Hill, Richard Klimesh, and Gene Ovnicek as co-authors has greatly improved the presentation of this material, which should make this chapter most useful to all who read it. (Chapter 14, "Methods of Therapy," from the fourth edition has been eliminated, since most of this material is covered throughout the fifth edition for specific lesions or diseases and because many other texts cover the topic more completely than I possibly could in one chapter.)

With the expansion of the literature pertaining to lameness diagnosis and the recognition of new diseases, the reader will soon recognize that the reference lists have expanded in all portions of the text. In all cases the authors tried to include reference material from journals and text sources other than those of English-speaking countries. This was difficult at times, since frequently only summaries and abstracts were written in English.

I am grateful and indebted to Mark Goldstein for his untiring efforts and the many tasks he performed to make the fifth edition possible. Mark scanned the entire fourth edition onto computer. This unfortunately had to be done because the majority of the fourth edition text was lost in the archives of computer services. Following scanning, Mark proofread the material word for word, including checking superscripts and reference formatting. This had to be done, since the accuracy of the scanner at that time was only about 70%. Mark also did all the literature searches for the entire text and copied and organized the literature for distribution to contributing authors. Additionally, Mark combined new and old references for the fifth edition and added their numbered callouts in the text. Mark, thanks for your loyal and untiring effort; without you it would have been very difficult to complete the fifth edition.

The addition of numerous illustrations and photographs represents a tremendous time commitment and effort on behalf of the Computer-Assisted Teaching Service laboratory at Colorado State University. For the majority of the new illustrations, I am deeply indebted to Jenger Smith for her skill and expertise in producing these fine illustrations for the fifth edition. Her desire to produce the best possible image and her untiring efforts are most appreciated. Additionally, I am grateful to Gale Mueller from Visible Productions for the excellent illustrations she made for Chapters 1, 3, and 7.

I am grateful to my colleagues, Drs. Baxter, Hendrickson, McIlwraith, and Trotter, including referring practitioners, for allowing me the courtesy of using some of their case material as examples. I also acknowledge the contribution of my colleagues and the surgical residents who have contributed to the care and treatment of some of the cases presented in this text. A special thanks is extended to the many practitioners who have referred cases that have been used in this text. Without their continued support, the accumulation of the case material would not have been possible. Additionally, I am grateful to the technicians who provided support in the care of these patients.

Dana Battaglia, managing editor, and the entire staff at Lippincott Williams & Wilkins have been most patient and helpful in the preparation of the fifth edition. I am grateful for their support and guidance. I also wish to thank Carroll Cann, former veterinary editor for Lippincott Williams & Wilkins, who provided early encouragement for this edition.

I hope the new fifth edition meets all the expectations and needs of those who read it. As always, I look forward to your cooperation in making corrections and suggested revisions for future editions.

Ft. Collins, CO TED S. STASHAK

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PREFACE TO THE FOURTH EDITION

When I was contacted by Mr. George Mundorff, executive editor for Lea & Febiger, regarding the possibility of revising the third edition of "Lameness in Horses" by Dr. O. R. Adams, I was excited but naive to the task at hand. Dr. Adams had, in his previous three editions, established the state of the art of lameness diagnosis and treatment, presenting it in a unique manner that appealed to veterinarians, horse owners and trainers, and farriers. Without a doubt, he defined and directly influenced the course of this subject more than any other individual during this time. I was truly fortunate to train under him during my internship and surgical residency at Colorado State University. His never-ending thirst for knowledge, his humor, his friendship, and his love of the veterinary profession have inspired me throughout this endeavor. I only hope that I have served his memory well and that he would be proud of this fourth edition.

After considerable discussion with Lea & Febiger and the assurance of Mrs. Nancy Adams, Dr. Adams' widow, I embarked on the revision with some basic changes in format in mind. These included the addition of new authors, changes in chapter sequence and presentation, the addition of new chapters and deletion of some old ones, and the transition from a monograph to a reference text. Because I wanted the fourth edition to represent the school where Dr. Adams attended and taught, I selected mostly authors from our faculty on the basis of their expertise and their ability to provide a broad base of opinion for the reader.

With the idea of approaching the discussion of lameness as one would approach a lameness examination itself, I changed the sequence of presentation. Using the newest accepted nomenclature, Chapter 1 deals with the functional anatomy of the equine locomotor system and represents a complete revision of Chapter 2 in the previous edition. Dr. Kainer starts with the forelimb, advancing from the foot up the limb, describing the regional anatomy of each site. The hindlimb is covered in similar fashion. The nomenclature may be confusing initially to older graduates of American veterinary schools, but recent graduates as well as foreign veterinarians will be well versed in this terminology. We felt it was time to make this transition since the new nomenclature has been in use for at least 4 years. (Older terms are included parenthetically.)

Following a format similar to the previous edition, Chapter 2 deals with the relationship between conformation and lameness. I have eliminated "The Examination for Soundness," which was Chapter 3 in the previous edition, because it discussed many topics unrelated to lameness and, simply, because the subject of soundness is so comprehensive it could be covered in a separate text. The present Chapter 3 deals with the diagnosis of lameness. After defining lameness and establishing how to determine which limb is lame, the description of the physical examination begins at the foot of the forelimb and proceeds upward. Emphasis is placed on recognition of problems peculiar to the region examined. Following this is a description and illustration of perineural and intrasynovial anesthesia.

The next logical step in the diagnosis of lameness is radiology, which is discussed in Chapter 4. This chapter is comprehensive; nothing like it has been published elsewhere. The format of the text and illustrations should answer any question the reader may have regarding the techniques for taking radiographs and interpreting them. The artwork beautifully illustrates the different structures seen on various radiographic views, and the illustrations are labeled so that anatomic sites are easily identified.

Chapters 5–7 are new. Discussing the role of nutrition in musculoskeletal development and disease, Chapter 5 illustrates a unique approach not used elsewhere. Dr. Lewis provides a comprehensive review of specific nutritional disorders, their causes, and their treatment for all phases of growth and development in the foal, during pregnancy and lactation in the mare, and during maintenance of the working horse. This information will benefit both the horseman and the veterinarian. Chapter 6, by Dr. Turner, starts with a brief review of endochondral ossification and then discusses the diseases associated with bones and muscles and their treatment. In Chapter 7, Dr. McIlwraith describes the developmental anatomy of joints and related structures, disease processes, clinical signs, and treatments. Both of

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these chapters present in-depth reviews, with major emphasis on the pathogenesis and pathobiology of the diseases. They are heavily referenced and will be of major interest to the veterinary profession.

Representing a complete revision of Chapter 8, "Lameness" updates the reader on new diseases as well as new findings and treatment for previously recognized entities. Unlike past editions, this material is heavily referenced. Information regarding the prevalence of the disease within various breeds according to sex and age introduces each subject. The format of the chapter has been changed to start with diseases relating to the foot region and then proceeding upward anatomically, consistent with the way most equine practitioners approach a systematic examination. Specific diseases of each region are discussed separately. This chapter, though referenced heavily and written technically, should be of interest to the horseman as well as the veterinary profession. I am particularly grateful to Dr. Alan Nixon for his thorough and comprehensive review of the diagnosis and treatment of the "wobbler's syndrome" in horses. His presentation is clear and well illustrated, giving the reader the confidence to differentiate among the diseases that cause this syndrome.

Chapters 9–12 were written primarily for the horseman and farrier, though they will also be of interest to the veterinarian, particularly the equine practitioner. I have updated these chapters with new information, as well as listing what the horseman should look for when the horse is properly trimmed and shod. Chapter 13, "Natural and Artificial Gaits," is essentially unchanged. Chapter 14, "Methods of Therapy," has been updated and includes an extensive revision of different methods of external coaptation. This chapter is primarily directed toward the veterinary profession, though the horse owner will obtain insight into why different treatments are selected.

With the explosion of literature pertaining to musculoskeletal disease in the horse and the demands put on authors and editors alike, it became obvious that a transition from a monograph to a reference text was timely. To this end the authors have attempted to provide the latest information. As with any large text, however, authors and editors alike feel somewhat frustrated because at the time of publication some of this information will be out of date. With few exceptions, we stopped referencing material published in 1985. Occasionally publications in 1985 changed the presentation of the materials so much that it could not be denied and therefore was included.

I am grateful to Dr. Robert Kainer, professor of anatomy and author of the first chapter, for taking the time to review and advise me on the nomenclature used in this book. A special thanks is extended to Dr. A. S. Turner for his review and comments on Chapter 8. The fine contributions of all the authors are sincerely appreciated. I want to thank Dr. Robert Perce (California) and Mr. Richard Klimesh (farrier, Colorado) for their advice on the chapters dealing with trimming and shoeing horses.

The addition of many new illustrations and photographs represents a tremendous time commitment and effort on behalf of the Office of Biomedical Media at Colorado State University. For the illustrations, I am indebted to Mr. Tom McCracken and Mr. John Dougherty for their expertise and the cooperation they have given me. For the photographs I am grateful to Mr. Al Kilminster and Mr. David Clack, for their expertise, cooperation, and commitment to excellence. For the design of the book cover, I thank Mr. Dave Carlson.

Most of the manuscript was typed by Mrs. Helen Acevedo. Her cooperation and patience with the many revisions necessary to complete this text are gratefully appreciated.

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Mr. Christian C. Febiger Spahr Jr., veterinary editor; Mr. George Mundorff, executive editor; Mr. Tom Colaiezzi, production manager; Ms. Constance Marino; Mrs. Dorothy Di Rienzi, manager of copy editors; and the entire staff at Lea & Febiger have been most helpful in the preparation of this book. I am grateful for their support and guidance.

I hope this book will be useful to all who read it. I hope to receive your cooperation in making corrections and suggested additions for further revisions.

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COMMON TERMINOLOGIES AND ABBREVIATIONS

Terminology	Abbreviations
Distal or third phalanx	P3; coffin bone
Middle or second phalanx	P2
Proximal or first phalanx	P1
Metacarpus/metatarsus	MC/MT or MC3/MT3; cannon bone
Second and fourth metacarpal/metatarsal bones	MC2, MC4, MT2, MT4; splint bones
Proximal sesamoid bones	PSB
Distal sesamoidean ligaments	DSL
Distal sesamoidean impar ligament	DSIL
Collateral suspensory ligaments of navicular bone	CSLs
Collateral ligaments	CLs
Deep digital flexor tendon	DDFT or DDF
Superficial digital flexor tendon	SDFT or DDF
Accessory ligament of deep digital flexor tendon	ALDDFT, ICL or inferior check
Accessory ligament of superficial digital flexor tendon	ALSDFT, SCL, or superior check
Digital flexor tendon sheath	DFTS
Common digital extensor tendon	CDET
Long digital extensor tendon	LoDET
Lateral digital extensor tendon	LDET
Lateral digital flexor tendon	LDFT
Distal interphalangeal joint	DIP joint or coffin joint
Proximal interphalangeal joint	PIP joint or pastern joint
Tarsometatarsal joint	TMT joint
Distal intertarsal joint	DIT joint
Proximal intertarsal joint	PIT joint
Tarsocrural joint	TC joint
Distal tarsal joints	DT joints
Metacarpo/metatarsophalangeal joint	MCP/MTP or fetlock joint
Medial femorotibial joint	MFT joint
Lateral femorotibial joint	LFT joint
Femoropatellar joint	FP joint
Scapulohumeral joint	SHJ or shoulder joint
Sacroiliac joint	SI joint
Computed tomography	CT
Magnetic resonance imaging	MRI
Increased radiopharmaceutical uptake	IRU
Ultrasound/Ultrasonography	US

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Cross sectional area	CSA
Osteochondrosis/osteochondritis dissecans	OC/OCD
Osteoarthritis	OA
Developmental orthopedic disease	DOD
Subchondral cystic lesion	SCL
Angular limb deformity	ALD
Suspensory ligament/suspensory ligament branches	SL/SLBs
Proximal suspensory desmitis	PSD
Medial patellar ligament	MPL
Middle patellar ligament	MidPL
Lateral patellar ligament	LPL
Upward fixation of patella	UFP
Medial patellar ligament desmotomy	MPD
Tarsal sheath	TS
Sustentaculum tali	ST
Peroneus tertius	PT
Lateral palmar	LP
Palmar digital	PD
Deep branch lateral plantar nerve	DBLPN
Mediolateral	ML
Dorsopalmar/plantar	DP
Nonsteroidal anti-inflammatory drug	NSAID
Hyaluronan or hyaluronic acid	HA
Polysulfated glycosaminoglycans	PSGAG; Adequan
Platelet-rich plasma	PRP
Interleukin receptor antagonist protein/autologous conditioned serum	IRAP/ACS
Mesenchymal stem cell	MSC
Triamcinolone	TA
Methyl prednisolone acetate	MPA or Depo-medrol
Dimethyl sulfoxide	DMSO
Diclofenac cream	Surpass
Extracorporeal shockwave treatment	ESWT or shockwave
Intra-articular	IA
Intravenous	IV
Intramuscular	IM
Regional limb perfusion	RLP

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ABOUT THE COMPANION WEBSITE

This book is accompanied by a companion website:

www.wiley.com/go/baxter/lameness



The website includes: Short "how to" video clips that demonstrate a variety of different physical examination (palpation, hoof testing, flexion tests) and perineural and intrasynovial injection techniques. The goal is for the reader to clearly see these procedures being performed on live horses. The select perineural and intrasynovial injection video clips contain extensive anatomic detail inserted directly into the live demonstrations to better illustrate the techniques. Important anatomic landmarks are emphasized and clearly labeled within the videos.

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CHAPTER 1

Functional Anatomy of the Equine Musculoskeletal System

Anna Dee Fails

ANATOMIC NOMENCLATURE AND USAGE

Veterinary medical anatomists have been using the Nomina Anatomica Veterinaria, created by the International Committee on Veterinary Gross Anatomical Nomenclature since 1968 to standardize the names of anatomical structures.[46] This chapter endeavors to use the most current, correct terms as outlined in that publication. Nonetheless, equine practitioners need to be equally fluent in older terminology, which is likely to be in wide usage among horse owners and equine professionals. This chapter will provide useful and common synonyms for many structures, along with their more technically correct terms.

Figure 1.1 provides the directional terms for veterinary anatomy that will be used in this chapter. With the exception of the ocular and oral cavity structures, the terms anterior, posterior, superior, and inferior are not applicable to quadrupeds.

THORACIC LIMB

Digit and Fetlock

The digit is composed of distal (third), middle (second), and proximal (first) phalanges and associated structures (Figure 1.2). The fetlock consists of the metacarpophalangeal (fetlock) joint and the structures surrounding it. Because the digits and fetlocks of the thoracic limb and the pelvic limb are similar in most respects, the following descriptions pertain to both limbs unless otherwise indicated. When referring to structures of the forelimb, the term "palmar" is used; this will obviously be replaced with "plantar" when referring to the hindlimb. Likewise, such terms as metacarpophalangeal and metatarsophalangeal are counterparts in fore- and hindlimbs, respectively.

Foot

The foot consists of the hoof and all it encloses: the connective tissue corium (dermis), digital cushion, distal phalanx (coffin bone), most of the cartilages of the distal phalanx, distal interphalangeal (coffin) joint, distal part of the middle phalanx (short pastern bone), distal sesamoid (navicular) bone, podotrochlear bursa (navicular bursa), several ligaments, tendons of insertion of the common digital extensor and deep digital flexor muscles, blood vessels, and nerves. Skin between the heels is also part of the foot.

Hoof Wall, Sole, and Frog

The hoof is continuous with the epidermis at the coronet, and the underlying corium of the hoof is likewise continuous with the dermis of the skin. The ground surface of the hoof comprises the sole, frog, heels, bars, and ground surface of the wall (Figure 1.3). The ground surface of the forefoot is normally larger and rounder than that of the hindfoot, reflecting the corresponding shape of the distal surface of the distal phalanx (coffin bone).

The hoof wall extends from the coronary band (also called the coronet), the transition between skin and hoof, distad to the ground. The surface of the wall is divided into the toe, medial and lateral quarters, and heels (Figures 1.3 and 1.4). From the toe, where it is thickest, the wall becomes progressively thinner and more elastic toward the heels, where it thickens again when it reflects to become the bars. Ranges for the angle of the toe between the dorsal surface of the hoof wall and the ground surface of the hoof vary widely.[1,16] In the ideal digit, the dorsal surface of the hoof wall and the dorsal surface of the pastern should be parallel, reflecting the axial alignment of the phalanges.

The vascular and densely innervated collagenous connective tissue deep to the hoof is the corium. The corium

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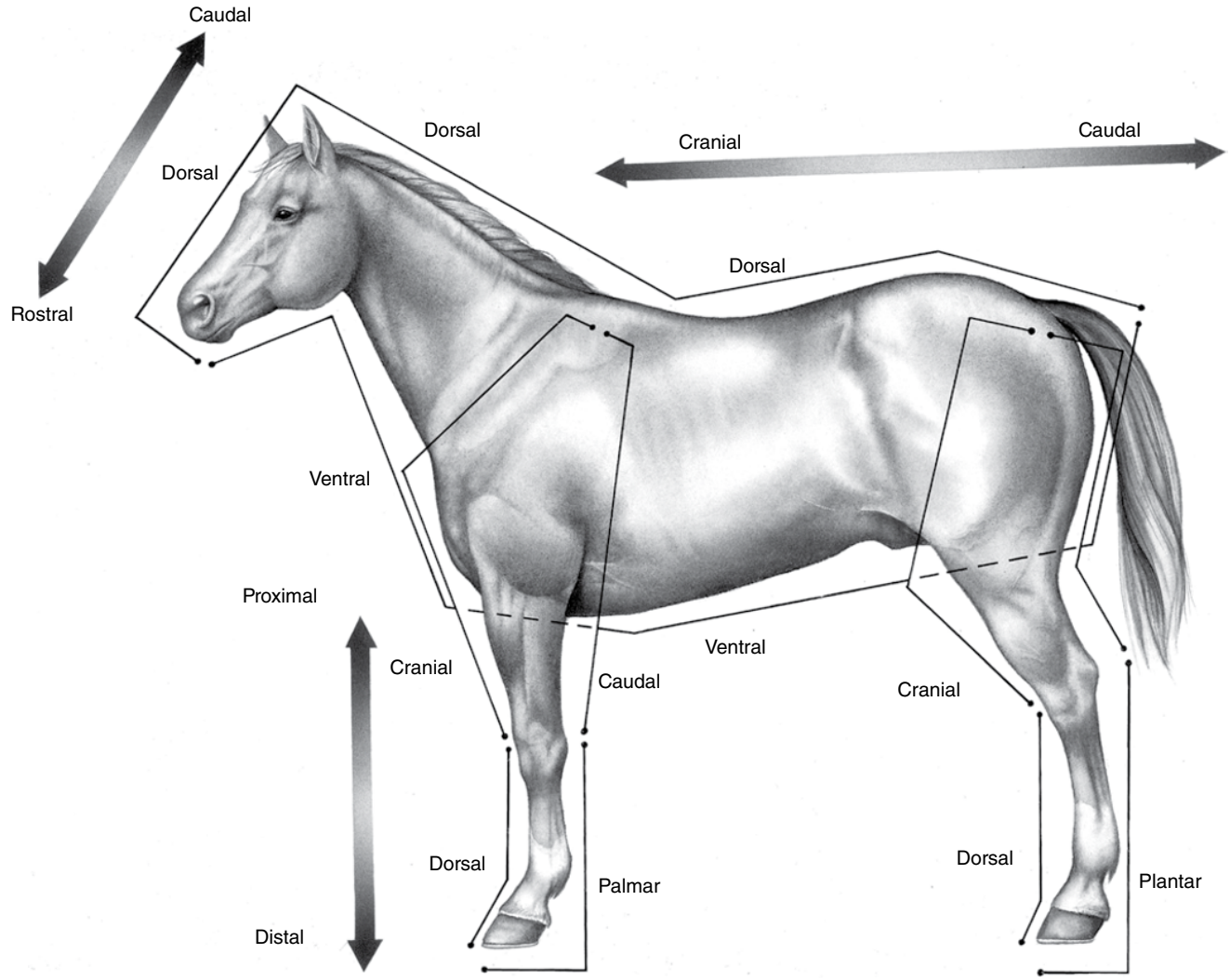


Figure 1.1. Positional and directional terms.

provides sensation, vascular supply, and attachment for the overlying stratified squamous epithelium that constitutes the hoof or ungual epidermis (*L. ungula*, hoof). Regions of the corium are named according to the parts of the hoof under which they are located: perioplic corium, coronary corium, laminar corium, corium of the frog (cuneate corium), and solar corium. Histologically, coronary corium gives rise to elongated, distally directed papillae. Laminar corium forms a series of sheets that interdigitate with epidermal laminae of the stratum internum of the hoof wall. Shorter papillae extend from the perioplic, solar, and cuneate coria.

In the coronary region, the deepest layer (the stratum basale) of the ungual epidermis is a single layer of proliferating keratinocytes lying upon and between long dermal papillae. Cellular division here pushes cells distad into the stratum medium of the hoof wall, forming the epidermis that undergoes cornification.[2] Nearly the entire hoof is composed of a thick layer of anucleate squamous keratinocytes.

For the most part, the keratinaceous tissues of the hoof are devoid of nerve endings; as a consequence it is the "insensitive" part of the foot. However, a few sensory nerve endings from nerves in the corium penetrate between cells of the deepest layer of the epidermis.

Three histological layers comprise the hoof wall: the stratum externum, stratum medium, and stratum internum (Figure 1.5). The superficial stratum externum, commonly called the periople, is a thin layer of horn extending distad from the coronet a variable distance; this thin, soft layer wears from the surface of the hoof wall so that it is present only on the bulbs of the heels and the proximal parts of the hoof wall. The bulk of the wall is the stratum medium consisting of cornified horn.[2] The stratum internum comprises the epidermal laminae.

Distal to the coronary sulcus (Figure 1.4), about 600 primary epidermal laminae of the stratum internum interleave with the primary dermal laminae of the laminar corium (Figures 1.6 and 1.7). Approximately 100 microscopic secondary laminae branch at an angle from each primary lamina, further binding the hoof and corium together (Figures 1.3–1.6). The epidermal laminae are routinely referred to as "insensitive," whereas the dermal laminae are called "sensitive." In the strictest sense, though, only the *keratinized* parts of the primary epidermal laminae are insensitive; the deepest layer of

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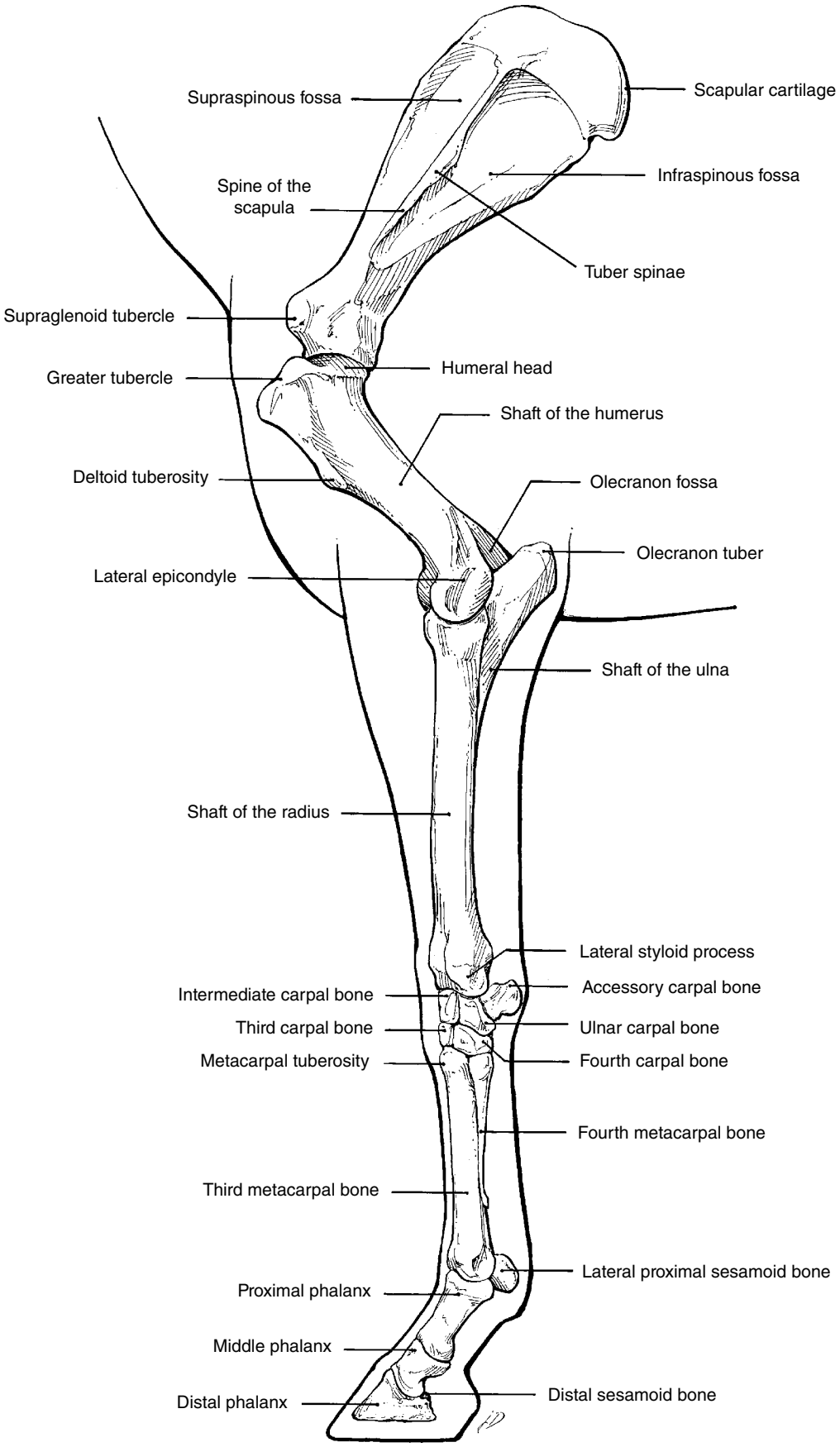


Figure 1.2. Bones of the left equine thoracic limb. Lateral view.

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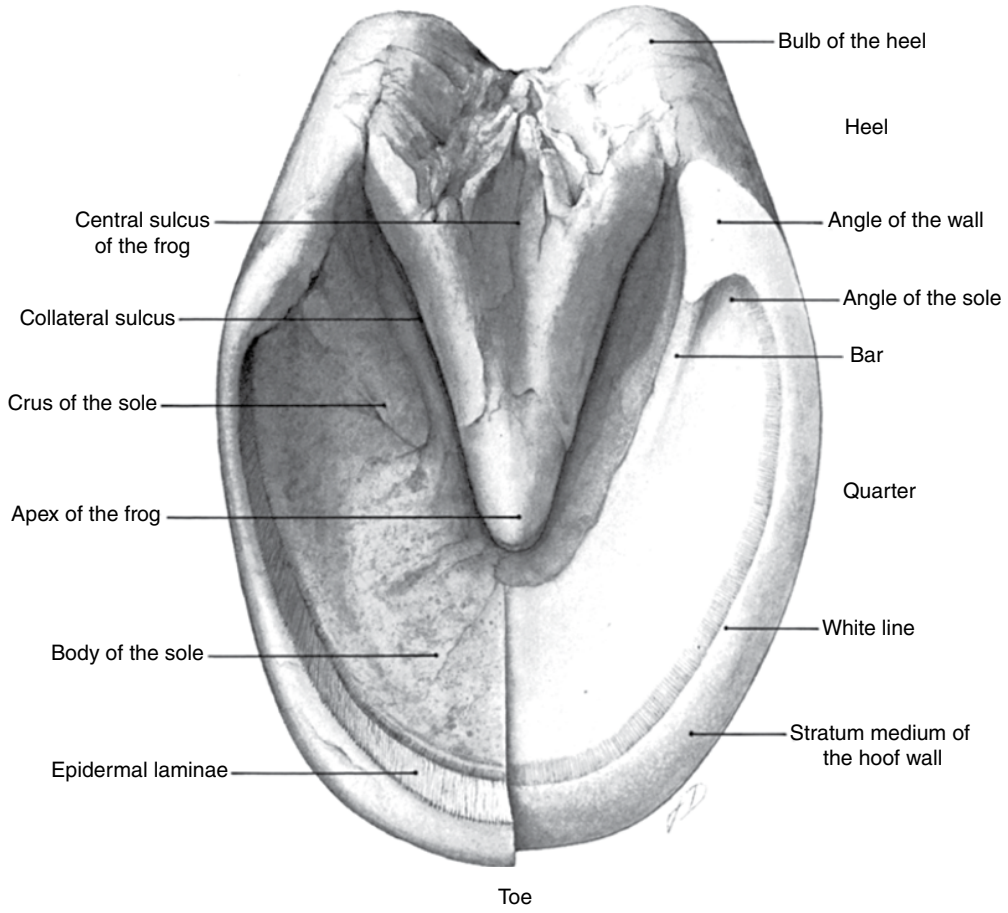


Figure 1.3. Topography of the solar surface of the hoof. The right half has been trimmed to emphasize the region of the white line.

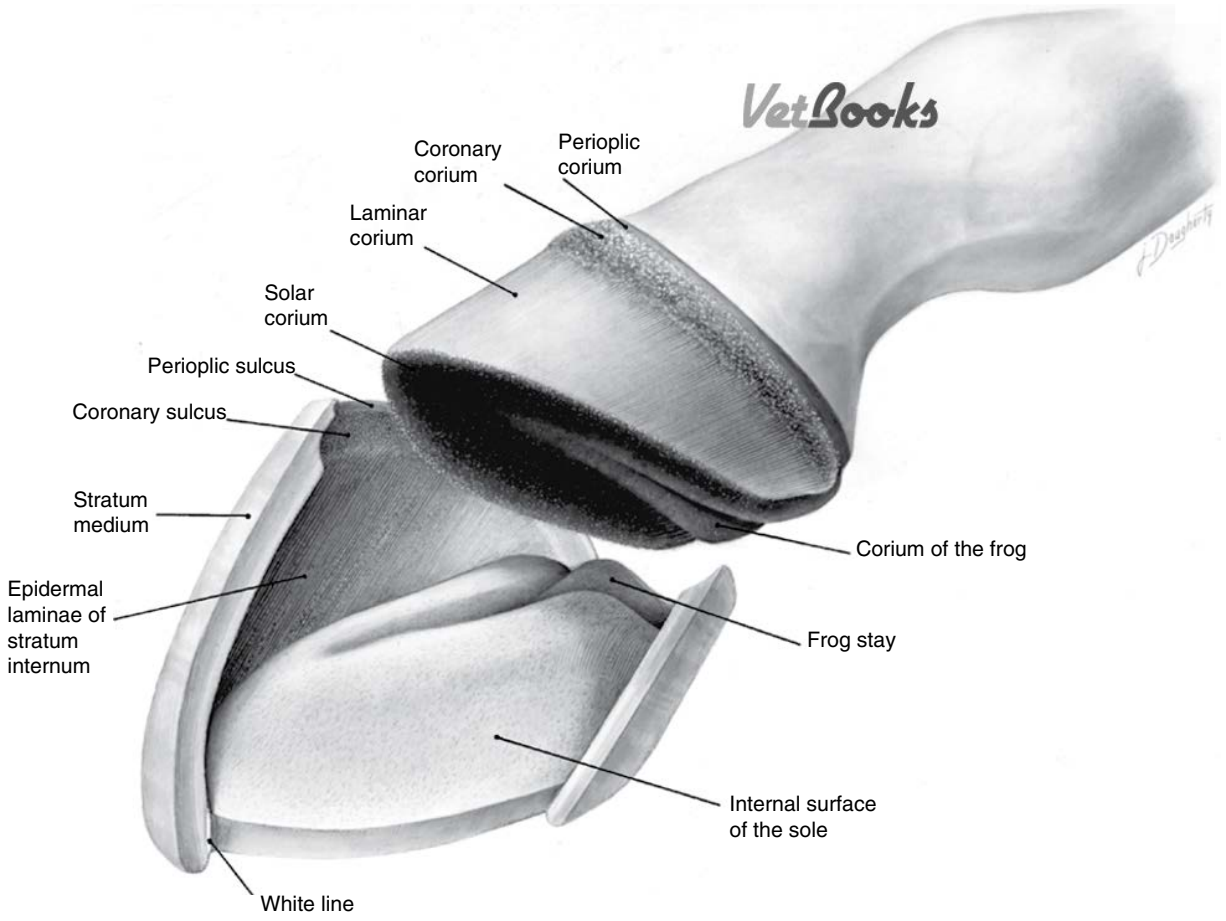


Figure 1.4. Dissected view of relationships of the hoof to underlying regions of the corium (dermis).

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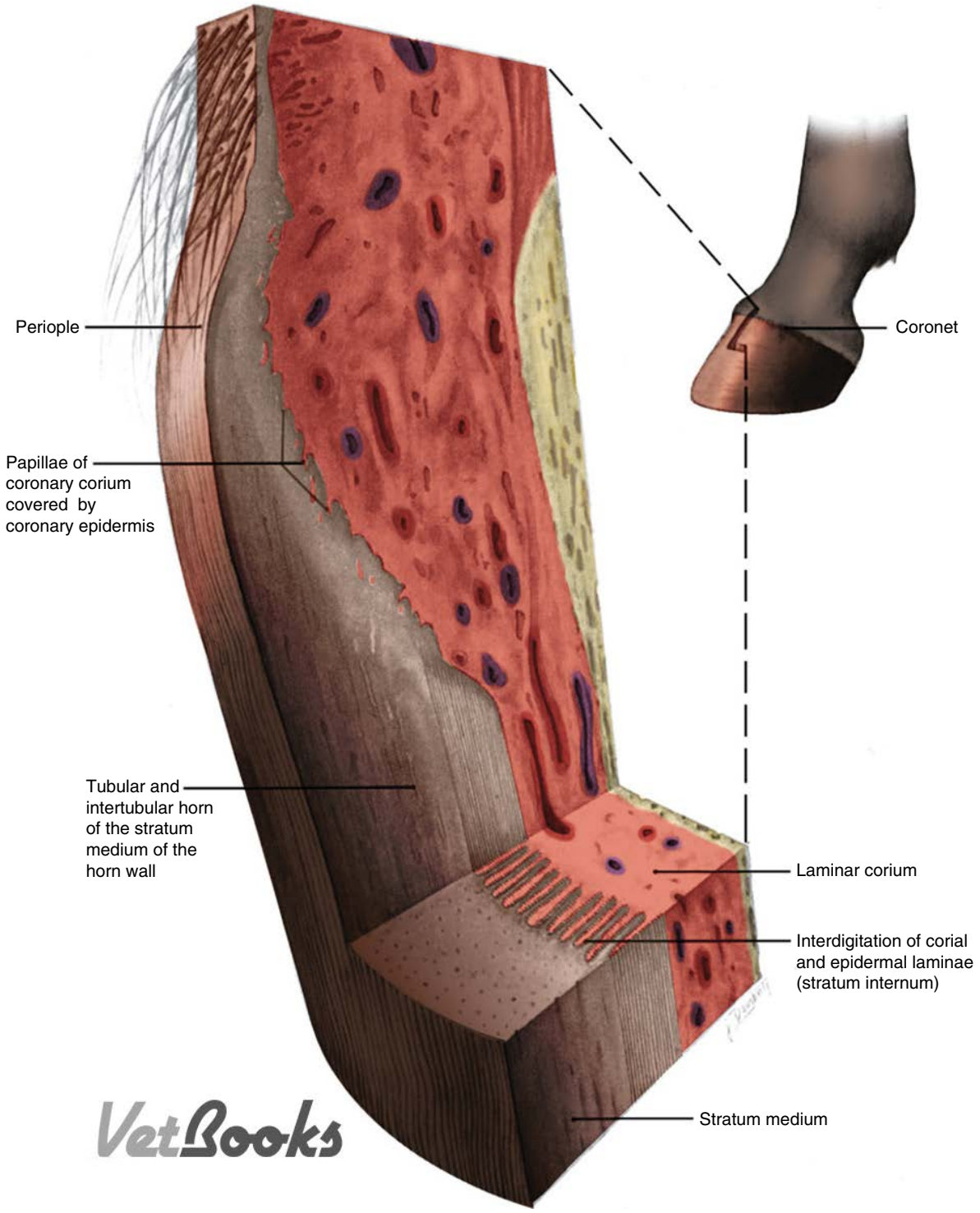


Figure 1.5. Three-dimensional dissection of coronary region of the hoof wall.

the epidermis, the stratum basale, including all of the secondary epidermal laminae, and the laminar corium are both innervated and therefore "sensitive."[42]

Growth of the hoof wall is primarily from the coronary epidermis toward the ground. Trauma or inflammation stimulates greater production of horn. Ultrastructural studies indicate that during growth of the hoof, primary epidermal laminae move past the secondary epidermal laminae by breaking and then reforming desmosomes between the two cell populations.[23] The relationship between the epidermal and dermal laminae plus the blending of the laminar corium with the periosteum of the distal phalanx suspend and support the bone, aiding in the dissipation of concussion and the movement of blood.

The growth of the wall progresses at the rate of approximately 6 mm per month, taking from 9 to

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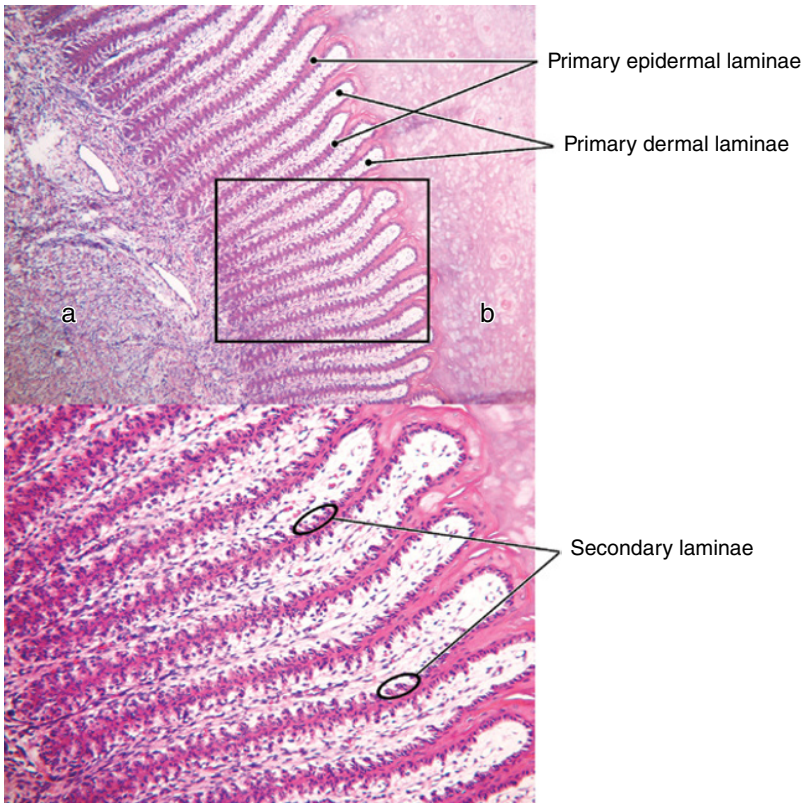


Figure 1.6. Photomicrograph of laminae of the equine hoof. In the top image, a indicates corium; b is the epidermis (hoof wall). Laminae extending from the corium (primary dermal laminae) are the so-called "sensitive laminae." Laminae extending from the epidermal portions of the hoof (primary epidermal laminae) are the "insensitive laminae." The box indicates the region enlarged in the lower image. Here, smaller interdigitating projections, the secondary laminae, can be seen arising perpendicular to the primary laminae.

12 months for hoof generated at the coronary band to reach the ground. The wall grows more slowly in cold and/or dry environments. The hoof wall grows evenly distal to the coronary epidermis so that the youngest portion of the wall in contact with the ground is at the heel (where it is shortest). Because this is the youngest part of the wall, it is also the most elastic, which allows it to accommodate heel expansion during concussion.

Stratum medium may be pigmented or nonpigmented. Contrary to popular belief, there is no difference in the stress–strain behavior or strength properties of pigmented versus nonpigmented equine hooves.[21] It has also been demonstrated that pigmentation has no effect on fracture toughness of hoof keratin.[3] On the other hand, water content of the hoof significantly affects its mechanical properties. In the natural hydration gradient in the hoof wall, the moisture content decreases from deep to superficial.[21] Very dry or extremely hydrated hoof wall is more likely to crack than normally hydrated hoof wall. A normally hydrated hoof is better able to absorb energy without mechanical failure.[4]

The slightly concave sole does not normally bear weight on its ground surface except near its junction with the white line, but it bears internal weight transmitted from the distal phalanx through the solar corium. In the unworn, untrimmed hoof wall, insensitive laminae can be seen on the internal surface of the wall where it makes contact with the ground (Figure 1.3). When the hoof is trimmed, the white line where the wall meets the sole is more clearly discerned. The sensitive corium is immediately internal to the white line that serves as a landmark for determining the proper position and angle for driving horseshoe nails.[14]

The frog (cuneus ungulae) is a wedge-shaped mass of keratinized stratified squamous epithelium made softer than other parts of the hoof by its greater water content.[21] Apocrine glands, spherical masses of tubules in the corium of the frog, deliver secretions to the surface of the frog.[25] The ground surface of the frog presents a pointed apex and central sulcus bordered by two crura. Paracuneal (collateral) sulci separate the crura of the frog from the bars and the sole. The palmar aspect of the frog blends into the bulbs of the heels. Compression of the frog during weight-bearing is transferred to the fibrofatty digital cushion deep to the heels; this force assists with movement of venous blood from the interior of the hoof capsule to the veins of the distal limb.

The corium blends with the periosteum of the distal phalanx, serving (particularly in the laminar region) to connect the hoof to the bone. The corium, the hoof's homolog to the dermis of skin, is composed of dense white fibrous connective tissue that is rich in elastic fibers, highly vascular, and well supplied with nerves. Corial arterial supply derives from numerous branches radiating outward from the terminal arch in small canals extending from the solar canal in the distal phalanx and from the dorsal and palmar branches of the distal phalanx, themselves branches of the digital arteries (Figure 1.8).

The coronary and perioplic coria and the stratum basale of the coronary and perioplic epidermis constitute

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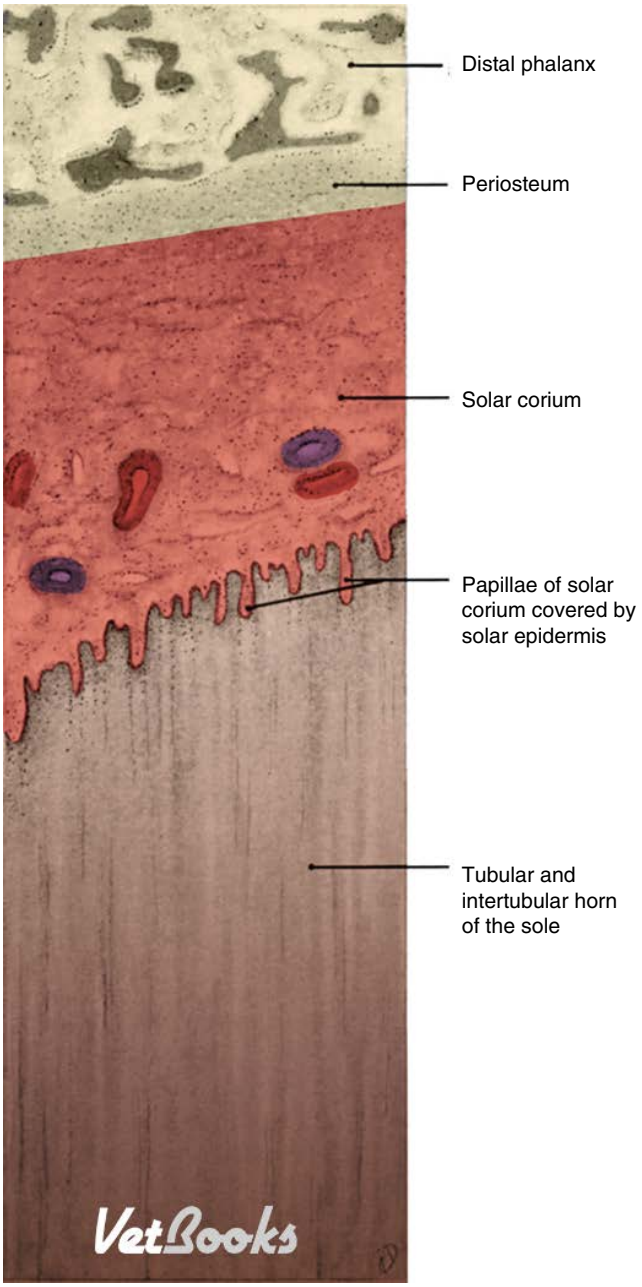


Figure 1.7. Histological relationships of periosteum, corium, and horn of the sole.

the coronary band (Figure 1.5). Deep to the coronary band, the subcutis is modified into the highly elastic coronary cushion. The coronary band and cushion form the bulging mass that fits into the coronary sulcus of the hoof (Figure 1.4). Part of the coronary venous plexus is within the coronary cushion. The plexus receives blood from the dorsal venous plexus in the laminar corium.

Internal Structures of the Foot

The "collateral" cartilages of the distal phalanx (often "lateral cartilages"; most correctly ungual cartilages) lie deep to the hoof and the skin, covered on their abaxial surfaces by the coronary venous plexus. They extend from each palmar process of the bone and project proximad to the coronary band of the hoof where they may be palpated (Figure 1.9). The cartilages are concave on their axial surfaces, convex on their abaxial surfaces, and thicker distally where they attach to the bone. Each cartilage is perforated in its palmar half by several foramina for the passage of veins connecting the palmar venous plexus with the coronary venous plexus.

Five ligaments stabilize each ungual cartilage (Figure 1.9):

1. A short, prominent ligament extends from the dorsal surface of the middle phalanx to the dorsal part of the cartilage.
2. A poorly defined elastic band extends from the side of the proximal phalanx to the proximal border of the cartilage and also detaches a branch to the digital cushion.
3. Several short fibers attach the distal part of the cartilage to the distal phalanx.
4. A ligament extends from the dorsal aspect of the cartilage to the termination of the tendon of insertion of the common digital extensor muscle. The dorsal part of each cartilage also serves as part of the distal attachment for the respective collateral ligament of the coffin joint.
5. An extension of the collateral sesamoidean ligament (CSL) attaches the end of the navicular bone to the cartilage of the distal phalanx.

Between the cartilages on the palmar side of the foot is the digital cushion, a highly modified subcutis consisting of a meshwork of collagen, elastic fibers, adipose tissue, and small masses of fibrocartilage (Figure 1.10). Only a few blood vessels ramify in the digital cushion. Dorsoproximally the digital cushion connects with the distal digital annular ligament. The apex of the wedge-shaped digital cushion is attached to the deep digital flexor tendon (DDFT) as the latter inserts on the solar surface of the distal phalanx. The base of the digital cushion bulges into the bulbs of the heels. The digital cushion serves an anticoncussive function.

As the DDFT courses to its insertion on the distal phalanx, it is bound down by the distal digital annular ligament, a sheet of deep fascia supporting the terminal part of the tendon and sweeping proximad to attach on each side of the proximal phalanx (Figure 1.11). The tendon passes over a complementary fibrocartilaginous plate on the proximal extremity of the palmar surface of the middle phalanx; this is the middle scutum (*L. shield*), which provides a smooth gliding surface for the tendon. Then the tendon gives off two small secondary attachments to the distopalmar surface of the second phalanx; these are part of the so-called T ligament (Figures 1.10 and 1.12).

Continuing toward its primary attachment on the flexor surface of the distal phalanx, the DDFT passes over the navicular (podotrochlear) bursa, interposed between the tendon and the fibrocartilaginous distal scutum covering the flexor surface of the navicular bone. From the exterior of the foot, the navicular bursa lies deep to the approximate middle third of the frog on a plane parallel to the coronet over the quarters of the hoof wall.

The proximal border of the navicular bone (distal sesamoid bone) possesses a groove containing foramina for the passage of small vessels and nerves. The distal border of the bone has a small, elongated facet that

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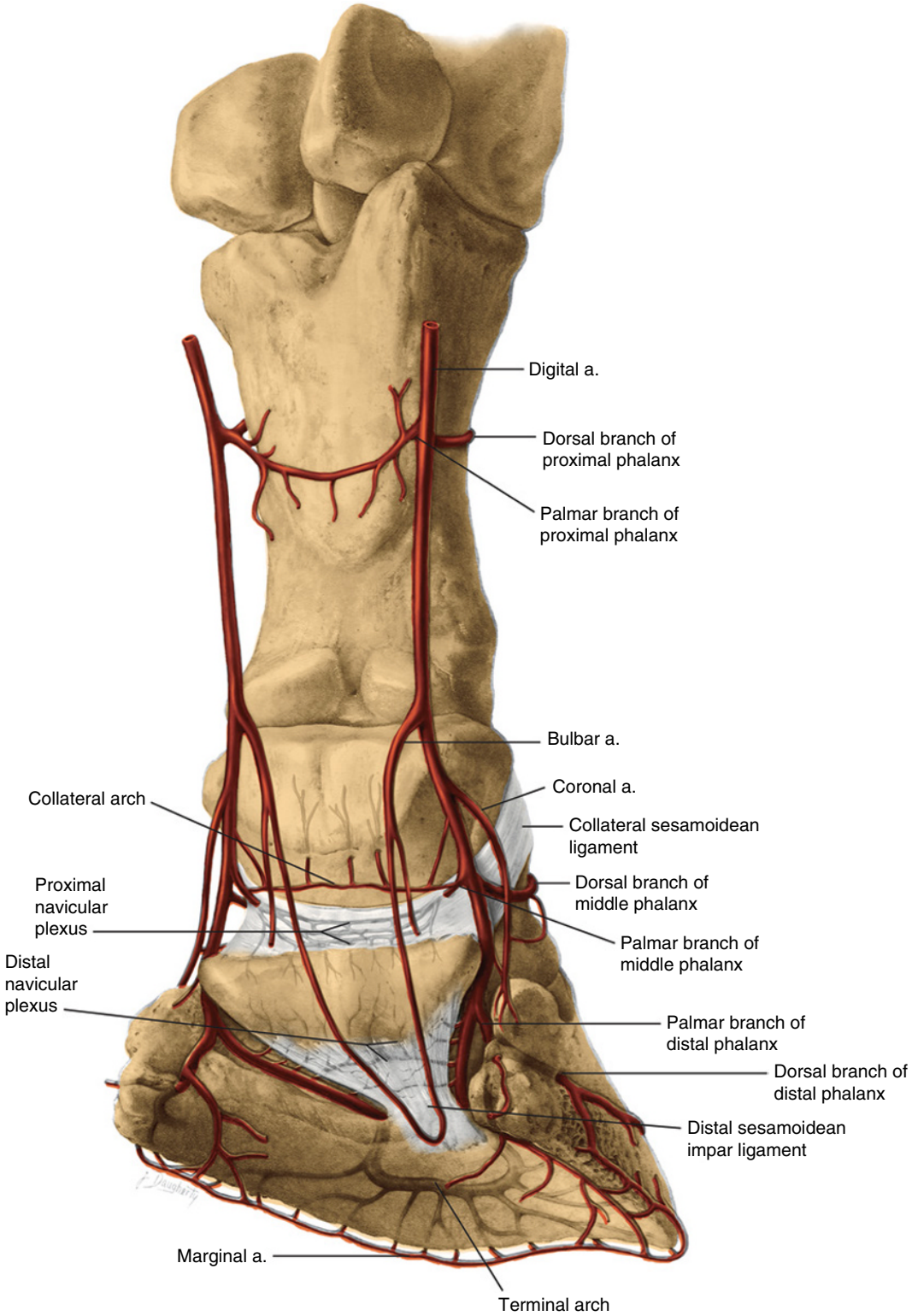


Figure 1.8. Arterial supply to the digit of the forelimb.

articulates with the distal phalanx. Several large, irregular fossae lie in an elongated depression palmar to that facet (Figure 1.13). The main articular surface of the navicular bone contacts the middle phalanx.

The navicular bone is supported in its position by three ligaments comprising the navicular suspensory apparatus. Medial and lateral collateral sesamoidean (suspensory navicular) ligaments arise from the distal end of the proximal phalanx (Figure 1.12). These sweep obliquely distad, each ligament crossing the pastern joint and attaching broadly along the proximal border of the navicular bone. Along this border, each ligament

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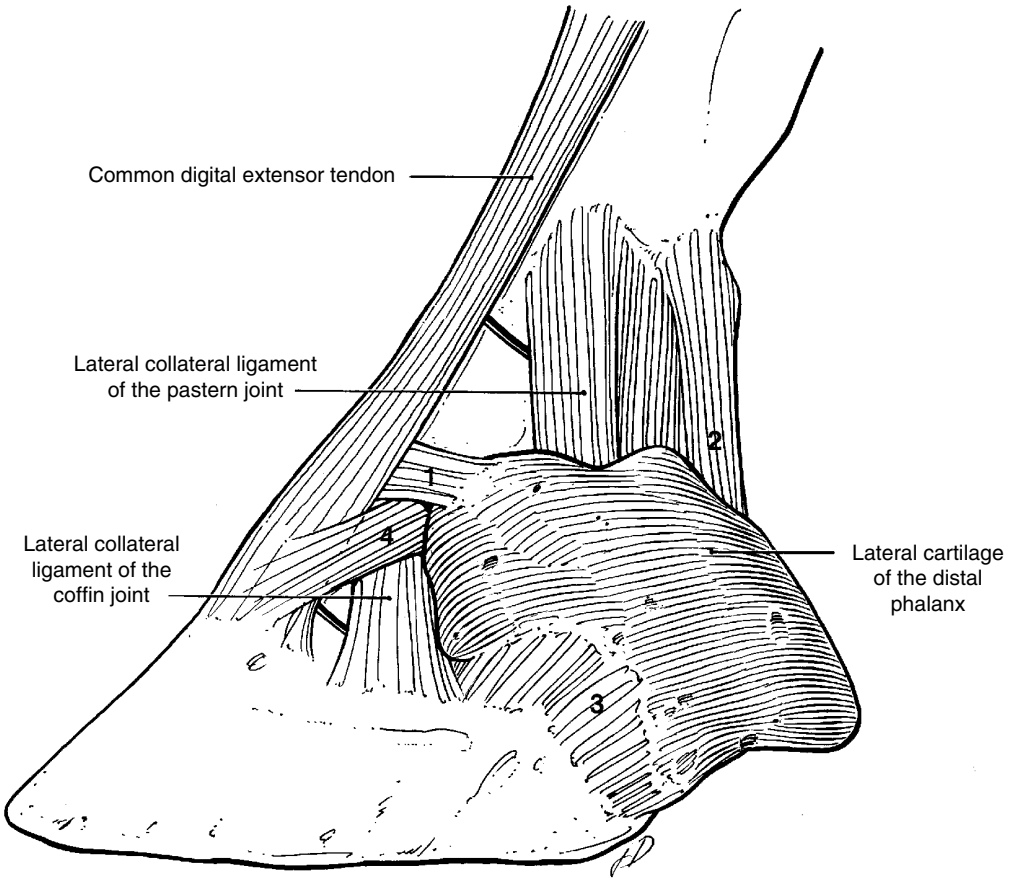


Figure 1.9. Four of the ligaments (1, 2, 3, and 4) that stabilize the cartilage of the distal phalanx. The fifth ligament listed in the text is not depicted as it attaches to the medial aspect of the cartilage.

meets and blends with its contralateral partner. This attachment on the proximal border is joined by branches from the deep digital flexor; together, these constitute the "T ligament" that attaches to the palmar surface of the middle phalanx. Fibers of the CSLs also attach the end of the navicular bone to the palmar process and cartilage of the distal phalanx.

Distally, the navicular bone is stabilized by the distal sesamoidean impar ligament, extending from the distal border of the bone to intersect with the insertion of the DDFT (arrow, Figure 1.10).[6]

The distal articular surface of the middle phalanx, the articular surface of the distal phalanx, and the two articular surfaces of the navicular bone form the coffin joint, a hinge joint of limited range of motion. Short collateral ligaments arise from the distal end of the middle phalanx, pass distad deep to the cartilages of the distal phalanx, and terminate on either side of the extensor process and the dorsal part of each cartilage.

The synovial membrane of the distal interphalangeal (coffin) joint has a dorsal pouch that extends proximad on the dorsal surface of the middle phalanx deep to the common digital extensor tendon nearly to the pastern joint. The synovium has a complex relationship on its palmar side to the ligaments and tendons that are found here. The proximal portions wrap around the distal ends of the CSLs. The distal pouch forms a thin extension between the articulation of the navicular bone and the distal phalanx. This pouch's synovial membrane surrounds the distal sesamoidean impar ligament on each side where the distal interphalangeal joint is closely associated with the neurovascular bundle that will enter the distal phalanx. Although a direct connection between the distal interphalangeal joint and the navicular bursa is rare, passive diffusion of injected dye and anesthetic occurs.[7]

The common digital extensor tendon terminates on the extensor process of the distal phalanx, receiving first branches from the suspensory ligament at the level of the pastern and then an additional ligament from each ungual cartilage as it inserts (Figure 1.9).

Pastern

Deep to the skin and superficial fascia on the palmar aspect of the pastern, the proximal digital annular ligament covers the superficial digital flexor as it bifurcates. In this location, it binds down both digital flexor tendons.

Two distinct ligaments of the ergot diverge from beneath the horny ergot on the palmar skin of the fetlock. Each ligament descends obliquely just deep to the skin. Distally it widens and blends into the dense connective tissue of the distal digital annular ligament.

The superficial digital flexor tendon (SDFT) terminates by bifurcating into two branches that insert on the

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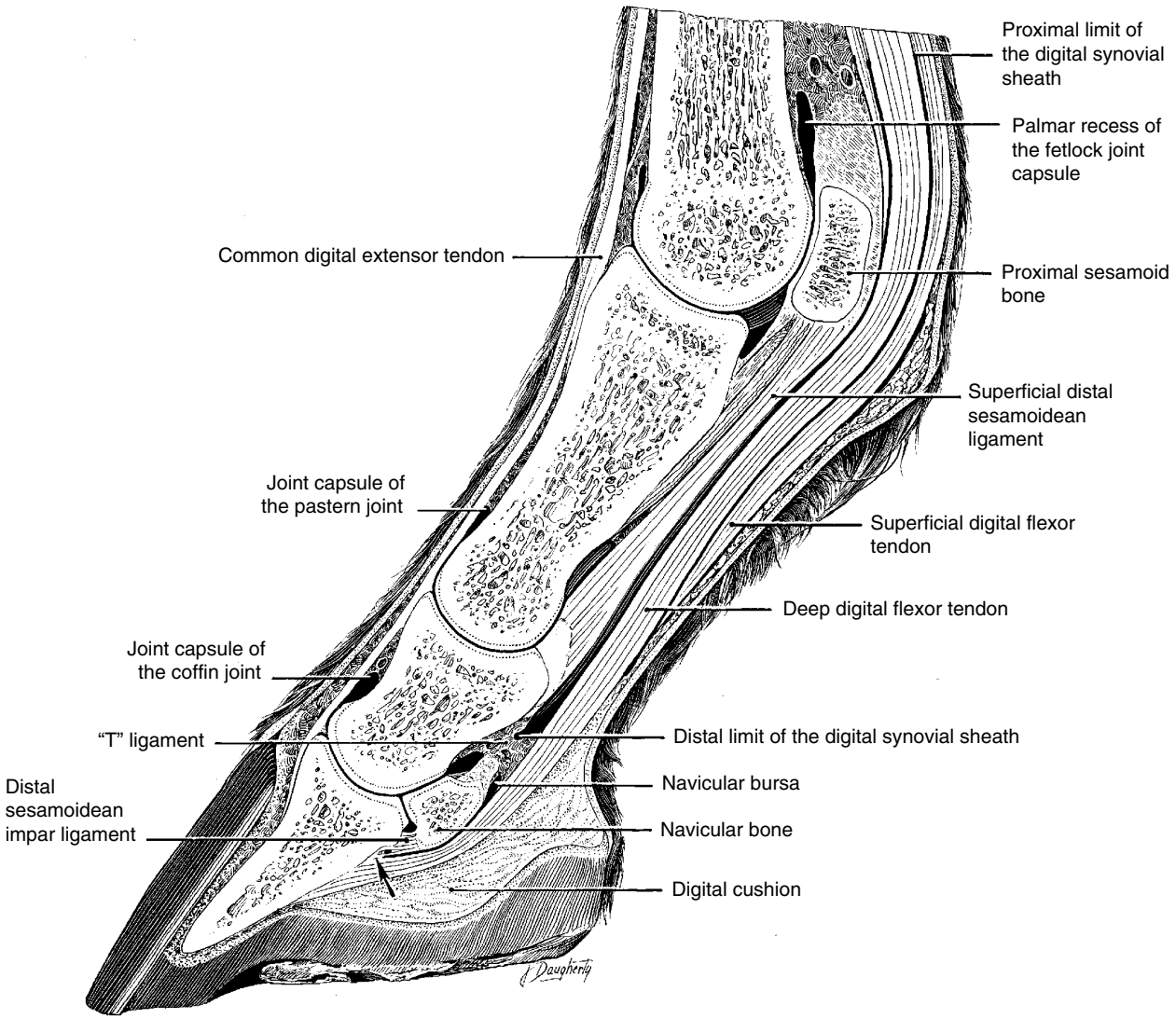


Figure 1.10. Sagittal section of equine fetlock and digit.

proximal extremity of the middle phalanx just palmar to the collateral ligaments of the proximal interphalangeal joint. Traditionally, the insertion of the SDFT has been described as also having additional attachments on the distal extremity of the proximal phalanx; radiographic studies have concluded that these attachments are not strictly part of the flexor tendon and instead represent associated palmar ligaments.[44] The DDFT descends between the two branches of the SDFT. A digital synovial sheath surrounds both tendons and continues in association with the DDFT as far as the "T ligament" (Figures 1.10 and 1.11).

Deep to the digital flexor tendons, a series of ligaments (often collectively referred to as the distal sesamoidean ligaments) extend distad from the bases of the two proximal sesamoid bones (Figure 1.11). These are the functional continuation of the suspensory apparatus into the digit. The most superficial of these is the straight sesamoidean ligament, which attaches primarily to the palmar aspect of the middle phalanx and less robustly to the palmar first phalanx. The wedge-shaped middle (oblique) sesamoidean ligament attaches distally to a rough area on the palmar surface of the proximal phalanx. Deep to these is the pair of cruciate ligaments that cross midline, each attaching distally to the contralateral eminence on the proximal end of the proximal phalanx. Deepest of the distal sesamoidean ligaments are a pair of short sesamoidean ligaments extending from the dorsal aspect of the base of each proximal sesamoid bone to the edge of the articular surface of the proximal phalanx (Figure 1.11).

An extensor branch of the suspensory ligament passes from the abaxial surface of the respective proximal sesamoid bone dorsodistad obliquely across the proximal phalanx to the dorsal surface where each branch blends with the common digital extensor tendon near the distal end of the proximal phalanx. An elongated bursa under each extensor branch is extensive enough to be considered a synovial sheath.[16]

In the dorsal aspect of the pastern, the common digital extensor tendon is attached to the proximal ends of the proximal and middle phalanges on its way to its definitive insertion on the extensor process of the distal phalanx. A bursa often occurs under this tendon near its union with the extensor branches of the suspensory ligament. The tendon of the lateral digital extensor muscle

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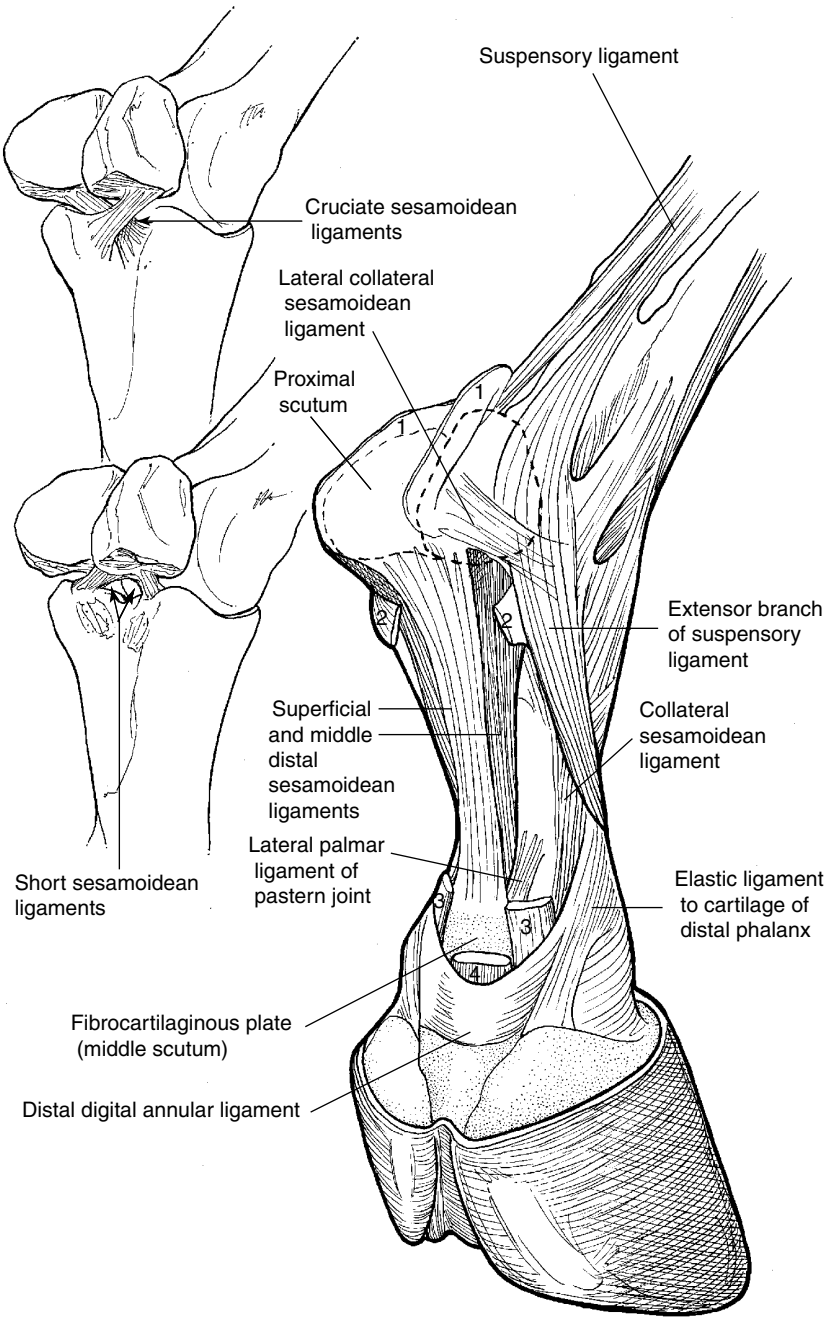


Figure 1.11. Sesamoidean ligaments. Dashed lines indicate positions of the proximal sesamoid bones embedded in the metacarpointersesamoidean ligament. Numbers indicate cut stumps of (1) palmar annular ligament, (2) proximal digital annular ligament, (3) superficial digital flexor, and (4) deep digital flexor tendon.

inserts lateral to the common digital extensor tendon on the proximal dorsal surface of the proximal phalanx.

The proximal interphalangeal (pastern) joint is formed by the condyle on the distal end of the proximal phalanx and two corresponding concave articular foveae on the proximal end of the middle phalanx. Two short collateral ligaments and four palmar ligaments stabilize these bones. The collateral ligaments are oriented vertically between the eminences on the bones rather than parallel to the axis of the digit. The axial pair of palmar ligaments extends from the ridges on the palmar side of the proximal phalanx to the region on each side of the straight sesamoidean ligaments' attachment on the middle phalanx. The abaxial palmar ligaments pass from the lateral aspects of the proximal phalanx to the palmar surface of the middle phalanx. The axial ligaments blend somewhat with the branches of the SDFT and the straight sesamoidean ligament, and they may be difficult to discern as individual entities.

The joint capsule of the pastern joint blends with the deep surface of the common digital extensor tendon dorsally where it is accessible for arthrocentesis (Figure 1.10). It also blends with the collateral ligaments of the joint. The palmar aspect of the capsule extends slightly proximad, compressed between the middle phalanx and the terminal branches of the SDFT and the

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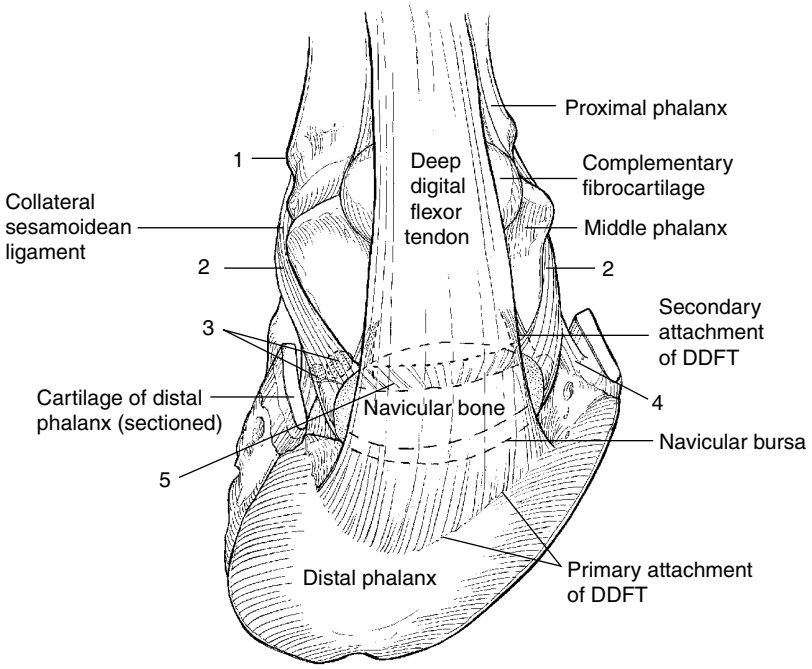


Figure 1.12. Attachments of deep digital flexor tendon and collateral sesamoidean ligaments (CSLs). (1) Attachment of CSL to proximal phalanx, (2) attachment of CSL to middle phalanx, (3) abaxial outpocketings of palmar pouch of the synovial cavity of the distal interphalangeal joint, (4) attachment of CSL to cartilage of the distal phalanx, and (5) attachment of medial and lateral CSLs to navicular bone.

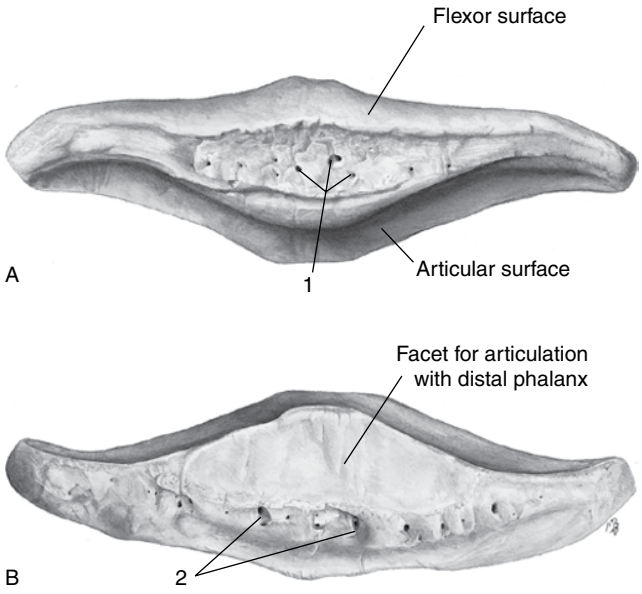


Figure 1.13. Distal sesamoid (navicular) bone. (A) Proximal view. (B) Distal view. (1) Foramina and (2) fossae.

straight sesamoidean ligament. These taut overlying structures subdivide the capsule into medial and lateral pouches that are accessible for arthrocentesis.

Fetlock

The fetlock of the thoracic limb is the region around the metacarpophalangeal (fetlock) joint. On the palmar aspect of the fetlock, the ergot is a prominent cutaneous feature. Its dermal base gives origin to the two distally diverging ligaments of the ergot. Deep to the skin and superficial fascia, the palmar annular ligament of the fetlock encircles the digital flexor tendons and their digital synovial sheath, binding them in the groove between the proximal sesamoid bones. Distally, the palmar annular ligament of the fetlock blends with the proximal digital annular ligament.

The fetlock joint is formed by the distal end of the third metacarpal bone (the cannon bone), the proximal end of the proximal phalanx, the two proximal sesamoid bones, and the extensive fibrocartilaginous palmar ligament that the proximal sesamoids are embedded. The articular surface on the third metacarpal bone, its trochlea, is sharply divided by a sagittal ridge, and this ridge fits into a complementary sagittal groove in the articular surface of the proximal phalanx.

Collateral ligaments of the fetlock joint extend distad from the eminence and depression on each side of the distal cannon bone. The superficial part of each ligament attaches to the edge of the articular surface of the proximal phalanx; the shorter, stouter deep part of the ligament attaches to the abaxial surface of the adjacent proximal sesamoid and the proximal phalanx.

The smooth depression between the proximal sesamoid bones through which the digital flexor tendons pass is formed by the fibrocartilage of the palmar ligament that covers the flexor surfaces of the proximal sesamoid bones. Immediately distal to the canal formed by the encircling annular ligament and the groove between the proximal sesamoids, the DDFT perforates through a ringlike opening in the SDFT, the manica flexoria.

The common and lateral digital extensor tendons pass over the dorsal aspect of the fetlock joint where a bursa

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lies deep to each tendon. Small but common subcutaneous bursae may occur on the palmar surface of the fetlock joint and on the lateral aspect of the joint just proximal to the extensor branch of the suspensory ligament.[30]

The palmar part of the fetlock joint capsule is thicker and more voluminous than the dorsal part. A consistent bursa deep to the digital flexor tendons at the distal end of the cannon bone lies against the thickened capsule and may communicate with the joint cavity.[16] The palmar recess (pouch) of the fetlock joint capsule extends proximad between the cannon bone and the suspensory ligament. This pouch is palpable and even visible in the presence of joint effusion.

Support for the fetlock and stabilization during weight-bearing and locomotion is rendered by the suspensory apparatus, a part of the stay apparatus. The suspensory apparatus of the fetlock comprises the suspensory ligament (interosseus medius muscle) and its extensor branches to the common digital extensor tendon, the proximal sesamoids embedded in the palmar ligament, and the distal sesamoidean ligaments extending from the bases of the proximal sesamoid bones to the proximal and middle phalanges.

Blood Vessels of the Digit and Fetlock

Arterial Supply

The arterial supply to the digit and fetlock of the thoracic limb is derived principally from the medial palmar artery. This substantial vessel divides in the distal fourth of the metacarpus between the digital flexor tendons and the suspensory ligament into the medial and lateral digital arteries. An anastomotic branch from the distal deep palmar arch unites with the lateral digital artery to form the superficial palmar arch. Branches from this arch directly supply the fetlock joint (Figure 1.8).

Each digital artery becomes superficial on the fetlock. The artery lies palmar to its satellite vein, running between the palmar digital nerve and its dorsal branch (Figures 1.14 and 1.15). As each digital artery courses distad over the fetlock, it gives off branches to the fetlock joint, digital extensor and flexor tendons, digital synovial sheath, ligaments, fascia, and skin.

Distal to the fetlock, the digital arteries run parallel with the borders of the DDFT, giving off branches that create encircling anastomoses around the proximal and middle phalanges. The anastomosis associated with the proximal phalanx is created from dorsal and palmar branches of the proximal phalanx that encircle the digit (Figure 1.8). The palmar branch joins the contralateral vessel between the straight and oblique sesamoidean ligaments. The dorsal branch anastomoses with the contralateral vessel deep to the common digital extensor tendon.

At the level of the middle phalanx, dorsal and palmar branches again arise. The dorsal branch of the middle phalanx anastomoses with the contralateral branch deep to the common digital extensor tendon to form a coronary arterial circle. This vascular complex supplies branches to the distal interphalangeal joint, common digital extensor tendon, perioplic and coronary coria, fascia, and skin. The palmar branches of the middle phalanx run parallel to the proximal border of the distal sesamoid bone, uniting to complete the arterial circle around the middle phalanx. Branches from the palmar portion of the arterial circle supply an anastomotic proximal navicular plexus; this gives rise to several small arteries that enter the foramina along the proximal border of the navicular bone.[9,18] The bone receives approximately one-third of its blood supply from this plexus.

Immediately distal to the distal sesamoid bone, each digital artery gives off one to three small arteries that supply the distolateral border of the navicular bone. The digital arteries further give rise to branches that form a distal navicular plexus within the distal sesamoid impar ligament. Six to nine distal navicular arteries from the plexus enter the distal sesamoid bone through the distal border. These supply the distal two-thirds of the distal sesamoid bone.[18]

Near the level of the pastern joint, a prominent bulbar artery (artery of the digital cushion) arises from each digital artery (Figure 1.8). Their branches supply the frog, the digital cushion, palmar part of the cuneate corium, laminar corium of the heel and bar, and palmar parts of the perioplic and coronary coria. A small coronal artery arises from either the digital artery or the bulbar artery, and its branches supply the heel and perioplic corium.

Within the foot opposite each end of the navicular bone, an artery to the dermal laminae of the heel arising from the digital artery has been noted on radiographic angiograms.[10,18] At the level of the palmar process of the distal phalanx, the digital artery gives off the dorsal branch of the distal phalanx and then continues distad to form the terminal arch within the distal phalanx. The dorsal branch of the distal phalanx gives off a small artery supplying the digital cushion and corium of the frog and then passes through a notch or foramen in the palmar process. Emerging onto the dorsal side of the distal phalanx, the dorsal branch of the distal phalanx bifurcates. One branch supplies the corium of the heels and quarters; the other courses dorsad in a bony sulcus to supply the corium of the toe and to form anastomoses with other arteries on the distal phalanx (Figure 1.8).

The continuation of the digital artery enters a solar foramen and anastomoses with the contralateral artery to form the terminal arch within the solar canal (Figure 1.8). Branches from the terminal arch course through the bone, 4 or 5 of them emerging through foramina on the parietal surface to supply the proximal part of the laminar corium; another 8–10 vessels emerge through foramina near the solar border of the bone and anastomose to form the prominent marginal artery of the sole. This artery supplies the solar and cuneate coria.

The arterial network of the corium can be divided into three regions with independent blood supplies: (1) the dorsal coronary corium, (2) the palmar part of the coronary corium and laminar corium, and (3) the dorsal laminar corium and solar corium.[32] Other regions are supplied by multiple other small arteries. Angiographic studies indicate that blood flow within dermal laminae is from distal to proximal.[10,32]

Branches of the digital arteries in the hindfoot are essentially the same as in the forefoot except for the blood supply to the distal sesamoid bone. In 50% of hindfeet examined in a definitive study, the collateral arch from the plantar branches of the middle phalanx supplied the primary arteries to the proximal navicular network.[18]

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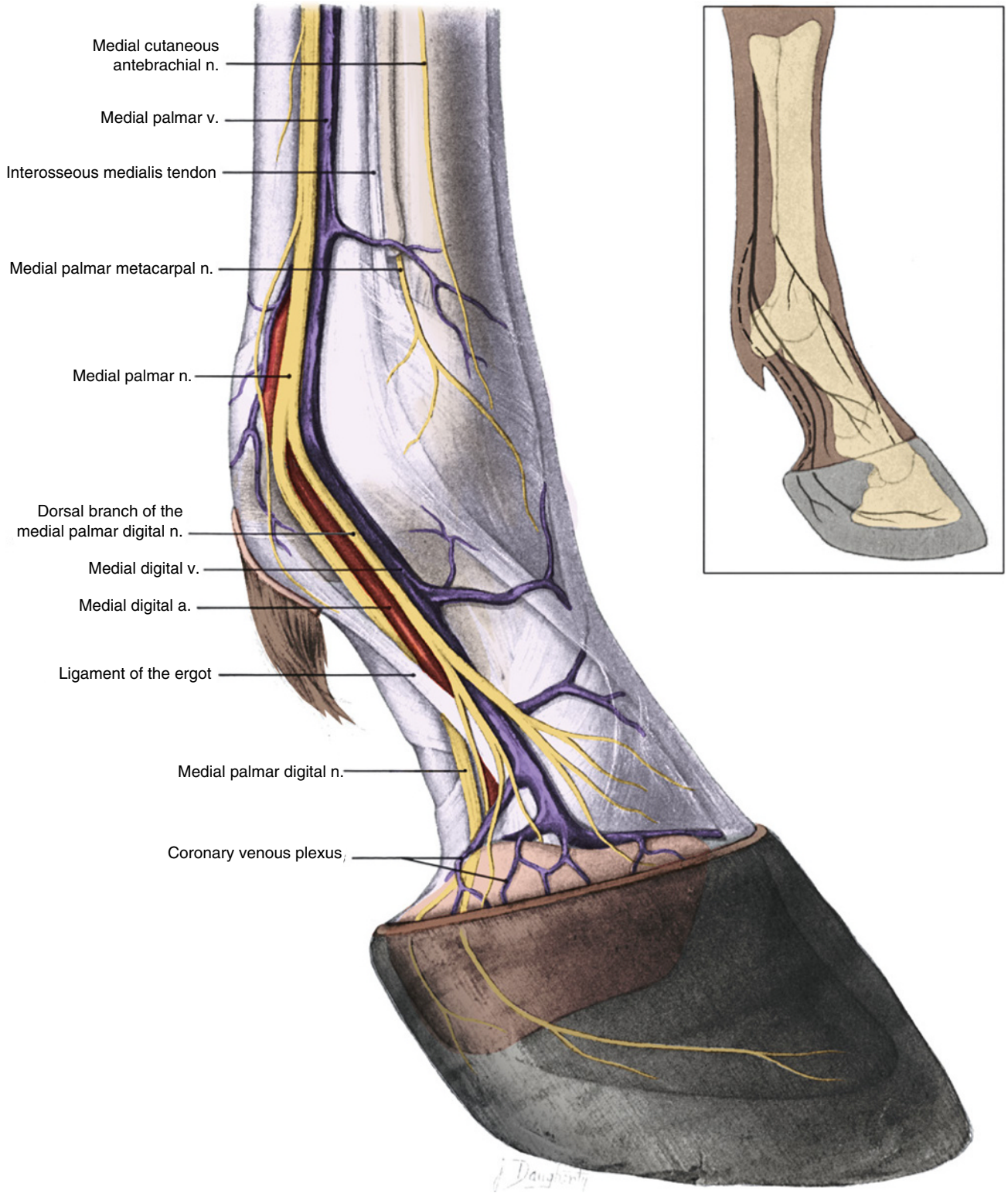


Figure 1.14. Medial aspect of distal metacarpus, fetlock, and digit with skin and superficial fascia removed. Inset: Schematic of the distribution of major nerves; dashed lines indicate variant branches.

Venous Drainage

Venous drainage from the laminar corium begins with veins from the laminar circulation continuing into the parietal (associated with the hoof wall) venous plexus and the coronary venous plexus (Figure 1.16). Veins from the perioplic and coronary coria drain toward the coronary venous plexus, and those from the solar and cuneate coria drain into the solar venous plexus.[26]

Veins in the solar canal come together at the level of the distal sesamoid bone to form the medial and lateral

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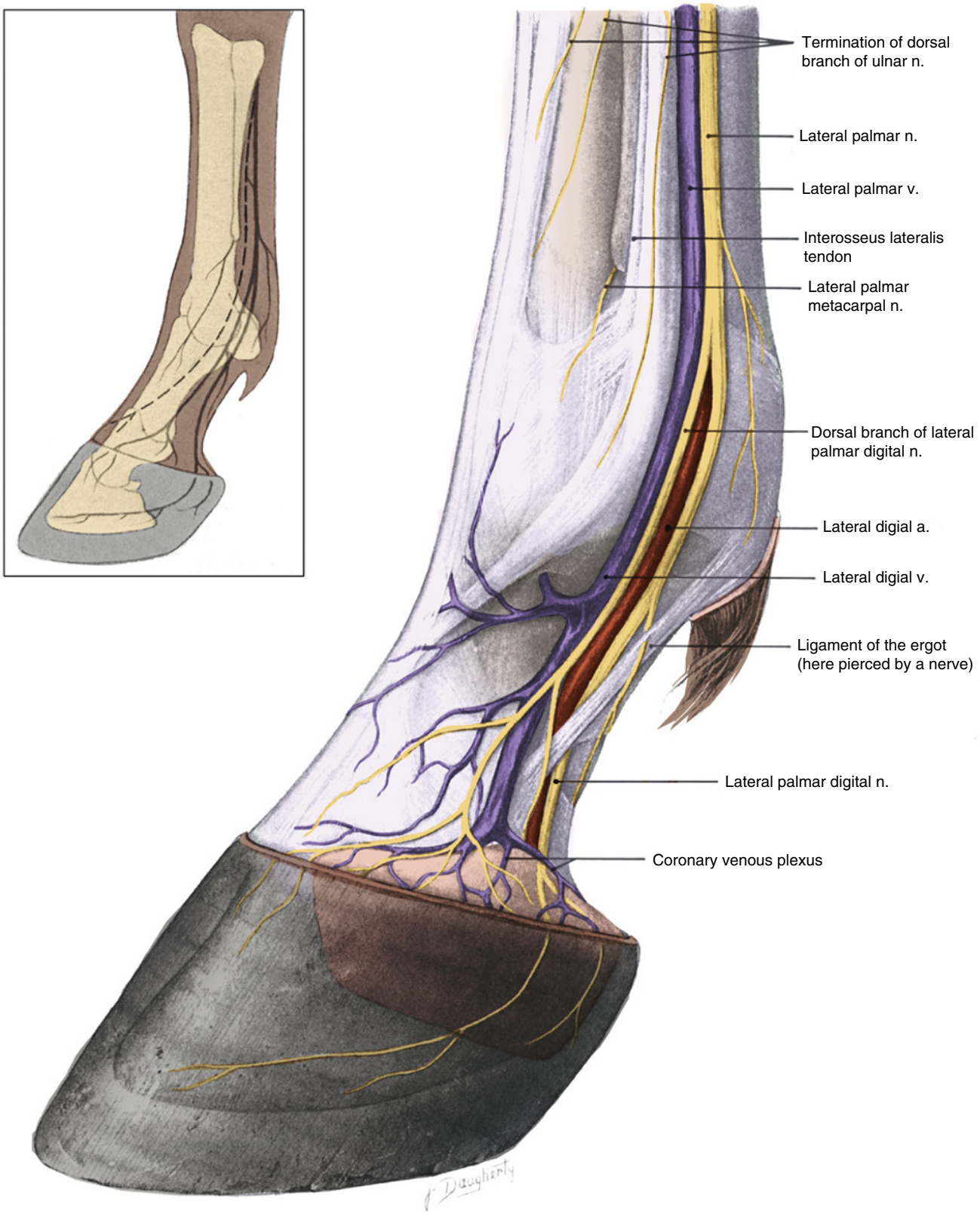


Figure 1.15. Lateral aspect of distal metacarpus, fetlock, and digit with skin and superficial fascia removed. Inset: Schematic of the distribution of major nerves; dashed lines indicate variant branches.

terminal veins. Each terminal vein joins with branches of an inner venous plexus to form a digital vein. The digital vein receives branches from the distal sesamoid bone, coronary vein, inner venous plexus, and large bulbar vein carrying blood from the heel. Most venous blood in the foot is drained by the veins located in the palmar aspect which are largely valveless. Some valves are present in the tributaries of the coronary and subcoronary

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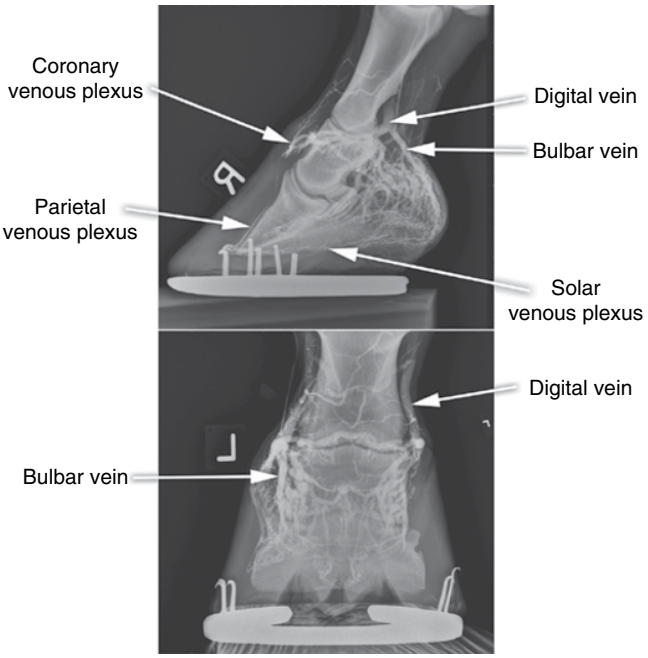


Figure 1.16. Venogram of equine foot. *Source*: Photo courtesy of Dr. Andrew Lewis.

veins and in the bulbar veins and their branches. Thus, the flow of blood may take different routes with the weight-bearing force essential to its proximal flow.[26]

Nerves of the Digit and Fetlock

As they descend to the fetlock, the medial and lateral palmar nerves supply small branches to the fetlock and flexor tendons. Each then gives off a dorsal branch at this level, continuing over the widest part of the joint as the medial and lateral palmar digital nerves (Figures 1.14 and 1.15). The corresponding digital artery lies between this dorsal branch and the palmar digital nerve. The dorsal branch courses distad between the digital vein and artery, branching midway down the pastern. In approximately one-third of the cases, an intermediate branch also arises from the dorsal aspect of the palmar digital nerve.[29] The dorsal (and intermediate, when present) branches supply sensory and vasomotor innervation to the dorsal part of the fetlock joint, dorsal parts of the interphalangeal joints, coronary corium and dorsal parts of the laminar and solar coria, and dorsal part of the cartilage of the distal phalanx.[5,12]

The main continuation of the palmar digital nerve descends palmar and parallel to the digital artery. The nerve and artery are deep to the ligament of the ergot as the latter descends obliquely across the lateral aspect of the pastern. A branch may arise from the lateral palmar digital nerve and perforate the lateral ligament of the ergot (Figure 1.15).

The palmar digital nerves supply the palmar parts of the fetlock joint capsule and of the digit: skin, pastern joint capsule, digital synovial sheath and flexor tendons, distal sesamoidean ligaments, coffin joint capsule, navicular bone and its ligaments, navicular bursa, palmar part of the cartilage of the distal phalanx, part of the laminar corium, coria of the sole and frog, and digital cushion.

A fine terminal branch of each palmar digital nerve and an accompanying small artery constitute a neurovascular bundle that descends adjacent to the synovial membrane of the distal interphalangeal joint to enter the distal phalanx.[5]

Additional cutaneous innervation of the fetlock is supplied by terminal branches of the medial cutaneous antebrachial nerve dorsomedially and the dorsal branch of the ulnar nerve dorsolaterally. Medial and lateral palmar metacarpal nerves emerge immediately distal to the distal end of the respective small metacarpal (splint) bone, supplying branches to the fetlock joint capsule and ramifying in the superficial fascia of the pastern. It has been reported that in some instances, a terminal branch from the medial palmar metacarpal nerve descends to the coronary band (Figure 1.14).[19,31] An occasional variant, a palmarly directed branch from the medial palmar nerve in the distal metacarpus, courses palmar to the medial palmar digital nerve, reaching the digital cushion (Figure 1.14). Another variant branch may arise from the lateral palmar nerve in the proximal metacarpus, cross over the fetlock, and extend obliquely to the coronary band (Figure 1.15).

Electrophysiologic studies confirm that stimuli on the medial half of the digit and fetlock of the forelimb are mediated by the median nerve and stimuli on the lateral half are mediated by the median and ulnar nerves.[5]

Although direct communication between the distal interphalangeal joint and the navicular bursa is very rare, indirect communication via diffusion of molecules has been demonstrated.[8] Dye injected experimentally into the distal interphalangeal joint diffused into the navicular bursa and also stained the synovial coverings of the CSLs and the distal sesamoidean impar ligament and the medullary cavity of the navicular bone.

Basic Functions of the Digit and Fetlock

In the standing position, the fetlock and digit are prevented from nonphysiologic hyperextension by the suspensory apparatus of the fetlock (interosseus muscle [suspensory ligament], palmar ligament and proximal sesamoids, and distal sesamoidean ligaments), digital flexor tendons, and collateral ligaments of the joints.

During flexion and extension of the digit, most of the movement comes from the fetlock joint. The least amount of movement is in the pastern joint, and movement in the coffin joint is intermediate. Although the pastern joint is a hinge joint, normally providing only limited flexion and extension, manipulation can produce transverse movement and some rotation when the joint is flexed.

When the unshod hoof contacts the ground, the heels usually strike first, followed in sequence by the ground surfaces of the quarters and toe. Expansion of the heels is facilitated by the elasticity of the hoof wall. Most of the impact is sustained by the hoof wall, and compression of the wall creates tension on the interlocking epidermal and dermal laminae and, hence, to the periosteum of the distal phalanx. The concave sole does not absorb much force although it is depressed somewhat by the downward force of the distal phalanx, causing expansion of the quarters. Descent of the coffin joint occurs as the navicular bone moves in a distopalmar direction,

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stretching its collateral (suspensory) and impar ligaments and pushing against the navicular bursa and tendon of the deep digital flexor muscle. Forces acting on the distal phalanx are indicated in Figure 1.17. Magnitude and direction of the forces may change with limb position and loading state.[22] Concussion is further dissipated by pressure from the frog being transmitted to the digital cushion and the cartilages of the distal phalanx.

Lateral expansion of the hoof and cartilages of the distal phalanx compresses the venous plexuses of the foot, forcing blood proximad into the digital veins. The hydraulic shock absorption afforded by the blood within the vessels augments the direct cushioning by the frog and digital cushion and the elasticity of the hoof wall.

During concussion, the palmar ligaments of the pastern joint, the straight sesamoidean ligament, and the tendon of the deep digital flexor provide the tension necessary to prevent overextension of the pastern joint. Contraction of the superficial digital flexor muscle tightens its insertions on the middle phalanx, preventing the pastern joint from buckling.

The suspensory apparatus of the fetlock and the digital flexor tendons ensure that overextension of the fetlock joint is minimized when the hoof strikes the ground. Under extreme loading conditions (e.g. at a gallop or when landing a jump), the palmar aspect of the fetlock comes very close to the ground. During this descent of the fetlock, the coffin joint is flexed by the increased tension on the DDFT.

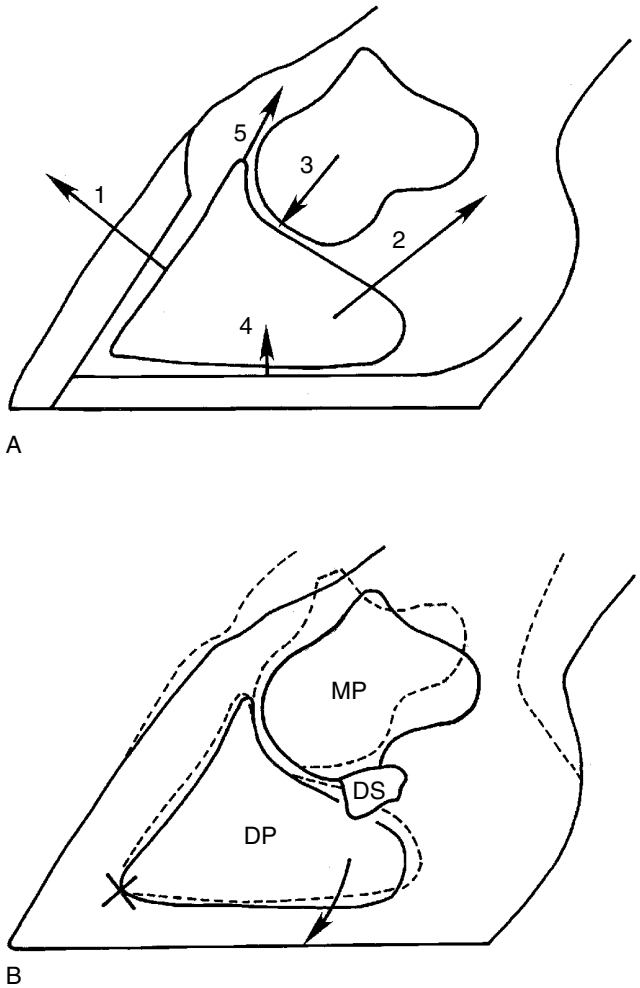


Figure 1.17. (A) Diagram of forces acting on distal phalanx. (1) Forces from laminae of wall, (2) tensile force from deep digital flexor tendon, (3) compressive force from middle phalanx, (4) compressive force from sole, and (5) tensile forces from extensor branches of suspensory ligament and common (long, in pelvic limb) digital extensor tendon. (B) Position changes in middle phalanx (MP), distal phalanx (DP), distal sesamoid (DS), and hoof wall resulting from weight-bearing. X = axis about which the distal phalanx rotates; arrow indicates rotation from unloaded (dotted line) to loaded (solid line) state. *Source*: Redrawn from Leach.[22]

Metacarpus

The equine metacarpus consists of the large third metacarpal (cannon) bone, the second (medial) and fourth (lateral) metacarpal bones (splint bones), and the structures associated with them. The shaft of each splint bone is united to the cannon bone by an interosseous ligament. Length and curvature of the shafts and the prominence of the free distal ends ("buttons") of the splint bones are variable. The proximal ends of the metacarpal bones articulate with the distal row of carpal bones. The second metacarpal articulates with the second and third carpals; the third metacarpal articulates with the second, third, and fourth carpals; and the fourth metacarpal articulates with the fourth carpal bone.

Dorsal Aspect

The structures of the dorsal aspect of the metacarpus receive their blood supply from small medial and lateral dorsal metacarpal arteries. These originate from the network of small arteries on the dorsum of the carpus (dorsal carpal rete) and descend between the cannon bone and the respective medial or lateral splint bone. The medial cutaneous antebrachial nerve (Figures 1.14 and 1.20) and the dorsal branch of the ulnar nerve (Figures 1.18 and 1.21) provide innervation to this region.

Deep to the skin, the common digital extensor tendon inclines laterad as it ascends from its central position at the fetlock across the dorsum of the cannon bone. Just distal to the carpus, the main tendon and the tendon of the much smaller radial head of the muscle run lateral to the extensor carpi radialis tendon on the prominent metacarpal tuberosity of the third metacarpal bone (Figure 1.20). The tendon of the lateral digital extensor muscle is lateral to the common extensor tendon, and the small radial tendon of the latter usually joins the lateral digital extensor tendon (Figure 1.21). Occasionally the radial tendon pursues an independent course to the fetlock. A strong fibrous band from the accessory carpal bone blends with the lateral digital extensor tendon as it angles dorsad in its descent from the carpus.

Medial and Lateral Aspects

The medial palmar vein is the direct continuation of the medial digital vein at the fetlock. In the distal half of the metacarpus, the vein is related palmarly to the medial

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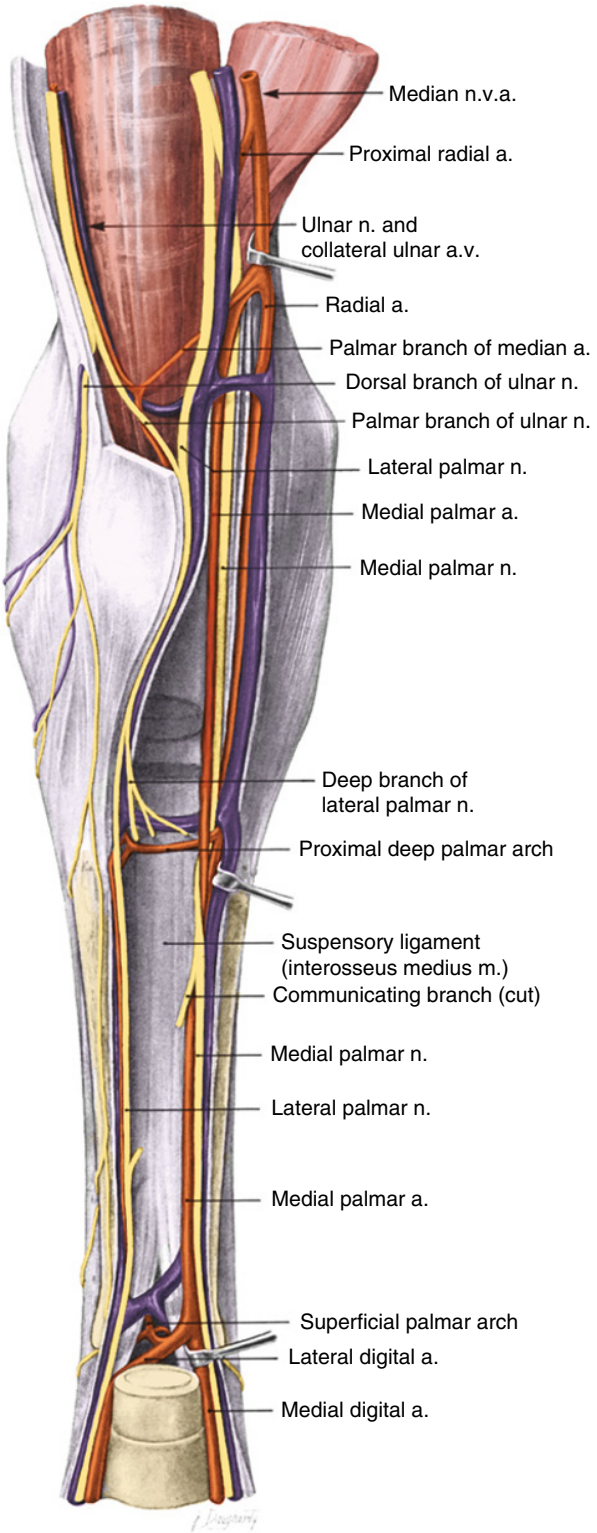


Figure 1.18. Caudal view of left carpus and metacarpus; most of the digital flexor tendons are removed.

palmar nerve (Figure 1.14); in the proximal half, the large medial palmar artery can be seen palmar to the vein (Figure 1.18). A similar relationship exists on the lateral side except that the corresponding lateral palmar artery is generally quite small. The palmar nerves run in the groove between DDFT and suspensory ligament, supplying the digital flexor tendons and the skin superficial to them. At the middle of the metacarpus, the medial palmar nerve detaches a communicating branch that angles distolaterad in the subcutaneous fascia superficial to the flexor tendons to join the lateral palmar nerve. Branches from the dorsal branch of the ulnar nerve ramify in the fascia and skin of the lateral aspect of the metacarpus. Branches from the medial cutaneous antebrachial nerve (itself a branch of the musculocutaneous nerve) supply the medial and dorsal skin of the metacarpus with the large dorsal branch reaching the skin over the dorsomedial aspect of the fetlock.

Palmar Aspect

The SDFT is deep to the skin and subcutaneous fascia throughout the length of the metacarpus. Dorsally, it is intimately related to the DDFT. The latter, in turn, lies against the palmar surface of the suspensory ligament (a.k.a. m. interosseus medius; middle or third interosseous muscle). The carpal synovial sheath, enclosing both digital flexor tendons, extends distad as far as the middle of the metacarpus. At this level, the DDFT is joined by its accessory ligament (carpal check ligament or "inferior" check ligament), a distal continuation of the palmar carpal ligament (Figure 1.32). The medial and lateral lumbricales muscles, fleshy in other species, are reduced in the horse to fibrous slips that originate from either side of the DDFT and insert in the fibrous tissue deep to the ergot. The lumbricales in the pelvic limb tend to be better developed. The digital synovial sheath around the digital flexor tendons is present through the distal fourth of the metacarpus (Figure 1.11).

The metacarpal groove, formed by the palmar surface of the third metacarpal bone and the axial surfaces of the second and fourth metacarpal bones, is occupied by the suspensory ligament. The suspensory ligament arises from the distal row of carpal bones and the proximal end of the third metacarpal bone (Figures 1.18 and 1.19). It is broad, relatively flat, and shorter than the suspensory ligament of the hindlimb. Variable amounts of muscle fibers are seen within the mainly collagenous suspensory ligament primarily in foals, in which these are gradually replaced by collagen with musculoskeletal maturation.[45] In the distal fourth of the metacarpus, the suspensory ligament bifurcates to become associated with the two proximal sesamoid bones. Each branch makes a broad attachment across the abaxial surface of proximal sesamoid bone and blends with the origin of the ipsilateral CSL (Figure 1.12). Two (medial and lateral) vestigial interosseous muscles originate on the heads of the respective splint bones; their slender tendons pass distad alongside the splint bones and end near the buttons by blending into the fascia of the fetlock.

A deep branch of the lateral palmar nerve supplies branches to and then perforates the suspensory ligament, whereupon it divides into the medial and lateral palmar metacarpal nerves. These nerves and their satellite vessels lie in the grooves formed by the cannon bone and the respective splint bones (Figure 1.19). After sending branches to the fetlock joint capsule, each palmar metacarpal nerve emerges distal to the distal extremity

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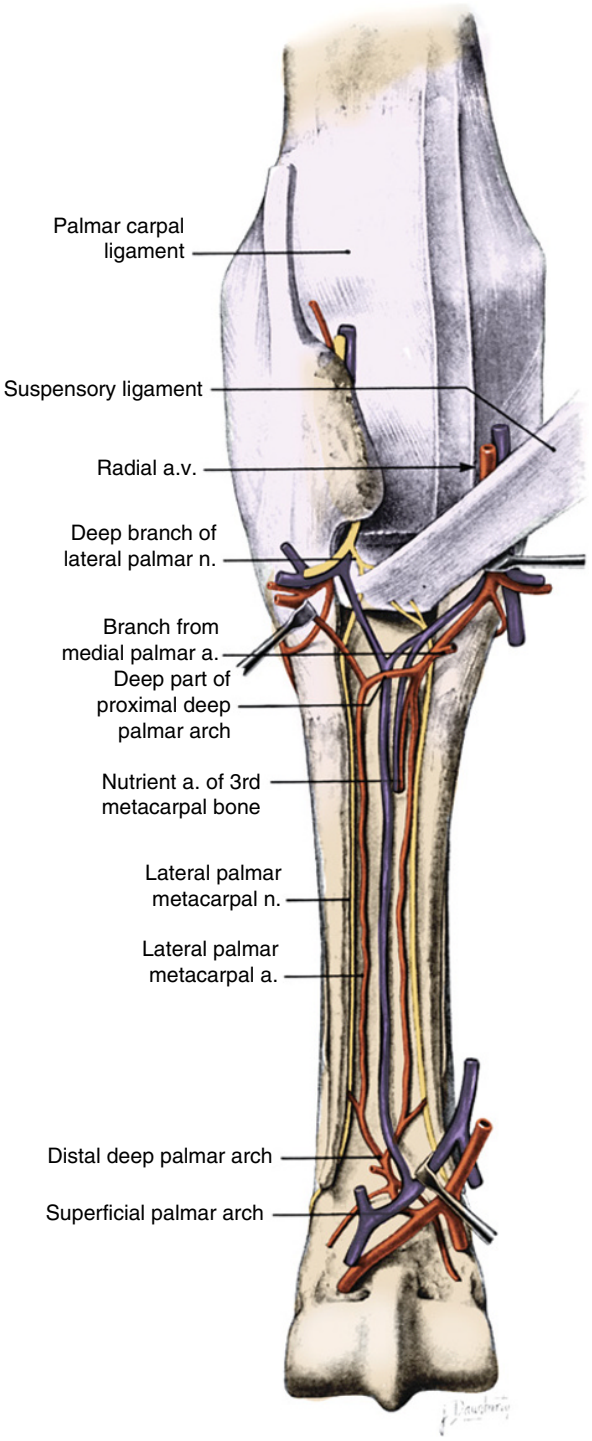


Figure 1.19. Deep dissection of caudal aspects of left carpus and metacarpus with medial palmar artery removed.

(the "button") of the respective splint bone to ramify in skin of the pastern.

The palmar metacarpal arteries originate from the proximal deep palmar arch, an anastomotic complex formed by the termination of the radial artery where it joins the smaller palmar branch of the median artery (Figure 1.19). Part of the arch lies between the carpal check ligament and the suspensory ligament. The medial palmar metacarpal artery supplies a nutrient artery to the third metacarpal bone. Small branches from the palmar metacarpal arteries extend through the interosseous spaces between cannon and splint bones to join the dorsal metacarpal arteries. In the distal fourth of the metacarpus, the palmar metacarpal arteries anastomose to form the distal deep palmar arch. A branch from this arch to the lateral digital artery is termed the superficial palmar arch.

A single, large palmar metacarpal vein courses proximad to join the venous deep palmar arch.

The vascular patterns described above are subject to considerable variations, but the variations are of no clinical significance.

Carpus

The carpal region includes the carpal bones (radial, intermediate, ulnar, and accessory in the proximal row; first, second, third, and fourth in the distal row), the distal end of the radius, the proximal ends of the three metacarpal bones, and the soft tissue structures adjacent to these osseous components.

Dorsal Aspect

A vascular network in the skin on the dorsal carpus, the rete carpi dorsale, is formed by branches from the cranial interosseus, transverse cubital, and proximal radial arteries. Medial and lateral cutaneous antebrachial nerves supply branches to the medial and dorsal aspects of the carpus. The dorsal branch of the ulnar nerve emerges between the tendon of the flexor carpi ulnaris muscle and the short tendon of the extensor carpi ulnaris or between the short and long tendons of the latter muscle (Figure 1.21). The nerve supplies branches to the fascia and skin of the dorsal and lateral aspects of the carpus.

The tendons of each of the antebrachial muscles that cross the carpus are invested with synovial sheaths, excepting the extensor carpi ulnaris (formerly ulnaris lateralis) and flexor carpi ulnaris mm. (Figures 1.20 and 1.21). The tendon sheath of the extensor carpi radialis muscle terminates at the middle of the carpus, and then the tendon becomes adherent to the retinaculum as it reaches its insertion on the metacarpal tuberosity (Figure 1.20). A distal bursa near the insertion often communicates with the carpometacarpal joint.

A subtendinous bursa lies between the tendon of the extensor carpi obliquus and the medial collateral ligament of the carpus (Figure 1.20). In most foals younger than 2 years, the bursa is a separate synovial structure; in older horses it communicates with the adjacent tendon sheath.[36]

The extensor retinaculum serves as the dorsal part of the common fibrous joint capsule of the carpal joints. It attaches to the radius, the dorsal intercarpal and dorsal carpometacarpal ligaments, the carpal bones, and the third metacarpal bone. Laterally and medially it blends with the collateral ligaments of the carpus.

Lateral Aspect

The lateral collateral carpal ligament originates from the styloid process of the radius (Figure 1.22). The

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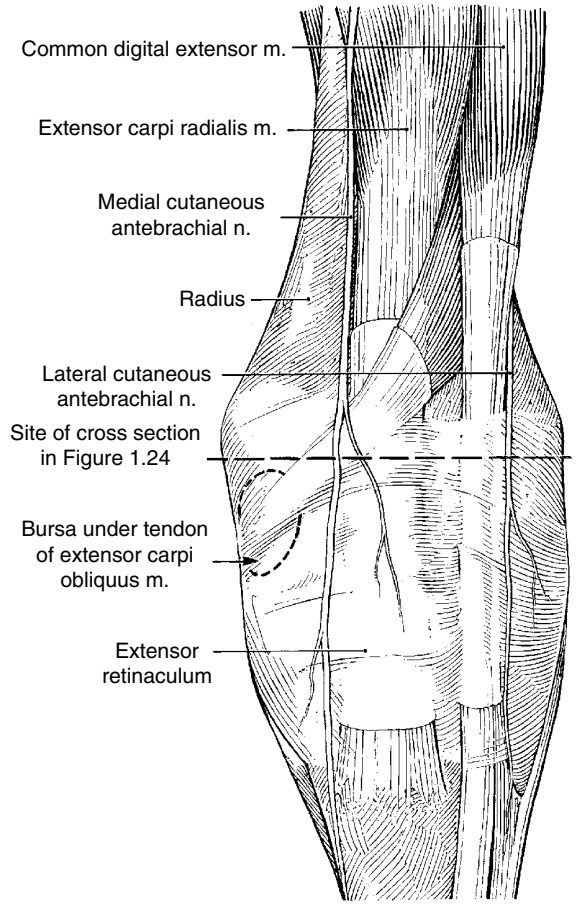


Figure 1.20. Dorsal view of left carpus.

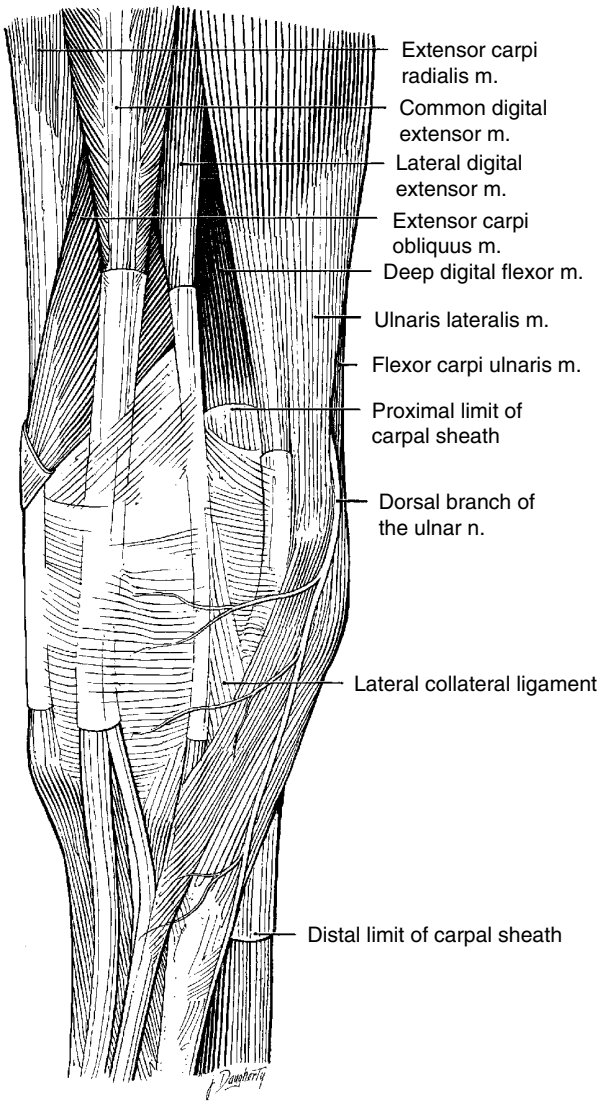


Figure 1.21. Lateral view of left distal forearm, carpus, and proximal metacarpus. Note that the ulnaris lateralis is now called extensor carpi ulnaris.

superficial part of the ligament attaches distally on the fourth metacarpal bone and partly on the third metacarpal bone. A canal between the superficial part and the deep part of the ligament provides passage for the tendon of the lateral digital extensor muscle and its synovial sheath. The deep part of the ligament attaches on the ulnar carpal bone.

Palmar to the lateral collateral carpal ligament, four ligaments attach to the accessory carpal bone, attaching it to adjacent bones (Figure 1.22). Two muscles have insertions on the accessory carpal bone. The short tendon of the extensor carpi ulnaris muscle (formerly ulnaris lateralis m.) attaches to the proximal border and lateral surface of the bone; the muscle's long tendon, enclosed in a synovial sheath, passes through a groove on the bone's lateral surface and then continues distad to insert on base of the lateral splint bone (Figure 1.21). Proximally, a pouch of the antebrachiocarpal joint capsule lies between the long tendon of the extensor carpi ulnaris and the lateral styloid process of the radius. The single tendon of the flexor carpi ulnaris muscle attaches to the proximal border of the accessory carpal bone. A strong fibrous band from the accessory carpal bone attaches to the lateral digital extensor tendon.

Medial Aspect

On the medial side of the carpus, the skin and fascia receive blood from branches of the radial artery. The medial cutaneous antebrachial nerve provides the innervation.

The medial collateral carpal ligament extends from the medial styloid process of the radius and widens distally to attach to the second and third metacarpal bones. Bundles of fibers also attach to the radial, second, and third carpal bones (Figure 1.23). The ligament also joins the flexor retinaculum on the palmar aspect of the joint. At this juncture a canal is formed that accommodates the passage of the tendon of the flexor carpi radialis and its synovial sheath as the tendon descends to the base of the medial splint bone. An inconstant first carpal bone may be embedded in the palmar part of the medial collateral carpal ligament adjacent to the second carpal bone.

Palmar Aspect

The flexor retinaculum is a fibrous band extending from the medial collateral ligament, distal end of the radius, radial and second carpal bones, and proximal

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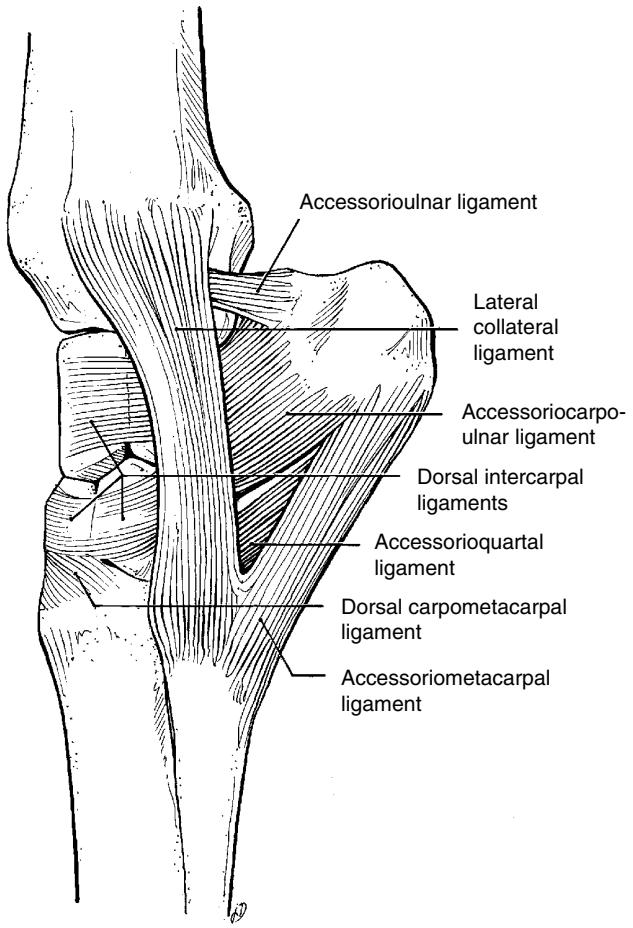


Figure 1.22. Carpal ligaments, lateral view.

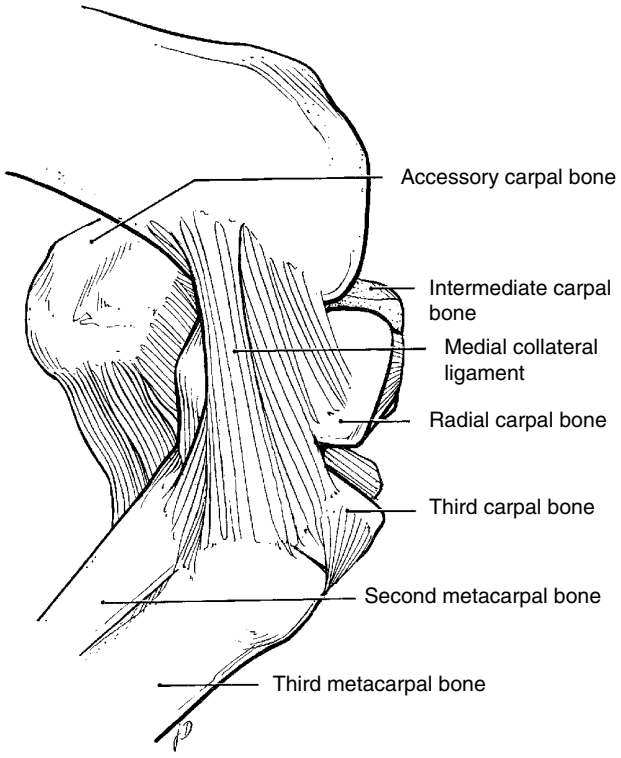


Figure 1.23. Carpal ligaments, medial view.

end of the second metacarpal bone laterad to the accessory carpal bone. By bridging the carpal groove, the flexor retinaculum forms the mediopalmar wall of the carpal canal. It blends both proximally and distally with the fascia of the limb. Proximally, the fan-shaped accessory ligament of the SDFT (radial check ligament) completes the medial wall of the carpal canal. The accessory carpal bone and its two distal ligaments form the lateral wall of the carpal canal. The palmar carpal ligament forms the smooth wall interposed between flexor tendons and the carpal bones; it serves as part of the common fibrous capsule on the palmar side of the carpus. Distally, the palmar carpal ligament gives origin to the carpal check ligament of the DDFT.

The carpal canal (Figures 1.24 and 1.25) contains the following structures: the SDFT and DDFT enclosed in the carpal synovial sheath, the medial palmar nerve and artery, and the lateral palmar nerve, artery, and vein. Medial to the carpal canal, just outside the flexor retinaculum, the tendon of the flexor carpi radialis descends to its attachment on the head of the medial splint bone. The radial artery and vein lie palmar to this tendon embedded in the flexor retinaculum.

The carpal synovial sheath enclosing the digital flexor tendons extends from 8 to 10 cm proximal to the antebrachiocarpal joint to near the middle of the metacarpus (Figure 1.25). Fibers from the carpal check ligament of the SDFT blend into the medial aspect of the proximal end of the sheath. Between the flexor tendons, an intertendinous membrane attaches to the palmaromedial surface of the DDFT and the dorsomedial surface of the SDFT, dividing the carpal synovial sheath into lateral and medial compartments.[40]

In the forearm proximal to the carpus, the palmar branches of the median and collateral ulnar arteries anastomose deep to the flexor carpi ulnaris muscle (Figure 1.18). Small branches from the palmar branch of the median and proximal radial arteries supply the vascular network of the deep palmar carpal region, the rete carpi palmare. The small lateral palmar artery continues distad to near the proximal end of the fourth metacarpal bone where it participates with the radial artery in forming the proximal deep palmar arch. Branches from the radial artery (also a terminal branch of the median artery) extend around the medial aspect of the carpus to contribute to the dorsal carpal rete.

Carpal Joints

The bones of the carpus abut one another at a number of intercarpal joints, but these are capable only of minimal gliding movements. The overall movement of the carpus comes from the horizontally oriented joints: antebrachiocarpal (radiocarpal) between the radius and proximal row of carpal bones, middle carpal between proximal and distal rows of carpal bones, and carpometacarpal between the distal row of carpal bones and the metacarpals. Nearly all of the flexion and extension of the carpus come from the antebrachiocarpal and middle carpal joints; the carpometacarpal joint is a plane joint with minimal movement. The extensive antebrachiocarpal synovial sac extends between the carpal bones of the proximal row, including the accessory carpal bone. It typically does not communicate with the

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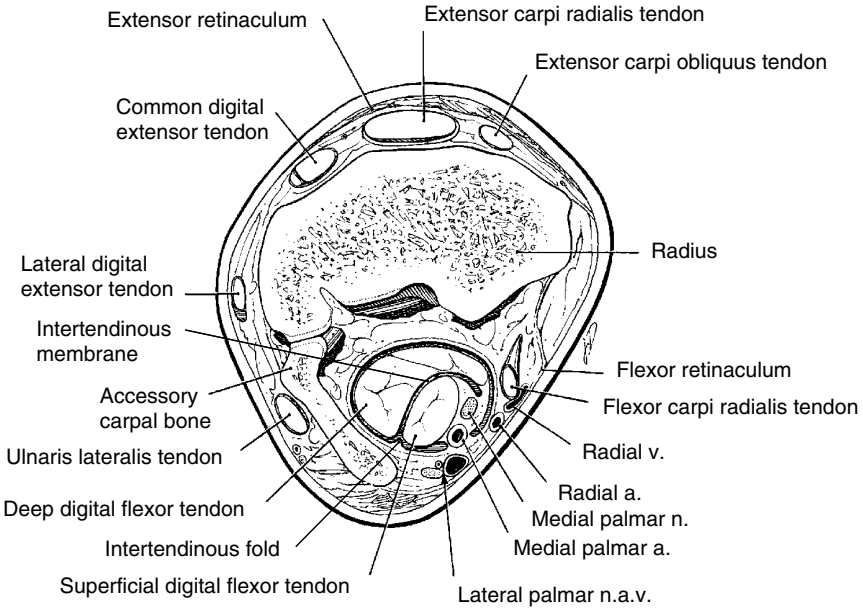


Figure 1.24. Cross section just proximal to the left antebrachiocarpal joint. Note that the ulnaris lateralis (tendon labeled here) is now called extensor carpi ulnaris.

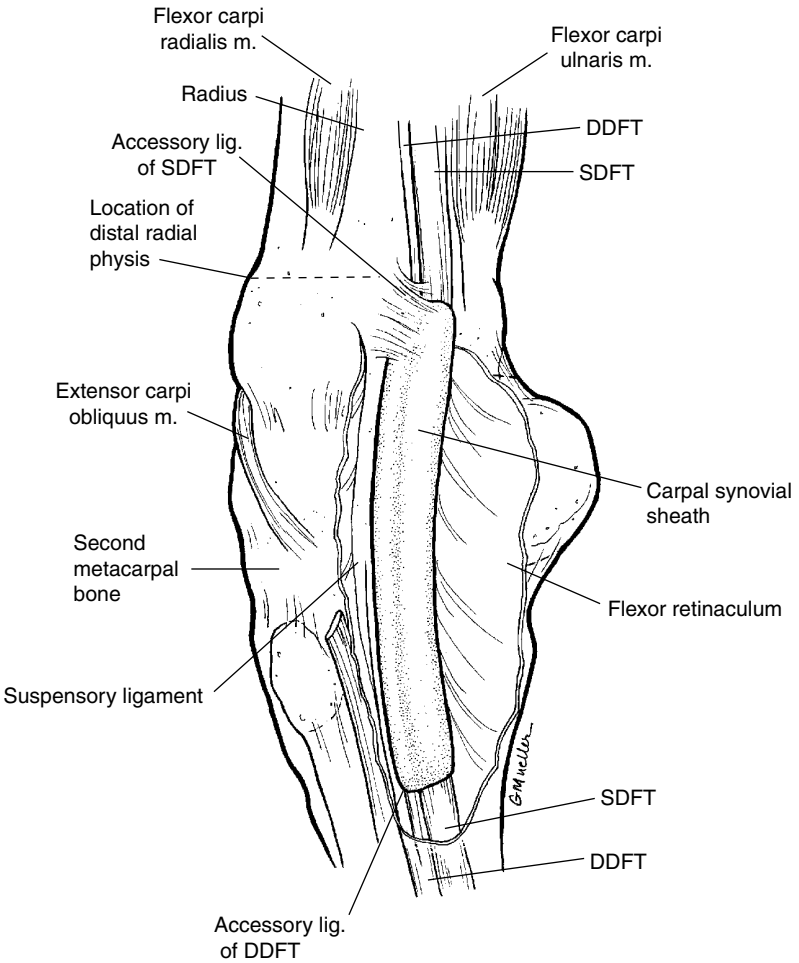


Figure 1.25. Palmaromedial view of carpus with flexor retinaculum cut and reflected. SDFT = superficial digital flexor tendon; DDFT = deep digital flexor tendon.

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synovial spaces of the more distal carpal joints. The middle carpal synovial sac communicates with the small carpometacarpal sac between the third and fourth carpal bones.

The carpus is flexed by the combined action of the flexor carpi radialis, flexor carpi ulnaris, and probably the oddly named extensor carpi ulnaris. It is extended by the extensor carpi radialis and extensor carpi obliquus (a.k.a. abductor digiti I longus) muscles and also by the digital extensors. The thick palmar carpal ligament uniting the palmar aspect of the carpal bones serves to prevent overextension of the antebrachiocarpal and middle carpal joints.

Antebrachium

The antebrachium (forearm) includes the radius and ulna and the muscles, vessels, nerves, and skin surrounding the bones. The prominent muscle belly of the extensor carpi radialis muscle creates the distinctive bulging contour on the cranial antebrachium. A horny cutaneous structure, the chestnut, is present on the medial skin of the distal one-third of the forearm. The chestnut is considered to be a vestige of a carpal pad.[28]

There is extensive overlapping among adjacent sensory cutaneous branches of the axillary, radial, musculocutaneous, and ulnar nerves in the forearm.[5] The axillary nerve gives rise to cutaneous branches to the lateral aspect of the brachium and terminates as the cranial cutaneous antebrachial nerve, which courses distad over the extensor carpi radialis muscle (Figure 1.30).

The lateral cutaneous antebrachial nerve arises from the superficial branch of the radial nerve as the latter runs between the extensor carpi radialis and the lateral head of the triceps brachii (Figure 1.30). In its course the lateral cutaneous antebrachial nerve descends to supply the skin on the craniolateral part of the distal forearm. Terminal branches often course as far as the carpus and proximal metacarpus (Figure 1.20).

The medial cutaneous antebrachial nerve continues distad from the musculocutaneous nerve, coursing over the biceps brachii muscle and then along the deep face of the lacertus fibrosus, and continues into the tendon of the extensor carpi radialis muscle. The nerve is readily palpable through the skin as it crosses the cranial edge and then the medial surface of the lacertus fibrosus, dividing here into two main branches (Figure 1.26). The larger branch accompanies the accessory cephalic vein. The nerve continues on the dorsomedial aspect of the carpus and metacarpus to the fetlock. The smaller branch runs briefly with the cephalic vein and then courses obliquely across the medial surface of the radius where the bone is subcutaneous. This branch of the nerve is sensory to the skin as far as the medial carpus.

Ascending over the transverse pectoral muscle, the cephalic vein runs in the groove between the descending pectoral and cleidobrachialis muscles. A small artery accompanies the cephalic vein in the groove. Under cover of the cutaneous colli muscle, the cephalic vein empties into the external jugular vein or occasionally into the subclavian vein. The more cranial accessory cephalic vein joins the cephalic vein close to the point at which the latter gives rise to the median cubital vein (Figure 1.26). The median cubital courses proximocaudad over the insertion of the biceps brachii and then passes over the median nerve and brachial artery to join the brachial vein in the distal part of the brachium. Midway in its course the median cubital vein may receive a large branch emerging from between the radius and the flexor carpi radialis muscle.

The caudal cutaneous antebrachial nerve (from the ulnar nerve) emerges through the pectoralis transversus to innervate the skin on the caudal aspect of the forearm.

Fascia

The deep antebrachial fascia is especially thick. It invests all of the muscles of the forearm and provides for attachment of the tensor fasciae antebrachii muscle medially, the cleidobrachialis muscle laterally, and the biceps brachii muscle cranially (the latter by means of the lacertus fibrosus). The deep fascia blends with the periosteum on the medial radius and with the collateral ligaments and bony prominences at the elbow. Intermuscular septa from the deep fascia extend between the bellies of the antebrachial muscles.

Extensor Muscles

The extensor carpi radialis is the largest of the extensor muscles of the antebrachium. It arises proximally on the lateral epicondyle and radial fossa of the humerus (in common with the tendon of origin of the common digital extensor) and is attached to the elbow joint capsule and the deep fascia in this region. The tendon of the extensor carpi radialis runs nearly the length of the muscle, becoming visible on its cranial surface about halfway on the antebrachium and completely replacing the fleshy muscle bellow at the junction of the middle and distal thirds.

The extensor carpi obliquus muscle originates broadly on the lateral side of the distal radius. Its muscle belly is at first deep to the common digital extensor; then its tendon crosses that of the extensor carpi radialis superficially as it angles distad over the carpus toward its insertion on the base of the medial splint bone. On the medial aspect of the carpus, a bursa underlies the tendon; it usually communicates with the tendon's synovial sheath in older horses (Figure 1.20).[30]

The common digital extensor muscle takes origin along with the extensor carpi radialis on the lateral epicondyle and radial fossa of the humerus, with additional attachments to the ulna, deep fascia, lateral aspect of the radius, and the lateral collateral ligament of the elbow. Its tendon of insertion, enclosed in its tendon sheath, occupies its respective groove on the distal extremity of the radius. A slender tendon from the very small radial head of the muscle accompanies the main tendon as the two tendons enter the synovial sheath proximal to the carpus. Distal to the carpus, it diverges laterad to unite with the tendon of the lateral digital extensor.

The lateral digital extensor originates from the radius, ulna, and lateral collateral ligament of the elbow joint, and the intermuscular septum from the deep fascia. It is a relatively weak muscle whose muscle belly lies adjacent to the radius and ulna between the extensor carpi ulnaris caudally and the larger common digital extensor muscle belly cranially (Figure 1.26). The muscle's tendon crosses

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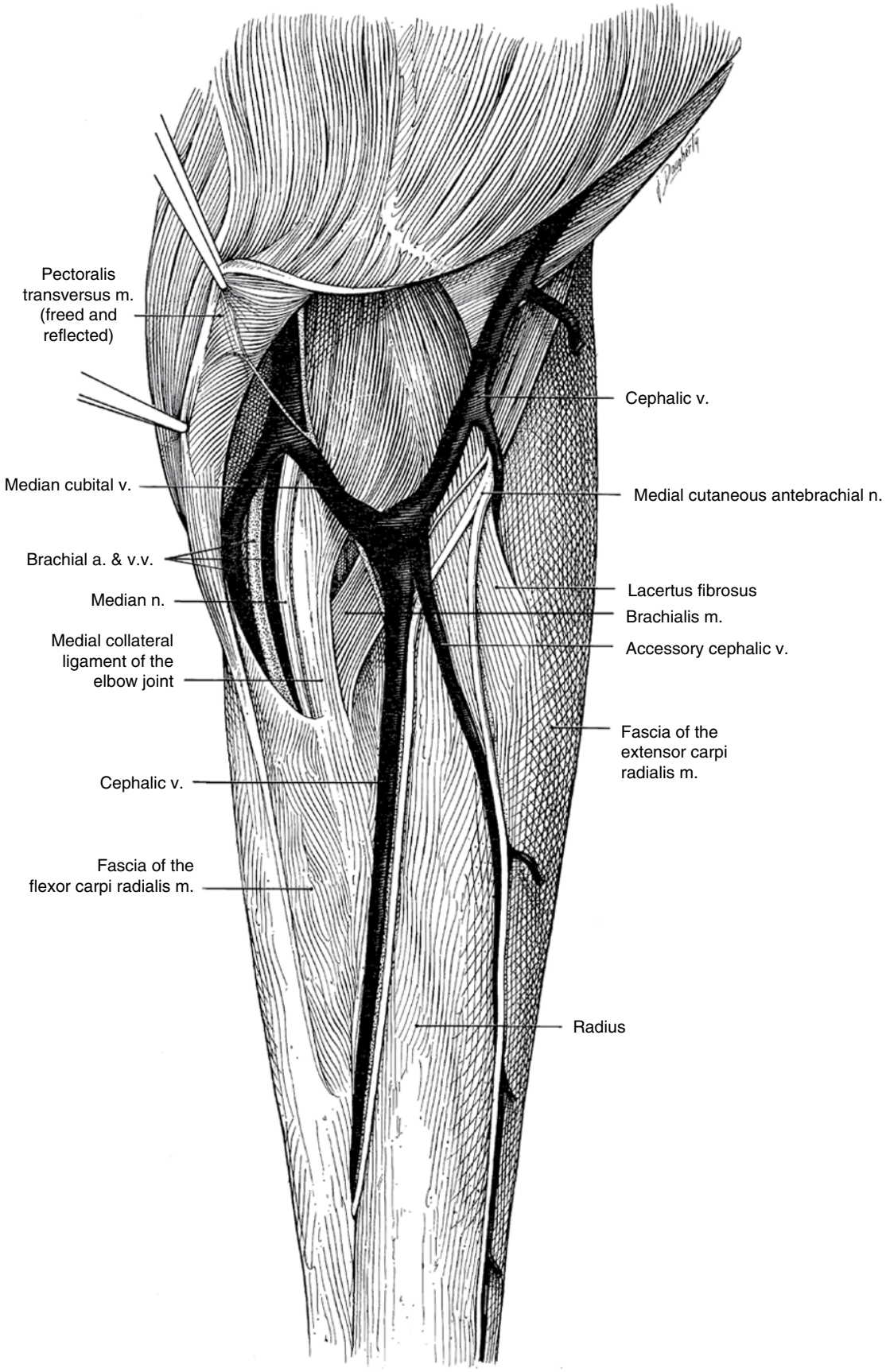


Figure 1.26. Craniomedial view of a superficial dissection of left elbow and forearm.

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the carpus surrounded by a tendon sheath within a fascial canal on its way to the proximal phalanx where it inserts.

Flexor Muscles

The flexor carpi radialis muscle is related to the mediocaudal surface of the radius (Figure 1.26), extending from the medial epicondyle of the humerus to the base of the medial splint bone. Caudal to the flexor carpi radialis, the flexor carpi ulnaris muscle consists of an ulnar head from the olecranon and a humeral head from the medial epicondyle; it inserts on to the accessory carpal bone.

Between flexor carpi ulnaris caudally and the lateral digital extensor laterally is the extensor carpi ulnaris muscle (formerly ulnaris lateralis) that originates on the lateral epicondyle of the humerus caudal to the lateral collateral ligament of elbow joint. A bursa lies deep to the muscle at this location; it often communicates with the elbow synovial joint.[30] The muscle extends distad to insert on the accessory carpal bone and, by means of a somewhat longer, sheathed tendon, to the proximal end of the fourth metacarpal bone.

The preceding three muscles flex the carpal joint and, through their humeral origins, extend the elbow joint. Although the extensor carpi ulnaris is morphologically in the extensor group and is supplied by the radial nerve, its attachments make it a flexor of the carpus.

The superficial digital flexor muscle has a single humeral head, which originates from the medial epicondyle of the humerus and then lies deep to the flexor carpi ulnaris and ulnar head of the deep digital flexor (which is quite superficial as it originates from the medial surface of the olecranon). The muscle belly of the superficial digital flexor lies flat against the deep digital flexor muscle. Under the proximal part of the flexor retinaculum, the tendon of the humeral head of the superficial digital flexor is joined by a flat, wide fibrous band, its accessory ligament (really a radial head of the muscle), which arises on a ridge on the mediocaudal surface of the distal half of the radius (Figure 1.32, later in text).

The deep digital flexor muscle has one large humeral head and two small heads originating from the ulna (olecranon) and radius. The long slender tendon of the ulnar head joins the main tendon of the humeral head proximal to the carpus just before the combined tendon becomes enclosed within the carpal synovial sheath alongside the SDFT. The tendon of the radial head also joins the main tendon near this level. The radial head is inconsistently identifiable; when present, it takes origin from the middle third of the caudal radius. A synovial pouch from the elbow joint capsule protrudes distad beneath the humeral head's origin on the medial epicondyle of the humerus.

Nerves and Deep Vessels

The deep branch of the radial nerve descends over the flexor surface of the elbow and supplies motor branches to the extensor muscles of the forearm and the extensor carpi ulnaris.

The ulnar nerve crosses the medial epicondyle of the humerus accompanied by the collateral ulnar artery and vein, descends obliquely across the medial head of the triceps brachii and the elbow, and then runs between the ulnar heads of the flexor carpi ulnaris and deep digital flexor. It supplies these two muscles and the superficial digital flexor. From here the nerve runs under the deep antebrachial fascia, passing between the superficial digital flexor and the extensor carpi ulnaris and finally between the latter and flexor carpi ulnaris muscles as they near their insertions. A few centimeters proximal to the accessory carpal bone, the ulnar nerve divides into palmar and dorsal branches.

Distal to the elbow the median nerve runs along the caudal border of the medial collateral ligament of the elbow joint, cranial to brachial veins and artery (Figure 1.26). In the proximal part of the forearm, the median nerve supplies branches to the flexor carpi radialis muscle, the humeral and radial heads of the deep digital flexor muscle, and the periosteum of the radius and ulna. At about the middle of the forearm, the median nerve divides into the medial and lateral palmar nerves that remain together in a common sheath before separating close to the carpus. The medial palmar nerve descends in the carpal canal; the lateral palmar nerve is joined by the palmar branch of the ulnar nerve and descends within the dense connective tissue of the flexor retinaculum (Figure 1.18).

Just distal to the elbow joint, the common interosseous artery, a branch of the brachial artery, gives off a small caudal interosseous artery and then, continuing as the cranial interosseous, passes through the interosseous space, supplying nutrient arteries to the radius and ulna. Together with the transverse cubital artery (arising proximal to the elbow joint), the cranial interosseous provides branches to the cranial and medial aspects of the forearm.

After giving off the common interosseous artery, the brachial artery continues as the median artery between the flexor carpi radialis and the radius. In the distal part of the forearm, the median artery angles caudad and gives rise to the proximal radial artery, a small vessel that courses to the palmar aspect of the carpus. The median artery terminates at the distal end of the forearm by trifurcating into the large medial palmar artery, the much smaller lateral palmar artery, and, medially, the radial artery (Figure 1.18).

Two median veins accompany the median artery and nerve: a proximal continuation of the lateral palmar vein, which ascends caudal to the artery, and a vein formed by branches from the caudal antebrachial muscles, which ascends cranial to the artery.

Elbow Joint

The two principle flexors of the equine elbow (cubital) joint are the biceps brachii and the brachialis, with lesser contributions made by the extensor carpi radialis and common digital extensor muscles. The principal extensors are the tensor fasciae antebrachii, triceps brachii, and the anconeus; these are assisted by the flexors of the carpus and digit.

The terminal part of the biceps brachii muscle crosses the joint cranially and inserts on the radial tuberosity and medial collateral ligament. This tendon of insertion also gives rise to the lacertus fibrosus, which blends into the deep fascia of the extensor carpi radialis (Figure 1.27).

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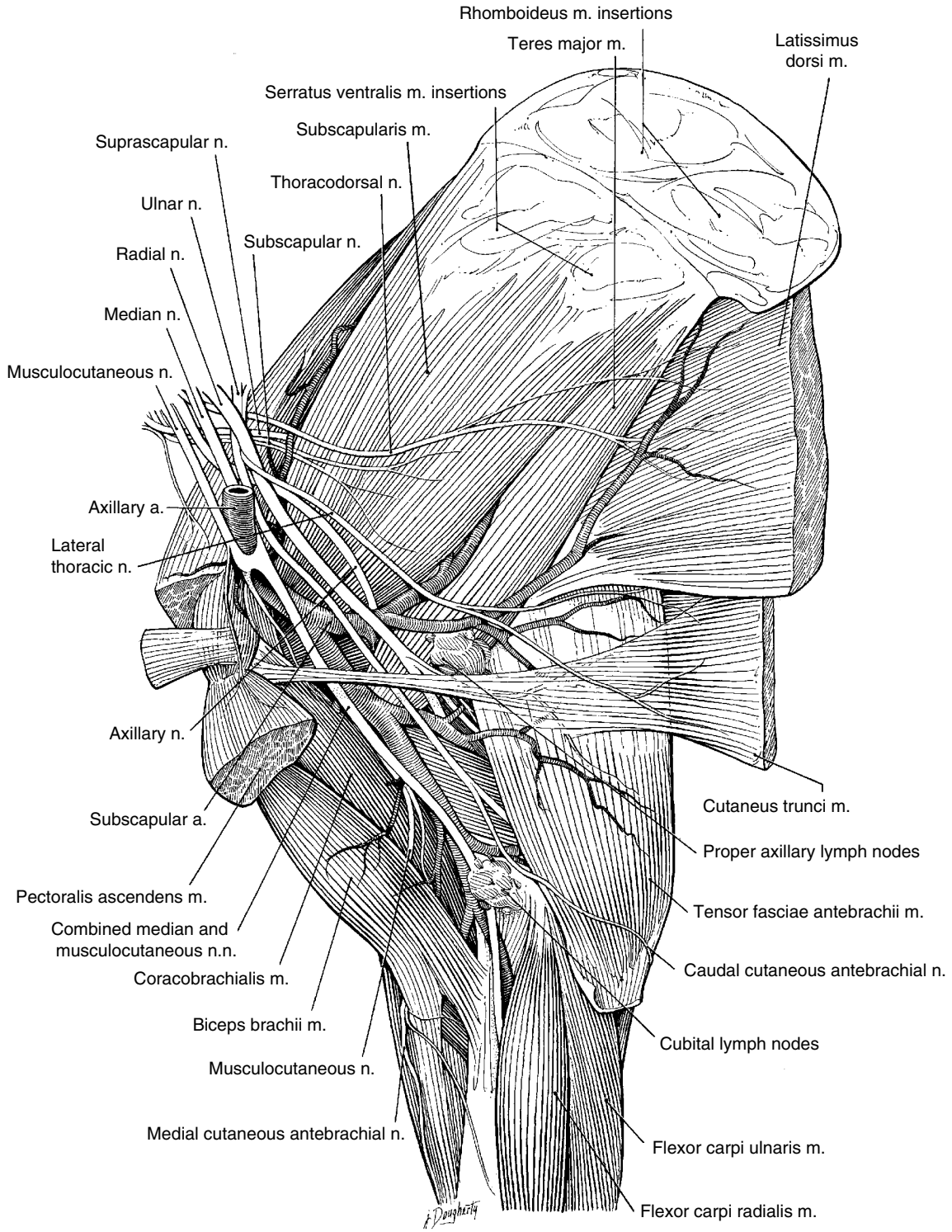


Figure 1.27. Medial view of left shoulder, arm, and proximal forearm. Veins are not depicted.

The terminal part of the brachialis muscle, curving around from its location in the musculospiral groove of the humerus, passes between the biceps brachii and extensor carpi radialis muscles to attach to the medial border of the radius deep to the medial collateral ligament (Figure 1.26). A bursa lies between the tendon and the collateral ligament.[30]

The median nerve, brachial artery, and the two brachial veins lie caudal to the medial collateral ligament of the elbow joint deep to the cranial part of the transverse pectoral muscle (Figure 1.26). Proximocaudal to the joint the collateral ulnar artery and vein and the ulnar nerve with its cutaneous branch (caudal cutaneous antebrachial nerve) cross obliquely between the medial head of the triceps brachii and tensor fasciae antebrachii.

All three principal extensors of the elbow insert on the olecranon tuberosity. A subcutaneous bursa may cover the caudal aspect of the olecranon tuberosity; a

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subtendinous bursa lies deep to the tendon of insertion of the long head of the triceps brachii muscle[30] (Figure 1.28). The medially located tensor fasciae antebrachii also inserts on and acts to tense the deep antebrachial fascia. The small anconeus muscle originates from the lateral edge of the olecranon fossa, inserts on the olecranon, and is attached on its deep surface to the elbow joint capsule, acting to elevate it when the joint is extended.

Laterally the elbow is covered by the distal part of the cutaneous omobrachialis muscle. A short, stout lateral collateral ligament extends from the lateral epicondyle of the humerus to the lateral tuberosity of the radius. The cranial part of the joint capsule is robust, but caudally it becomes thinner as it extends into the olecranon fossa deep to the anconeus muscle. The joint capsule is adherent to the anconeus muscle, the collateral ligaments, and tendons of surrounding muscles. Extensions

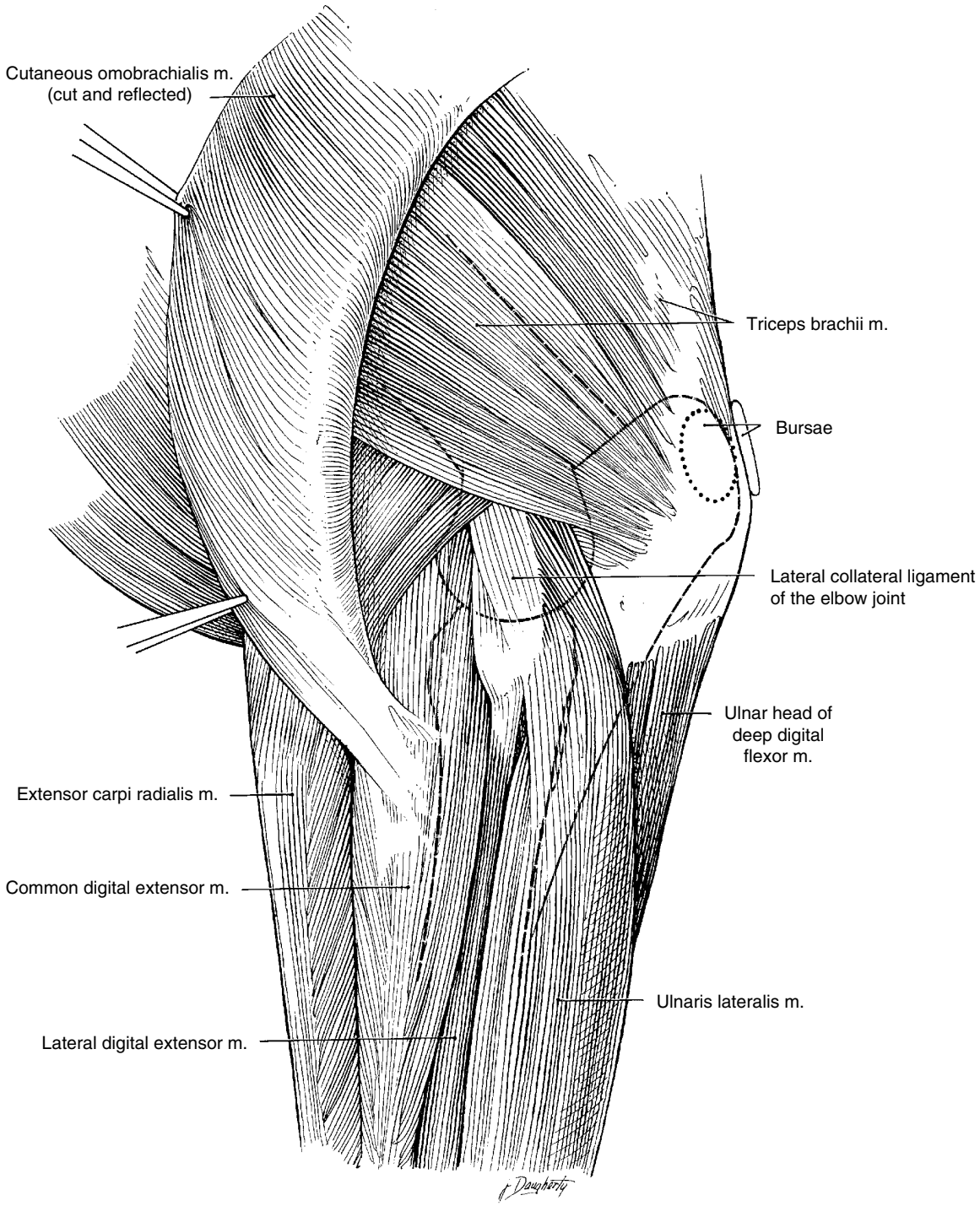


Figure 1.28. Lateral view of left elbow. Dashed lines represent the locations of bony elements. Note that the ulnaris lateralis is now called extensor carpi ulnaris.

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of the synovial lining of the joint project under the origins of the extensor carpi ulnaris and the digital flexor muscles and into the radioulnar articulation. The elbow joint is supplied by branches from the transverse cubital artery cranially and a branch from the collateral ulnar artery caudally.

The articular surface of the radius features a concave fovea, partially subdivided by two sagittal ridges. This surface plus the trochlear notch of the ulna articulates with the humerus, forming a hinge joint. The articular angle can range from approximately 60° to 150°.[16]

Arm and Shoulder

The "arm" (*L. brachium*) is the region of the limb between the elbow and shoulder (scapulohumeral) joints. Within the superficial fascia over the lateral aspect of the shoulder and arm, the cutaneous omobrachialis muscle arises in the region lateral to the scapula and extends as far distal as the elbow joint (Figure 1.28). The cutaneous muscle is innervated by the intercostobrachial nerve (a branch of the lateral thoracic nerve). The intercostobrachial nerve and branches of the axillary, radial, and spinal nerves provide cutaneous sensation to this region. Superficial blood vessels are branches of the caudal circumflex humeral vessels.

The cleidobrachialis muscle (distal part of the brachiocephalicus) covers the craniolateral aspect of the shoulder joint on the way to its insertion on the deltoid tuberosity, humeral crest, and the fascia of the arm (Figure 1.29). When the head and neck are fixed and the limb is free to swing, this muscle acts as an extensor of the shoulder joint, drawing the forelimb forward. With the limb fixed on the ground, its contraction draws the head and neck into lateral flexion.

Muscles Substituting for Shoulder Joint Ligaments

Cranially, the dense, partly cartilaginous tendon of the biceps brachii muscle originates on the supraglenoid tubercle of the scapula and occupies the intertubercular groove of the humerus. A connective tissue band extends from the lesser tubercle to the greater tubercle, serving as a retinaculum for the biceps tendon. A bursa lies under the tendon, partially extending around its sides. A tendinous band (an "internal tendon") is continuous from the tendon of origin distad through the muscle. In addition to the muscle's primary function of elbow flexion, the internal tendon of the biceps brachii passively fixes the elbow and shoulder in the standing position. The musculocutaneous nerve supplies the biceps brachii.

The supraspinatus muscle arises in the supraspinous fossa, the spine, and cartilage of the scapula. It divides distally to attach to the greater and lesser tubercles of the humerus, serving with the bicipital tendon to stabilize the shoulder joint cranially and to a smaller degree laterally (Figure 1.30). The supraspinatus is a shoulder extensor; it is innervated by the suprascapular nerve, which reaches the supraspinous fossa by coursing between the subscapularis and supraspinatus muscles and then wrapping around the distal fourth of the cranial border of the scapula.

The infraspinatus muscle extends from the scapular cartilage and infraspinous fossa to insert on the caudal and distal parts of the greater tubercle, distal to the insertion of the supraspinatus (Figure 1.30). The partly cartilaginous tendon is protected from the underlying caudal eminence by adipose tissue and a synovial bursa that may communicate with the shoulder joint cavity. The tendon is the main lateral support of the shoulder joint. It is assisted by the teres minor, a smaller muscle deep to the infraspinatus and arising from the infraspinous fossa, the caudal border, and a small tubercle on the distal end of the scapula. It inserts proximal to and on the deltoid tuberosity. The teres minor muscle flexes the joint and, together with the infraspinatus, abducts the arm. The infraspinatus also rotates the arm laterad. The infraspinatus muscle is supplied by the suprascapular nerve, and the teres minor by the axillary nerve.

The subscapularis muscle stabilizes the shoulder joint medially. This adductor of the arm originates in the subscapular fossa of the scapula and inserts on the lesser tubercle of the humerus. Caudal support to the joint is rendered by the long head of the triceps brachii, the only head of this muscle originating from the scapula.

Flexor Muscles of the Shoulder Joint

In addition to the long head of the triceps brachii muscle, four muscles flex the shoulder joint: laterally, the deltoideus and teres minor; medially, the teres major and coracobrachialis; and the latissimus dorsi. The first three muscles are innervated by branches from the axillary nerve; the coracobrachialis, by the musculocutaneous nerve; and the latissimus dorsi, by the thoracodorsal nerve.

The deltoideus lies on the lateral surface of the triceps brachii and partly on the infraspinatus and teres minor muscles. It originates from the caudal border of the scapula and the scapular spine via the aponeurosis covering the infraspinatus and inserts on the deltoid tuberosity of the humerus and the brachial fascia (Figure 1.30).

The teres major muscle extends from the caudal border of the scapula and crosses the medial surface of the triceps brachii to the teres major tuberosity of the humerus, where it inserts in common with the latissimus dorsi muscle (Figure 1.27).

The coracoid process of the scapula gives origin to the coracobrachialis muscle that crosses the medial aspect of the shoulder joint to attach to the humerus just proximal to the teres major tuberosity and on the middle of the cranial surface of the bone. A bursa lies between the tendon of origin of the coracobrachialis and the tendon of insertion of the subscapularis muscle.[30]

Shoulder Joint

The fibrous joint capsule of the shoulder joint attaches up to 2 cm from the margins of the articular surfaces. Two elastic glenohumeral ligaments reinforce the joint capsule on its cranial side as they diverge from the supraglenoid tubercle to the humeral tuberosities. A very small articularis humeri muscle lies on the flexion surface of the joint capsule, extending from the caudal part of the scapula to the caudal surface of the humerus just distal to the head. Innervated by the axillary nerve, the articularis humeri muscle tenses the joint capsule during flexion of the shoulder joint.

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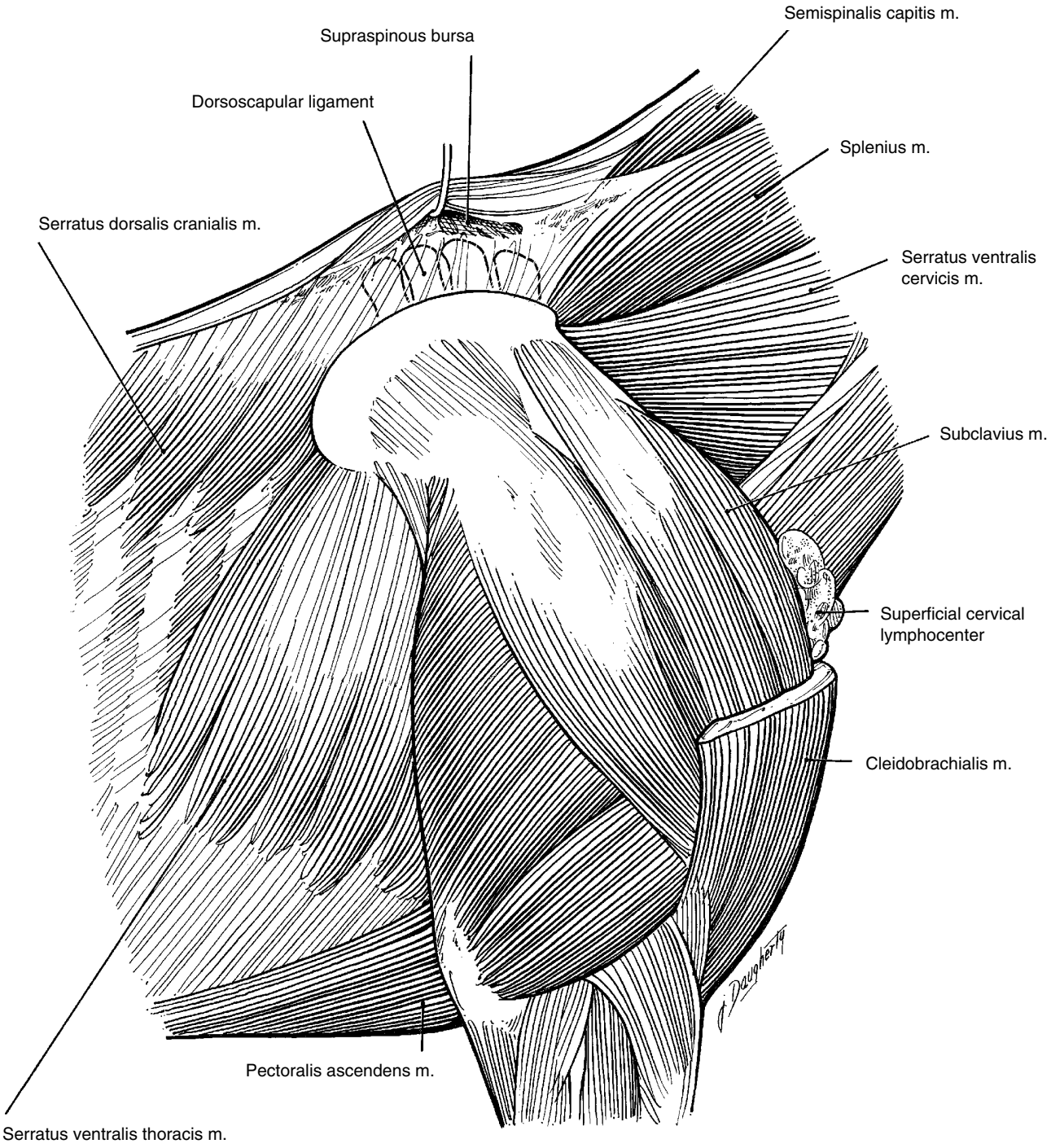


Figure 1.29. Right shoulder and dorsoscapular ligament. Spines of thoracic vertebrae 2–5 are outlined by dashed lines.

Within the shoulder joint the articular surface of the humeral head has approximately twice the area of the glenoid cavity of the scapula even with the small extension afforded by the glenoid lip around the rim. The great stability of this joint is largely owed to the support of the surrounding muscles. In spite of its classification as a ball-and-socket joint, major movements are limited to flexion and extension; muscles around the joint restrict abduction and adduction, and rotation is very limited. While standing, the caudal angle of the shoulder joint is 120°–130°. The angle can increase to approximately 145° in extension and decrease to 80° in flexion.[16]

Muscles of the Scapula

In the scapular region the flat, triangular trapezius muscle covers parts of eight underlying muscles. The cervical part of the trapezius arises by a thin aponeurosis from most of the funicular part of the nuchal ligament and inserts on the scapular spine and fascia of the shoulder and arm. The thoracic part of the trapezius takes an

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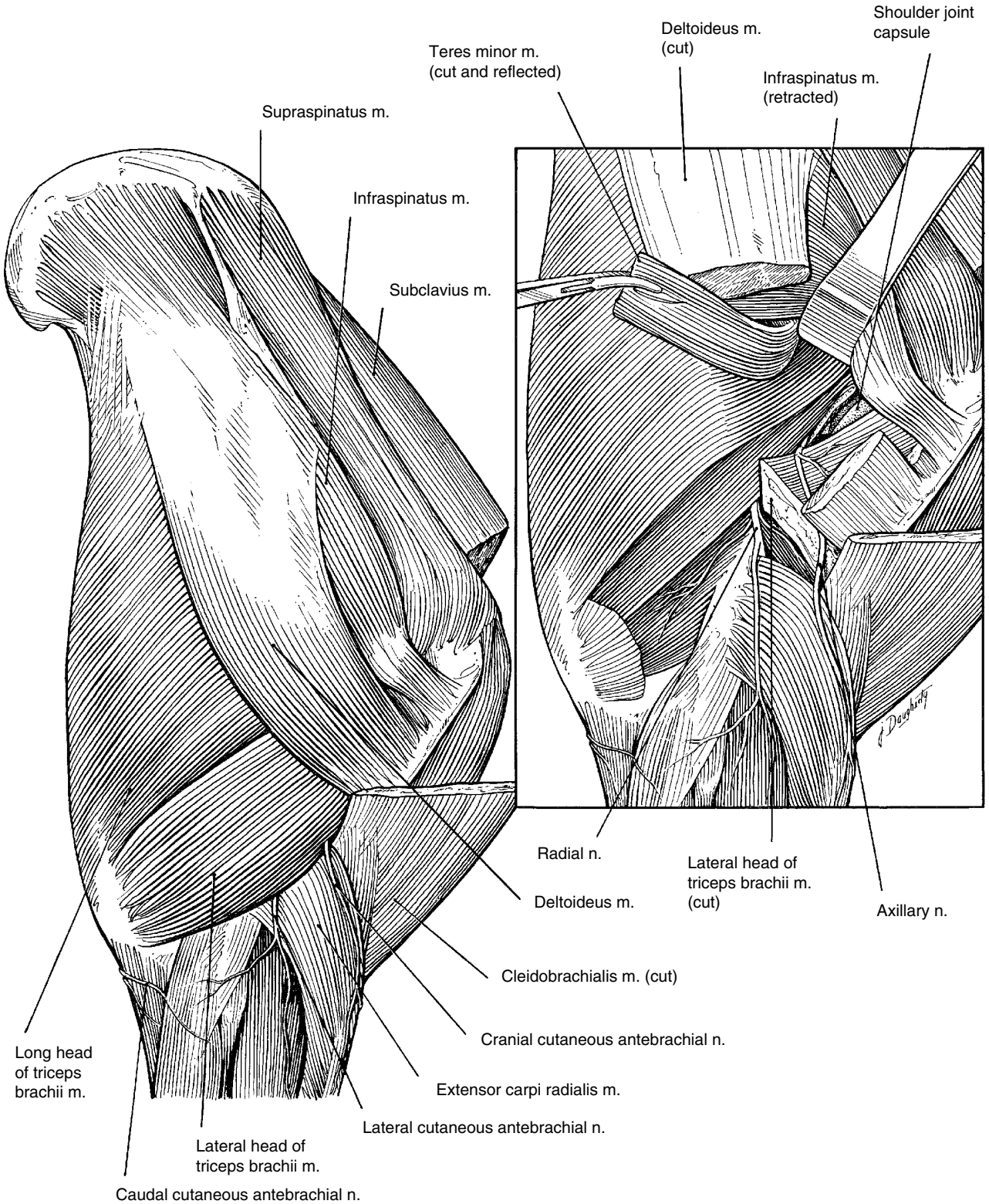


Figure 1.30. Lateral aspect of right shoulder. Inset: Deeper dissection exposing shoulder joint.

aponeurotic origin from the supraspinous ligament from the third to the tenth thoracic vertebrae and inserts on the tuber of the spine of the scapula. An aponeurosis joins the two parts of the trapezius. Innervated by the accessory nerve and dorsal branches of adjacent thoracic nerves, the trapezius muscle elevates the shoulder and draws it either craniad or caudad, depending on the activity of the cervical or thoracic parts, respectively.

Like the trapezius, the rhomboideus possesses cervical and thoracic parts. Lying deep to the trapezius, the rhomboideus cervicis originates from the funicular part of ligamentum nuchae; the rhomboideus thoracis originates

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from the superficial surface of the dorsal part of the dorsoscapular ligament. Both parts of the rhomboideus muscle insert on the medial side of the scapular cartilage (Figure 1.27). This muscle is innervated by the sixth and seventh cervical nerves and dorsal branches of nerves adjacent to the rhomboideus thoracis. The rhomboideus draws the scapula dorsocraniad, and, when the limb is stationary, the cervical part helps to raise the neck.

The latissimus dorsi muscle has a triangular shape with the origin arising through a broad aponeurosis from the thoracolumbar fascia. Thin near its aponeurotic origin, the muscle becomes thicker as it approaches its insertion, passing medial to the long head of the triceps brachii to converge on a flat, common tendon of insertion with the teres major muscle on the teres major tuberosity of the humerus (Figure 1.27).

From deep to superficial, the muscles contributing most substantially to the attachment of the thoracic limb to the trunk and neck are the serratus ventralis, pectoral muscles (including subclavius), brachiocephalicus, and omotransversarius. The cervical part of serratus ventralis extends from the transverse processes of the cervical vertebrae C4–C7 to the serrated face of the medial scapula and scapular cartilage; the thoracic part of serratus ventralis converges from the lateral surfaces of the first eight or nine ribs to the serrated face of the scapula and the scapular cartilage. Elastic sheets from the ventral part of the dorsoscapular ligament (Figure 1.31) are interspersed through the attachments of the serratus ventralis on the scapula. The right and left serratus ventralis muscles form a muscular sling suspending the thorax between the thoracic limbs. When both muscles contract, they elevate the thorax; acting independently, each serratus ventralis shifts the trunk's weight to the ipsilateral limb. During locomotion the cervical part of the muscle draws the dorsal border of the scapula craniad; the thoracic part draws the scapula caudad. When the limb is fixed, the cervical portion lifts the neck dorsad and/or pulls it laterad. The long thoracic nerve and branches from the fifth to the eighth cervical nerves supply this muscle.

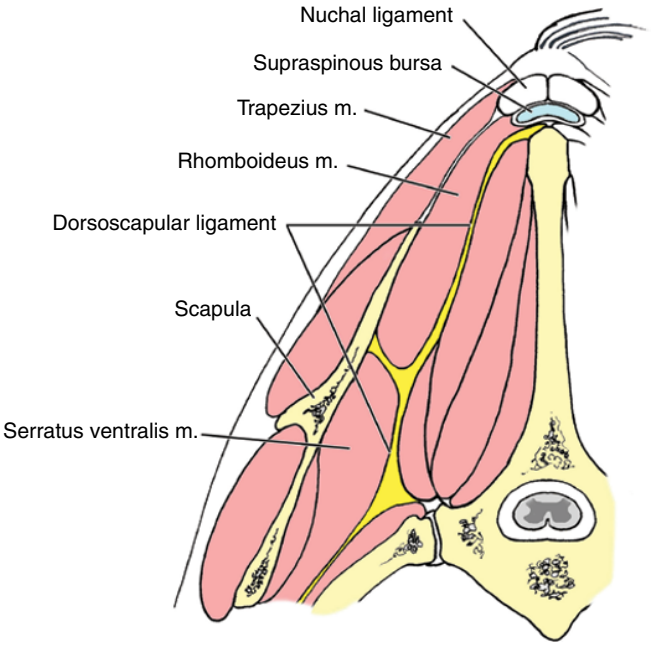


Figure 1.31. Cross section of right dorsoscapular ligament. The ligament covers the medial and ventral aspects of the rhomboideus and gives rise to sheets of elastic tissue that interdigitate with the fascicles of the serratus ventralis.

Pectoral muscles attach to the sternum. There are two superficial pectoral muscles: (1) the descending pectoral muscle passing from the cartilage of the manubrium of the sternum to the deltoid tuberosity and the crest of the humerus and brachial fascia and (2) the transverse pectoral muscle extending from the ventral part of the sternum to the superficial fascia on the medial aspect of the antebrachium and to the humeral crest. The largest pectoral muscle, the deep pectoral (ascending pectoral) muscle (Figure 1.29), passes from its attachments on the xiphoid cartilage, the ventral part of the sternum, the fourth to ninth costal cartilages, and the abdominal tunic to the cranial parts of the lesser and greater humeral tubercles. The subclavius has been traditionally grouped with the pectorals. It arises from the first four costal cartilages and the cranial half of the sternum and ends in an aponeurosis over the dorsal part of the supraspinatus muscle and the scapular fascia (Figure 1.29).

The superficial pectoral muscles adduct the limb. The deep pectoral and subclavius are also adductors, and, if the limb is fixed in the advanced position, they pull the trunk craniad. Cranial and caudal pectoral nerves (with musculocutaneous and intercostal nerves contributing to the cranial pectoral nerves) supply these muscles.

The brachiocephalicus is traditionally divided into two parts based on the embryological location (not apparent in the fully formed muscle) of the clavicular intersection. The cleidobrachialis part of the brachiocephalicus muscle extends from the clavicular intersection to the arm and is innervated by the axillary nerve. The mastoid part of the muscle (a.k.a. cleidomastoideus) lies between the intersection and its attachments to the mastoid process and nuchal crest of the skull, partly overlapping the omotransversarius muscle dorsally. The omotransversarius originates from the wing of the atlas and the transverse processes of the second, third, and fourth cervical vertebrae and inserts on the humeral crest and fascia of the shoulder and arm. The cleidobrachialis is innervated by the accessory nerve (cranial nerve XI), and the omotransversarius is innervated segmentally by cervical spinal nerves.

Dorsoscapular Ligament

Further attachment of the limb to the trunk is afforded by a thickened specialization of the thoracolumbar fascia, the dorsoscapular ligament (Figure 1.31). Its collagenous portion attaches to the third, fourth, and fifth thoracic spines under the nuchal ligament close to the supraspinous bursa.[15] This part of the dorsoscapular ligament passes ventrad, attaching to the medial surface of the rhomboideus thoracis muscle. As it curves ventral to the muscle, the collagenous part transitions to elastic tissue. A horizontal lamina of this elastic part forms the ventral sheath of the rhomboideus thoracis muscle. Vertical laminae project from the ventral aspect of the horizontal lamina, investing bundles of the serratus ventralis muscle that insert on the scapula (Figure 1.31).

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Nerves and Vessels

The ventral branches of spinal nerves C6 through T2 create the brachial plexus, which perforates the scalenus muscle and passes into the forelimb craniomedial to the shoulder joint (Figure 1.27). Vessels supplying the thoracic limb accompany the nerves in this location.

Suprascapular vessels accompany the suprascapular nerve, passing laterad between the subscapular and suprascapular muscles. The median nerve descends with the axillary artery, forming a loop medial to the artery by uniting with a large branch from the musculocutaneous nerve. Proximal branches from the musculocutaneous nerve supply the coracobrachialis and biceps brachii muscles. Distal to the axillary loop, the median and musculocutaneous nerves are contained in a common sheath, coursing distad along with the brachial artery. In the middle of the arm, the musculocutaneous nerve terminates by dividing into a distal branch supplying the brachialis muscle and the medial cutaneous antebrachial nerve that spirals around the biceps brachii to the lacertus fibrosus.

The axillary nerve crosses the medial surface of the subscapularis muscle, and, together with the large subscapular vessels (from the axillary vessels), the nerve passes laterad between the subscapularis and teres major muscles. Innervating these muscles and others of the caudal shoulder, the axillary nerve is accompanied by the caudal circumflex humeral artery, a branch of the subscapular artery. It ultimately gives rise to the cranial cutaneous antebrachial nerve.

The large radial nerve and smaller ulnar nerve descend close to each other medial to the subscapular artery and then caudal to the brachial vein. After supplying a branch to the tensor fasciae antebrachii muscle, the radial nerve plunges laterad between the teres major and the triceps to run along the musculospiral groove of the humerus. Here it gives off lateral cutaneous branches to the caudodistal aspect of the arm and supplies branches to the triceps brachii and anconeus muscles. Just proximal to the elbow joint, the radial nerve divides into deep and superficial branches. The deep branch supplies the craniolateral muscles of the antebrachium. The superficial branch winds laterad between the lateral head of the triceps brachii and the extensor carpi radialis muscles accompanied by the transverse cubital artery. It gives rise to the lateral cutaneous antebrachial nerve, which supplies sensory innervation to the fascia and skin of the lateral aspect of the forearm (Figure 1.30).

The ulnar nerve angles caudodistad to the middle of the arm. It gives rise to the caudal cutaneous antebrachial nerve that courses across the medial surface of the tensor fasciae antebrachii (Figure 1.27). The continuation of the ulnar nerve passes between that muscle and the medial head of the triceps brachii, accompanied here by the collateral ulnar vessels as they cross the medial humeral epicondyle.

After giving off the cranial circumflex humeral vessels in the proximal part of the brachium, the axillary vessels continue as the brachial artery and vein. As they descend the arm they give rise to the deep brachial vessels supplying the triceps muscle and then the collateral ulnar vessels caudally and the bicipital vessels cranially. The transverse cubital vessels are given off cranially and pass distolaterad deep to the biceps brachii and brachialis muscles to the cranial aspect of the cubital joint. The nutrient artery of the humerus may come from the first part of the collateral ulnar artery, or it may arise from the brachial artery.

Lymphatic Drainage

Lymphatic vessels from structures distal to the elbow are afferent to the cubital lymph nodes, a group of 5–20 small nodes found just proximal to the medial side of the elbow (Figure 1.27). Efferent vessels from the cubital lymph nodes end in the proper axillary lymph nodes on the medial surface of the teres major muscle (Figure 1.27). These also receive lymph from the muscles of the arm and shoulder and from the adjacent skin and the ventrolateral trunk. Vessels from the proper axillary lymph nodes carry lymph to the caudal deep cervical lymph nodes. Some efferents of the deep cervical nodes drain directly into the venous system; others pass to other regional nodes and therefore drain indirectly through these to the venous system.[28]

Lymphatic vessels from the skin of the entire thoracic limb, neck, and dorsolateral trunk and more proximal parts of the limb are afferent to the superficial cervical lymphocenter on the cranial border of the subclavius muscle, deep to the brachiocephalicus. Efferent lymphatic vessels from the superficial cervical lymph nodes terminate in the caudal deep cervical lymph nodes or by entering the common jugular vein.[16,28]

Stay Apparatus of the Thoracic Limb

Muscles, tendons, and ligaments constituting the stay apparatus of the thoracic limb fix the carpus, elbow, and shoulder joints in the standing position (Figure 1.32). This complex of structures functions almost entirely as a passive, force-resisting system.[35] It permits the horse to stand (and sleep) with a minimum of muscular activity. The elasticity of the tissues in the stay apparatus also stores energy during weight-bearing and redeliver that energy back to the limb during the swing phase of gait, markedly improving the efficiency of movement, especially when working at speed.

In the manus, elements of the stay apparatus resist nonphysiologic hyperextension of the fetlock and interphalangeal joints.

The DDFT forms a continuous ligamentous band from its accessory (carpal check) ligament to the solar surface of the distal phalanx. This is the only element that resists hyperextension of the distal interphalangeal joint; as a consequence, disruption of this part of the suspensory apparatus can produce a characteristic hyperextension of the coffin joint, identifiable as the foot is rotated back onto the heels and the toe lifts from the ground.

The four palmar ligaments stretched tightly across the pastern joint, the straight distal sesamoidean ligament, and the digital flexor tendons (with their accessory ligaments) prevent hyperextension of the pastern joint.

The suspensory apparatus is a ligamentous continuum extending from the proximal end of the third metacarpal bone to the proximal and middle phalanges. It consists of the suspensory ligament, palmar ligament with its embedded proximal sesamoid bones, and the distal sesamoidean ligaments. The SDFTs and DDFTs

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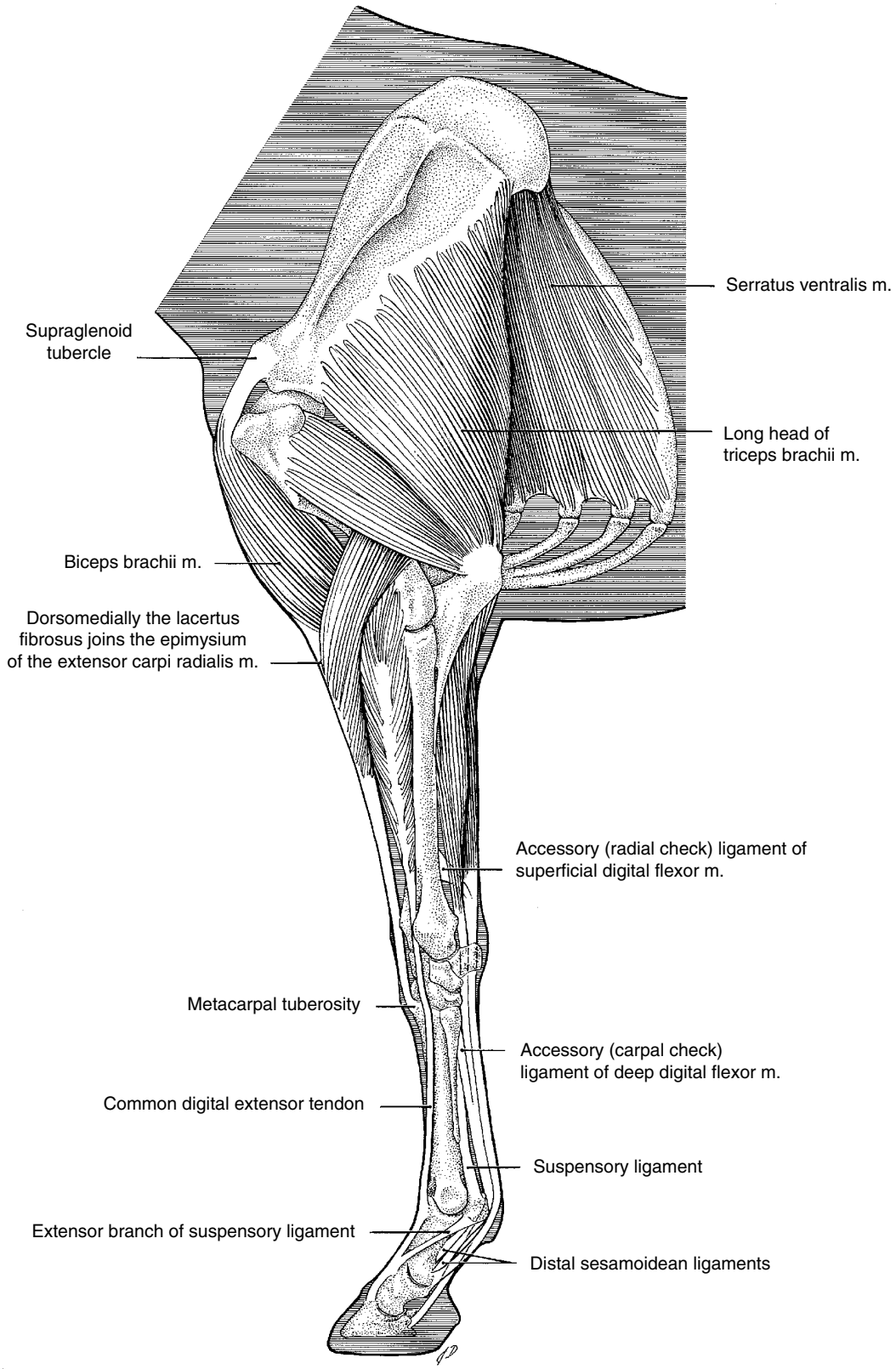


Figure 1.32. The passive stay apparatus of the left thoracic limb with assisting active elements.

and their accessory (check) ligaments assist the suspensory apparatus of the fetlock in suspending the fetlock and preventing excessive overextension of the metacarpophalangeal joint and collapse of the fetlock during loading.[11] Disruption of the suspensory apparatus alters its support of the fetlock, resulting in "sinking" or hyperextension of the fetlock.[35]

The skeletal elements of the carpus exhibit some stability in extension. Soft tissue stabilization is provided by the palmar carpal ligament and the collateral ligaments.

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Palmarly, the digital flexor tendons bridging the carpus in the carpal canal between their respective accessory ligaments, and dorsally the extensor tendons, principally the extensor carpi radialis tendon, lend further stability to the carpus.

A certain amount of muscle tone prevails in all "resting" muscles of the limb, even during most stages of sleep. Tension exerted by the long head of the triceps brachii muscle is essential to prevent flexion of the elbow joint.[33] The elbow's eccentrically placed collateral ligaments afford it surprising stability in the extended position, a stability enhanced by the triceps' tone. Flexion of the joint is further limited by the superficial digital flexor muscle descending from its attachment of the medial epicondyle of the humerus.[11]

A tendinous continuum extending from the supraglenoid tubercle of the scapula to the metacarpal tuberosity is formed by the main tendon of the biceps brachii muscle and its fibrous "internal tendon" and superficial connection to the fascia of extensor carpi radialis muscle via the lacertus fibrosis. Through the tendon of insertion of the extensor carpi radialis, a continuous band is therefore created from the scapula to the metacarpus. This complex prevents flexion of the loaded shoulder joint caused by the weight of the trunk being transferred to the appendicular skeleton via the scapular attachments of the serratus ventralis muscle and the dorsoscapular ligament.

HINDLIMB

Digit and Fetlock

The hindfoot is somewhat smaller and more elongate than the forefoot. Compared with the forefoot, the angle of the toe of the hindfoot is slightly greater (i.e. it is steeper).[1] The middle phalanx is narrower and longer, and the proximal phalanx somewhat shorter than their counterparts in the thoracic limb (Figure 1.33).

The long digital extensor muscle's tendon attaches to the dorsal surfaces of the proximal and middle phalanges and the extensor process of the distal phalanx, but the tendon of the lateral digital extensor usually does not reach the digit as it does in the thoracic limb. Digital flexor tendons, tendon sheaths, and bursae of the hind digit are not remarkably different. The suspensory apparatus of the fetlock and the configuration of the fetlock (metatarsophalangeal) joint are much the same as in the thoracic limb except that the dorsal articular angle of the fetlock is approximately 5° greater (i.e. is slightly more "upright").

Blood Vessels and Nerves of the Hind Digit and Fetlock

The principal blood supply to the fetlock and digit of the pelvic limb is derived from the continuation of dorsal metatarsal artery III, the distal perforating branch, which bifurcates into medial and lateral digital arteries in the distoplantar region of the metatarsus. A small secondary supply is contributed by medial and lateral plantar arteries that join the digital arteries to form the superficial plantar arch just proximal to the widening of the fetlock (Figure 1.34). Branches of the digital arteries form a pattern similar to that in the thoracic limb except for the blood supply to the navicular bone. In contrast to all arteries originating from palmar arterial branches of the middle phalanx, in the pelvic limb, half of the primary arteries originate from the plantar arterial branches of the middle phalanx, and half from the collateral arch (cf. Figure 1.8). More significantly, a greater number of vessels enter the distal border of the navicular bone from the distal anastomotic network in the hindfoot than the same region in the forefoot.[18]

Venous drainage of the digit of the pelvic limb is similar to that of the forelimb. The digital veins carry blood to the plantar common digital veins at the level of the fetlock.

The pattern of distribution of the plantar digital and plantar metatarsal nerves in the fetlock and digit of the pelvic limb is similar to the pattern of the counterpart nerves in the thoracic limb. Some differences exist, however. The dorsal branch of each plantar digital nerve is given off more distally than the corresponding branch of the forelimb. Medial and lateral dorsal metatarsal nerves (from the deep fibular—formerly peroneal—nerve) course distad subcutaneously parallel and dorsal to the medial and lateral plantar metatarsal nerves (Figures 1.34 and 1.35). The lateral plantar metatarsal nerve extends distad over the fetlock to the lateral aspect of the pastern, while the medial plantar metatarsal nerve may reach as far as the coronet; both dorsal metatarsal nerves continue into the laminar corium.[20] Terminal branches of the saphenous nerve medially, the superficial fibular nerve dorsally and laterally, and the caudal cutaneous sural nerve dorsolaterally complete the sensory innervation to the skin of the fetlock.

Metatarsus

The equine metatarsus is about 16% longer than the corresponding metacarpus, and the third metatarsal bone is more round in cross section than the third metacarpal bone.[16] The lateral splint bone, particularly its proximal extremity, is much larger than the medial splint bone.

Dorsal Aspect

Three superficial nerves supply sensory innervation to the skin of the dorsal, lateral, and medial aspects of the metatarsus. Dorsally and laterally the two terminal branches of the superficial fibular nerve descend as far as the fetlock. The terminal branch of the caudal cutaneous sural nerve descends obliquely from the lateral aspect of the hock to course over the dorsolateral part of the third metatarsal bone, terminating in the skin of the fetlock, and the terminal branch of the saphenous nerve supplies medial skin of the metatarsus down to the fetlock.

The dorsal common digital vein II (great metatarsal vein) ascends from a venous arch proximal to the proximal sesamoids as the primary continuation of the venous drainage on the medial side of the digit and fetlock. At first, the great metatarsal vein lies along the medial border of the suspensory ligament, and then it angles dorsad in a groove on the third metatarsal bone to the hock where it becomes the cranial branch of the medial saphenous vein (Figure 1.35).

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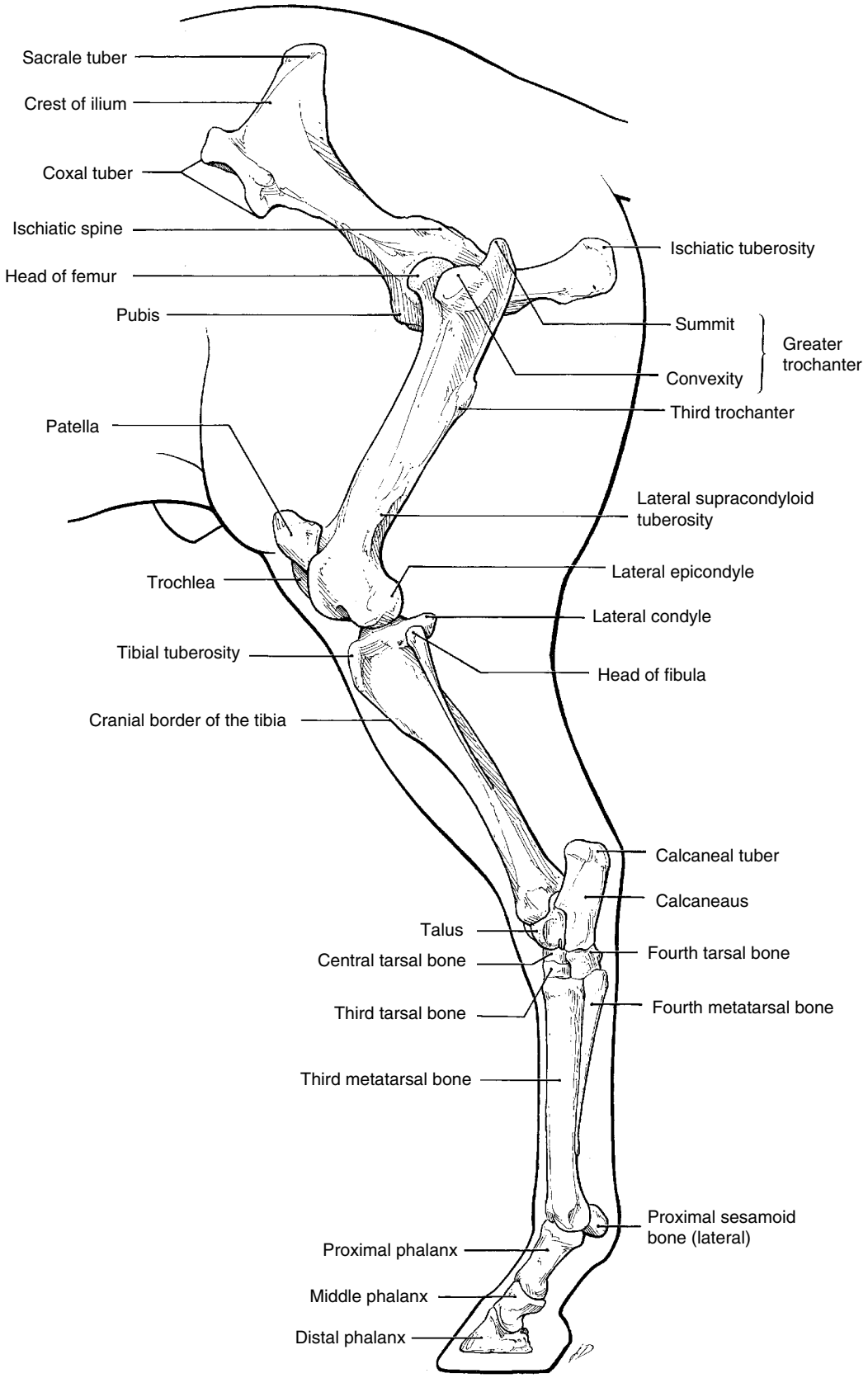


Figure 1.33. Bones of the left pelvic limb. Lateral view.

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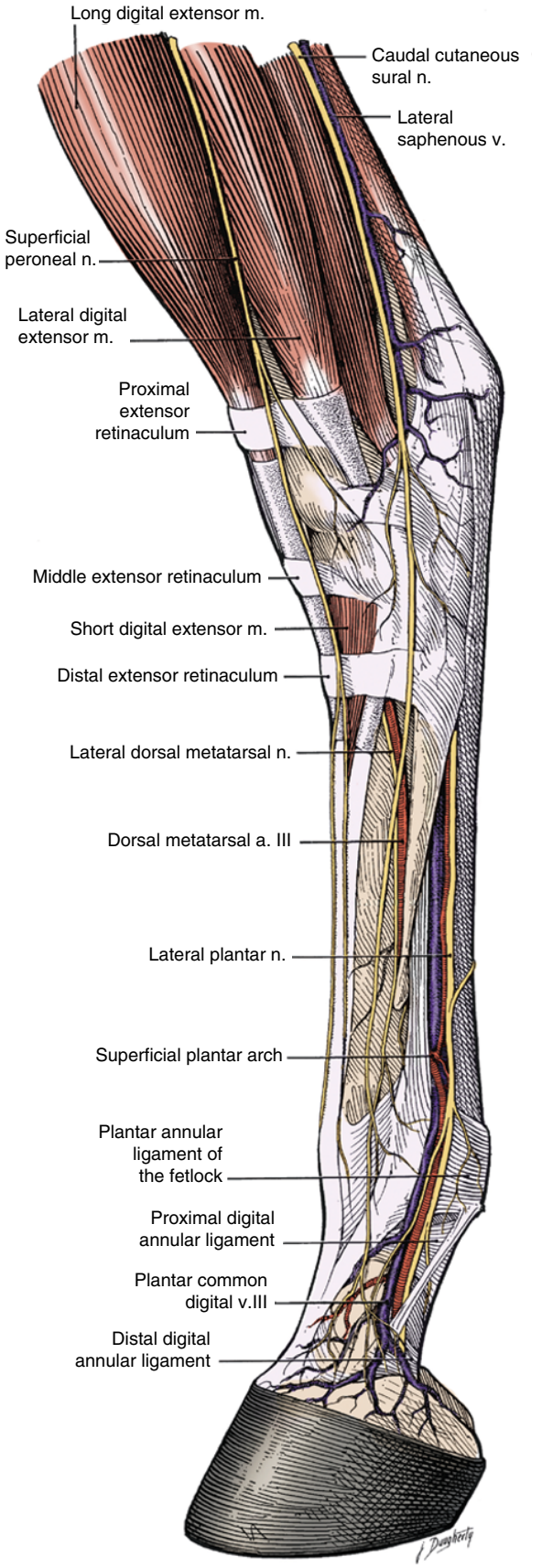


Figure 1.34. Lateral view of left distal crus and pes. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal) although both are widely used.

The tendon of the long digital extensor muscle extends the length of the metatarsus on the dorsal surface of the cannon bone. At the proximal third of the metatarsus, the tendon of the lateral digital extensor muscle joins the long digital extensor tendon. Rarely, the tendon of the lateral digital extensor courses separately to the proximal phalanx. The angle formed by the conjoined long and lateral digital extensor tendons is occupied by the thin, triangular short digital extensor muscle. This vestigial muscle originates on the lateral collateral ligament of the hock, the lateral tendon of the fibularis tertius, and the middle extensor retinaculum. It inserts on the long digital extensor tendon. All digital extensor muscles are bound down by the distal extensor retinaculum in the proximal third of the metatarsus (Figure 1.34).

Emerging under the distal edge of the distal extensor retinaculum, the large dorsal metatarsal artery III ("great metatarsal artery") runs obliquely to the groove between the third and fourth metatarsal bones. A very small satellite vein and the lateral dorsal metatarsal nerve run with the artery. The terminal branch of the caudal cutaneous sural nerve crosses superficial to the dorsal metatarsal artery III (Figure 1.34). Distally the artery passes between the cannon and lateral splint bones near the button of the splint. Here it continues as the distal perforating branch, sending branches to the distal deep plantar arch and then dividing into medial and lateral digital arteries. These continue along the plantar aspect of the third metatarsal bone in the distal fourth of the metatarsus.

The lateral dorsal metatarsal nerve remains superficial, courses to the fetlock, and descends on the dorsal pastern to terminate in the laminar corium. The medial dorsal metatarsal nerve supplies sensory fibers to the hock joint capsule and a motor branch to the short digital extensor muscle. The nerve courses between the long digital extensor tendon and the second metatarsal bone to be distributed distally to the pastern and laminar corium (Figure 1.35).

Lateral and Medial Aspects

The lateral and medial plantar nerves lie plantar to their satellite vessels along the dorsal borders of the digital flexor tendons (Figures 1.34 and 1.35). These nerves supply the lateral, medial, and plantar structures of the metatarsus. The lateral plantar nerve gives rise to a deep branch close to the tarsus; this is the parent trunk of the deeply located lateral and medial plantar metatarsal nerves that pursue courses homologous to the palmar metacarpal nerves in the forelimb. It likewise gives off a sensory branch to the suspensory ligament at this level.

At about the mid-metatarsus, the medial plantar nerve gives off the communicating branch that angles laterodistad superficial to the digital flexor tendons to join the lateral plantar nerve in the distal fourth of the metatarsus. The communicating branch is generally smaller than its counterpart in the metacarpus, and it may be absent.

On each side the very small medial and lateral plantar arteries arise from the deep plantar arch in the proximal metatarsus, itself supplied mainly by the proximal perforating branch from the dorsal pedal artery. The plantar arteries course down to the distal end of the

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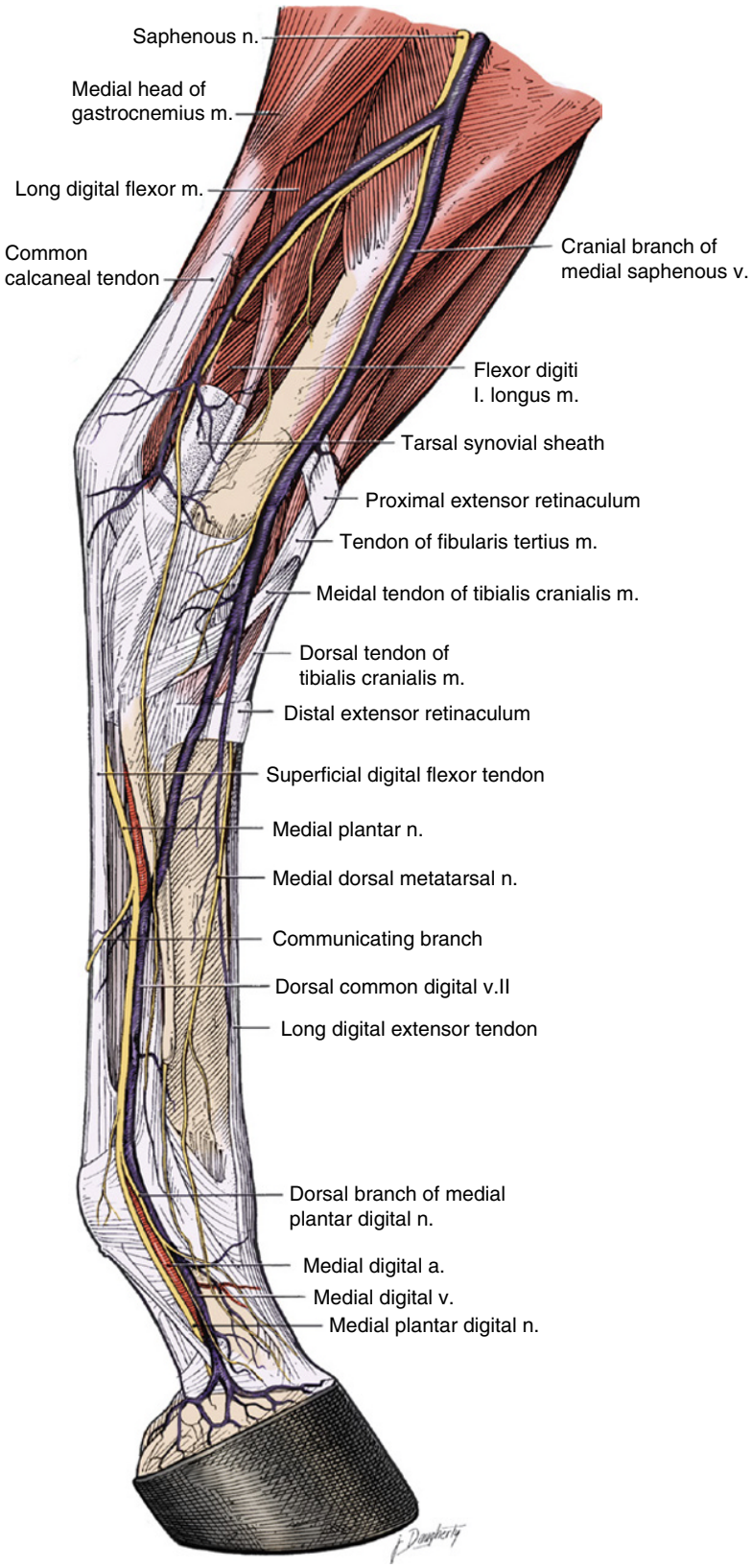


Figure 1.35. Medial view of left distal crus and pes. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

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metatarsus where they send branches to the respective digital arteries, forming the superficial plantar arch.

Plantar Aspect

The SDFT is similar to the corresponding tendon in the metacarpus.[11] The deep digital flexor muscle's principal tendon is intimately related to the SDFT. In the proximal third of the metatarsus, the tendon of the medial digital flexor muscle (a.k.a. the medial head of the deep digital flexor muscle) joins the main part of the DDFT. A poorly developed, slender accessory ligament (tarsal or "inferior" check ligament) arises from the plantar aspect of the fibrous joint capsule of the hock. Longer and more slender than its counterpart in the forelimb, it joins the DDFT near the middle of the metatarsus. The tarsal check ligament may be absent in some horses, and it is usually absent in mules and ponies.[16]

The suspensory ligament takes origin from a large area on the proximal aspect of the third metatarsal bone, with a smaller attachment on the distal row of tarsal bones. Lying within the plantar groove created by the metatarsal bones, the suspensory ligament of the hindlimb is relatively thinner, more rounded, and longer than the ligament of the forelimb. In some horses (e.g. Standardbreds), the suspensory ligament of the hindlimb contains more muscle than the suspensory ligament of the forelimb.[45] The two extensor branches course in a similar manner to those in the forelimb. Vestigial medial and lateral interossei and lumbricales muscles are present in the metatarsus as in the metacarpus.

Distribution of the medial and lateral plantar metatarsal arteries coursing distad under the suspensory ligament to the distal deep plantar arch is similar to the distribution of the palmar metacarpal arteries. Satellite veins accompany the arteries.

Tarsus (Hock)

The bones of the tarsus include the talus, calcaneus, and central, fused first and second, third, and fourth tarsal bones (Figure 1.33). Proximally, the trochlea of the talus articulates with the tibia in the tarsocrural (a.k.a. tibiotarsal) joint; the distal row of tarsal bones and the three metatarsal bones articulate in the tarsometatarsal joint. Extensive collateral ligaments span these and the intertarsal joints. In the horse, nearly all the movement of the hock arises from the tarsocrural joint.

Dorsal Aspect

The large cranial branch of the medial saphenous vein continues proximad and crosses the mediodorsal aspect of the tarsus, lying upon the dorsomedial pouch of the tarsocrural joint capsule (Figure 1.36). The dorsal pedal vein is continued proximad by the cranial tibial vein, which lies under the cover of the cranial tibial muscle. The superficial fibular (peroneal) nerve lies lateral and parallel to the tendon of the long digital extensor muscle. The middle extensor retinaculum leaves the lateral tendon of insertion of the fibularis (peroneus) tertius muscle, wraps superficial to the long digital extensor tendon and its sheath, and attaches to the calcaneus.

The long digital extensor tendon is located just lateral to the palpable medial ridge of the trochlea of the talus. Its synovial sheath extends from the level of the lateral malleolus distad nearly to the junction of the tendon with the tendon of the lateral digital extensor muscle (Figure 1.34). The short digital extensor muscle covers the tarsal joint capsule, the dorsal pedal artery, and the termination of the deep fibular nerve as it bifurcates into the two dorsal metatarsal nerves (Figure 1.34).

As it crosses the dorsal surface of the tarsocrural joint, the tendon of the fibularis tertius muscle is superficial to the tendon of the cranial tibial muscle (Figures 1.35 and 1.36). After passing deep to the proximal extensor retinaculum, it splits to create a sleevelike cleft through which the tendon of the cranial tibial and its synovial sheath pass. Having allowed passage of the cranial tibial tendon, the fibularis tertius divides into a dorsal and a lateral tendon of insertion. The dorsal tendon inserts on the third tarsal and third metatarsal bone (Figure 1.36). The lateral tendon extends distad deep to the long digital extensor tendon. It then bifurcates and inserts on the calcaneus and the fourth tarsal bone.

The tendon of the cranial tibial muscle, having emerged through the fibularis tertius, itself bifurcates into a dorsal tendon, which inserts on the cannon bone, and a medial ("cunean") tendon, which angles mediad to insert on the first tarsal bone after passing superficial to the medial tendon of insertion of the fibularis tertius. A large bursa (cunean bursa) is interposed between the cunean tendon and the long medial collateral ligament (Figure 1.37).

The cranial tibial artery is the main blood supply to the pes (tarsus, metatarsus, and digit). At the level of the tarsocrural joint, it is continued as the dorsal pedal artery (Figure 1.36). Branches from the dorsal pedal artery form the dorsal tarsal rete in the tarsal fascia. Small medial and lateral tarsal arteries arise from the dorsal pedal artery and supply respective sides of the tarsus. Before continuing as the dorsal metatarsal artery III, the dorsal pedal artery gives off the proximal perforating branch that traverses the vascular canal formed by the central, third, and fourth tarsal bones. This branch joins the proximal deep plantar arch. Satellite veins accompany the arteries.

Lateral Aspect

The caudal cutaneous sural nerve innervates the lateral aspect of the tarsus as it courses superficial to the calcaneus and from the more dorsally located superficial fibular nerve (Figure 1.34).

The tendon of the lateral digital extensor muscle is bound by a fibrous band in a groove in the lateral malleolus of the tibia and then passes through the long lateral collateral ligament of the tarsus as it angles dorsodistad (Figure 1.38). A synovial sheath enfolds the tendon from just proximal to the lateral malleolus to a point just proximal to the tendon's junction with the long digital extensor tendon. Plantar to the lateral extensor tendon, the lateroplantar pouch of the tarsocrural joint capsule protrudes between the lateral malleolus and the calcaneus.

Medial Aspect

The chestnut, the keratinaceous vestige of a tarsal pad, is located in the skin on the distomedial aspect of

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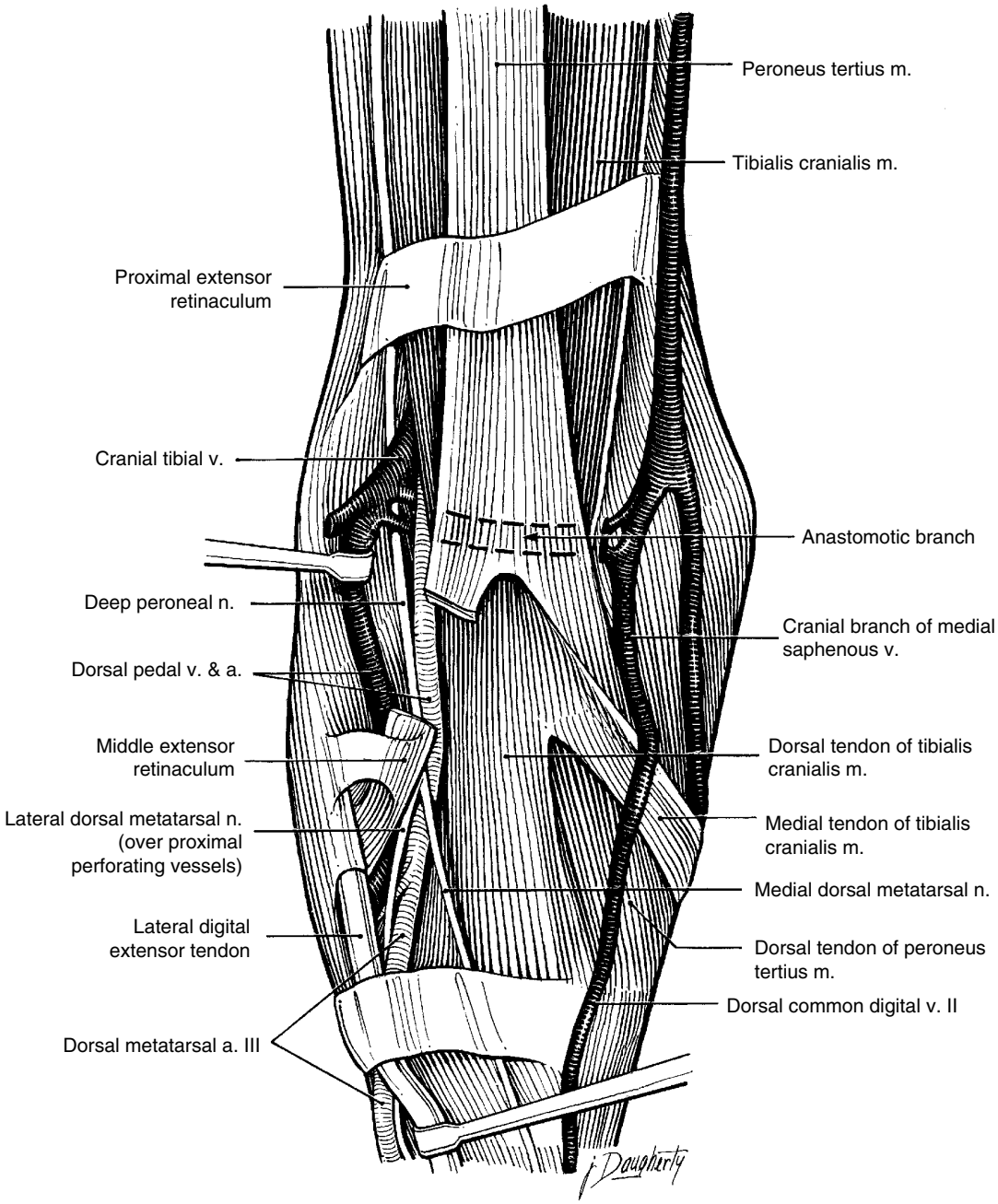


Figure 1.36. Dorsal dissection of right tarsus. The long digital extensor and short digital extensor muscles have been removed. The lateral tendon of the fibularis tertius is sectioned. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

the tarsus. Branches from the saphenous and tibial nerves supply sensory innervation to the medial aspect of the tarsus. The large cranial branch of the medial saphenous vein courses subcutaneously, superficial to the dorsomedial pouch of the tarsocrural joint capsule. At the level of the medial malleolus of the tibia, it sends a small anastomotic branch to the cranial tibial vein. The caudal branch of the medial saphenous vein receives blood from branches in the medial and plantar regions of the hock.

A palpable feature of the medial aspect of the hock is the medial (cunean) tendon of the cranial tibial muscle as it inserts on the fused first and second tarsal bone. The bursa between the cunean tendon and the distal part of the long medial collateral ligament of the tarsus is not normally palpable (Figure 1.37). The tendon of the medial digital flexor (a.k.a. medial head of the deep digital flexor muscle or long digital flexor muscle) passes through a fascial tunnel plantar to the medial collateral ligament. A synovial sheath surrounds this tendon from the distal fourth of the tibia to the tendon's junction with the main tendon of the deep digital flexor (Figure 1.39). A compartment of the tarsocrural joint capsule, the medioplantar pouch, is located a short distance plantar to the medial digital flexor tendon and proximal to the sustentaculum tali of the calcaneus at the level of the medial malleolus.

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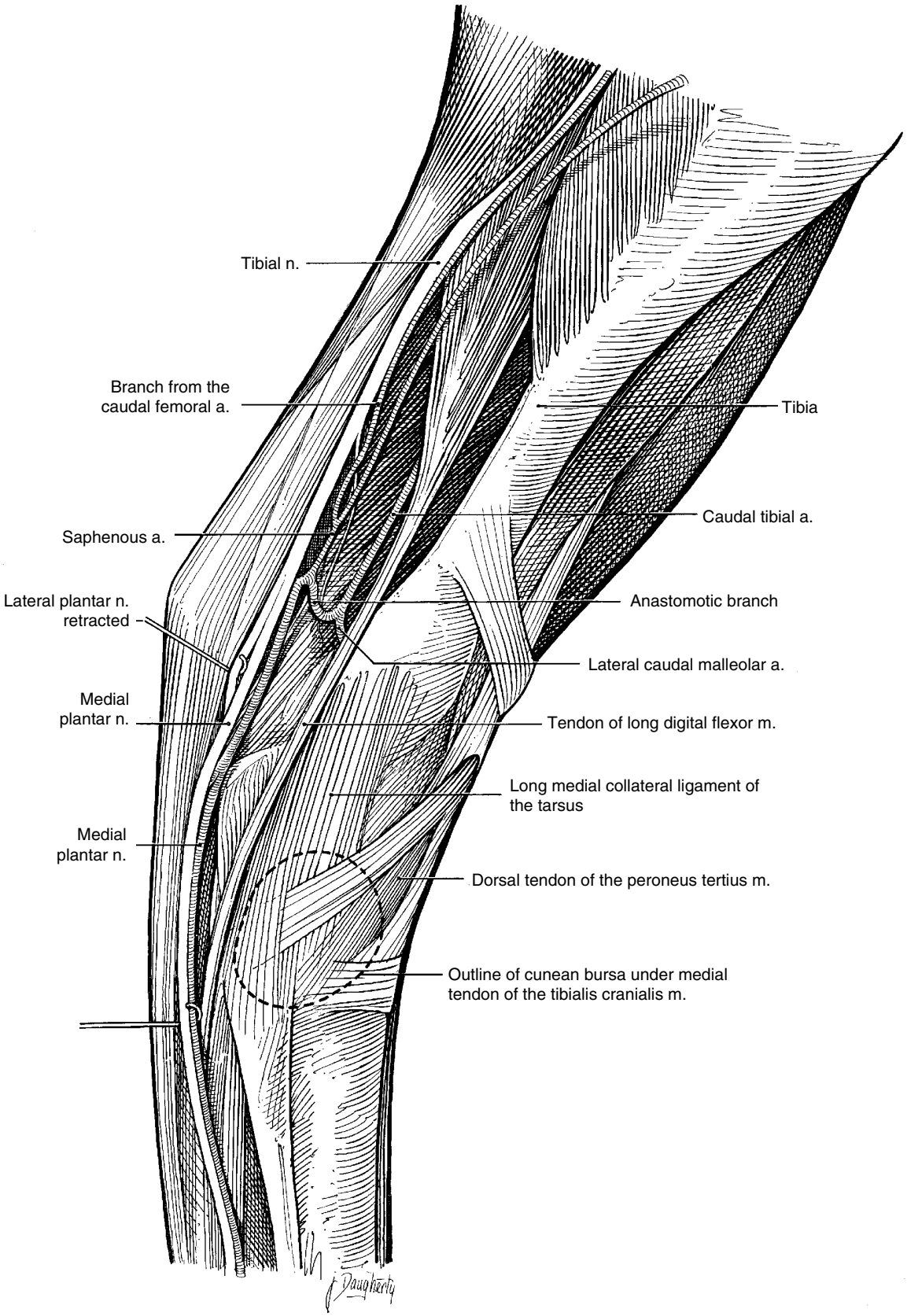


Figure 1.37. Medial dissection of left distal crus, tarsus, and metatarsus. Medial view. Please note that the term "fibularis" has superceded "peroneus" (fibular rather than peroneal), although both are widely used.

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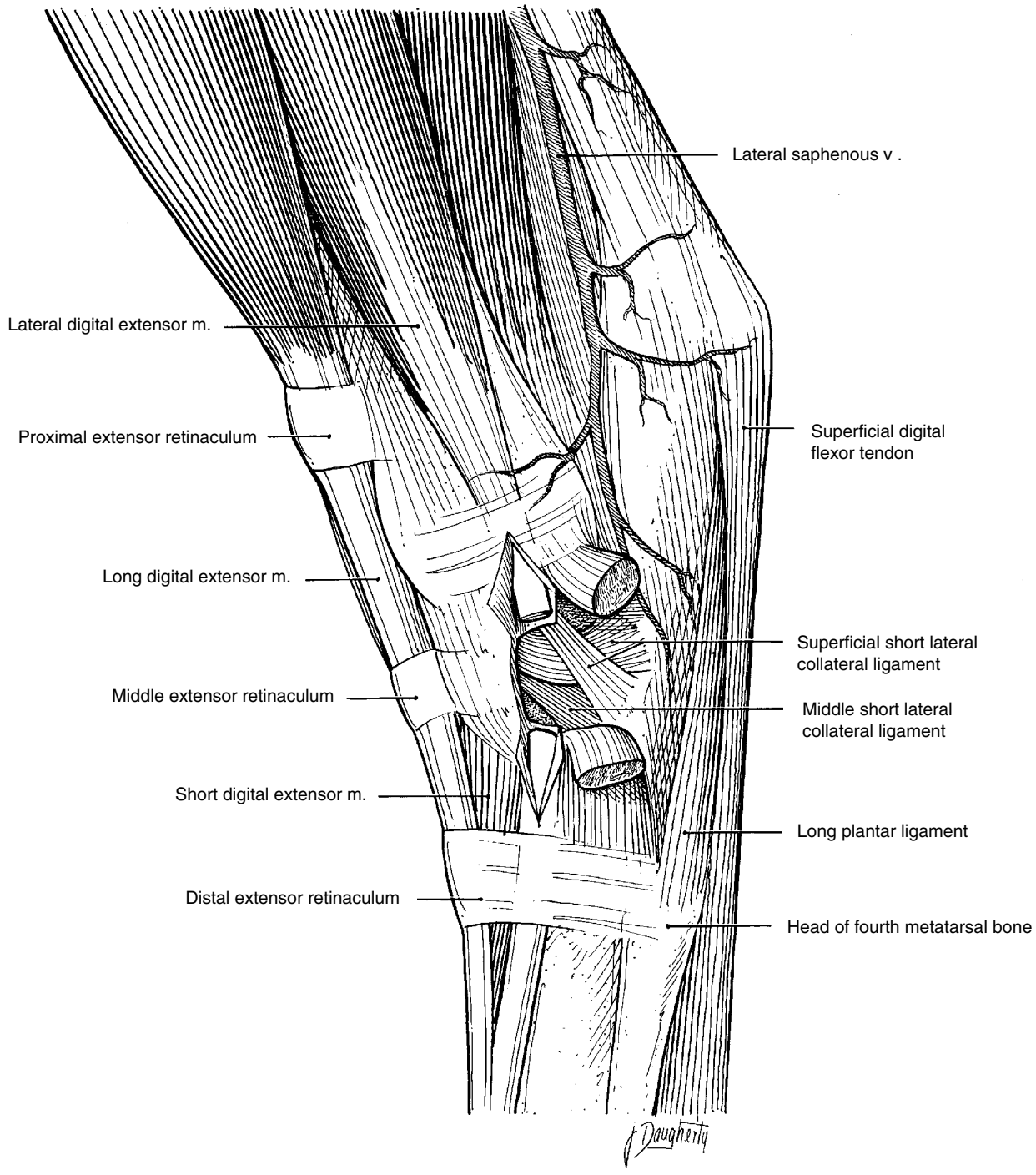


Figure 1.38. Lateral view of left tarsus. The long lateral collateral ligament has been cut and reflected. A section of the lateral digital extensor tendon has been removed.

The tarsal fascia thickens into a flexor retinaculum, bridging the groove on the calcaneus to form the tarsal canal containing the principal tendon of the deep digital flexor muscle. The tendon's synovial sheath, the tarsal sheath, extends from a level proximal to the medial malleolus to the proximal fourth of the metatarsus (Figure 1.35). After joining the anastomotic branch of the caudal tibial artery just proximal to the tarsus, the saphenous artery continues distad with the tendon (Figure 1.37). It bifurcates at the level of the sustentaculum tali of the calcaneus into small medial and lateral plantar arteries. Medial and lateral plantar nerves from the tibial nerve in the distal crural region also accompany the principal DDFT (Figure 1.37). At the level of the tarsometatarsal joint, the medial plantar nerve and artery cross obliquely over the plantar surface of the DDFT to the medial side of the tendon.

Plantar Aspect

In the distal third of the crus, the tendon of the superficial digital flexor muscle arcs around the medial side of

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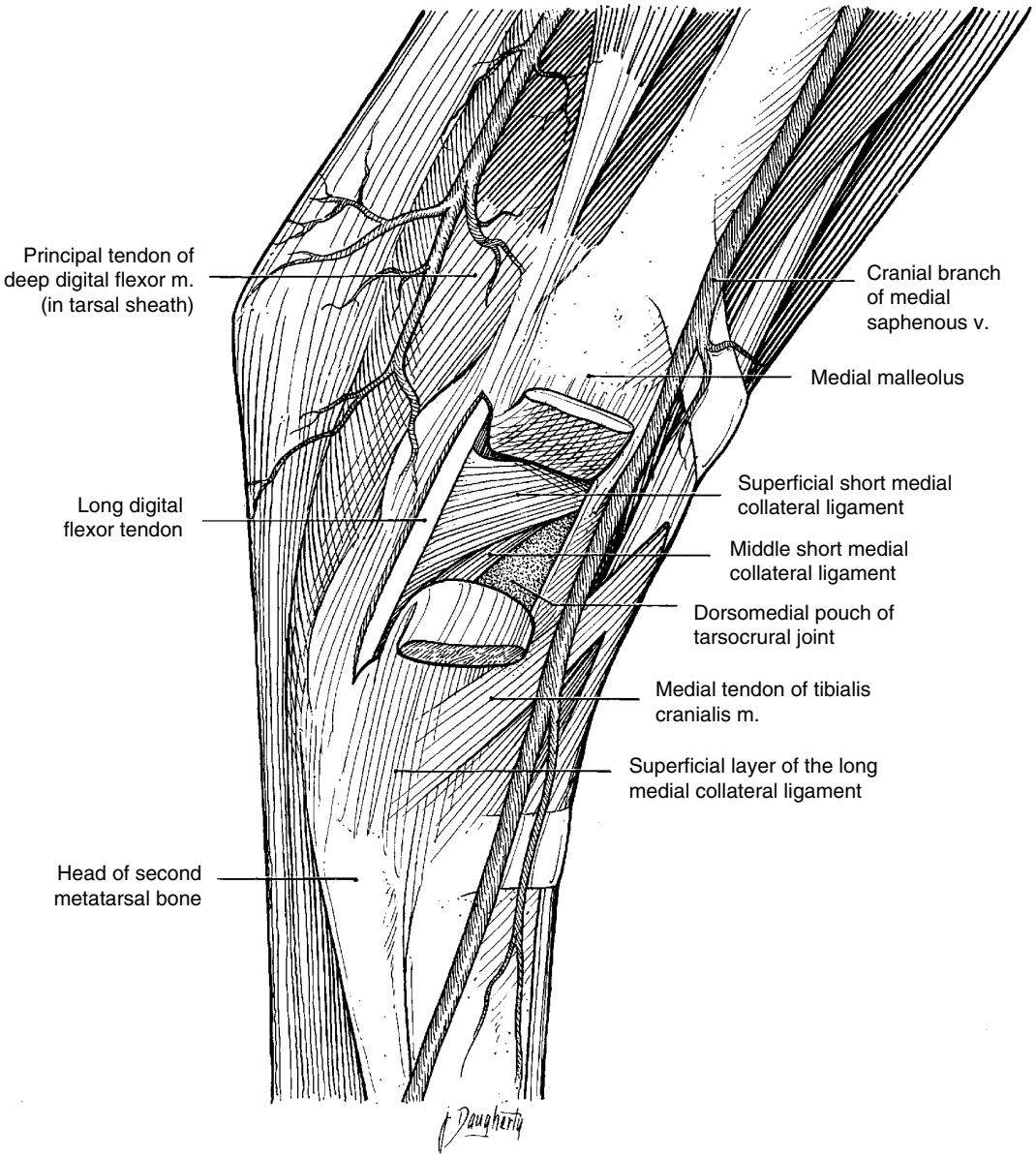


Figure 1.39. Medial view of left tarsus. The long medial collateral ligament has been cut and reflected. The tendon sheath of the long digital flexor tendon has been opened. Please note that "long digital flexor" is an alternative term for the medial head of the deep digital flexor muscle.

the tendon of the gastrocnemius muscle to become superficial as the tendons approach the calcaneal tuber. The SDFT flattens and is joined by aponeurotic connections of the biceps femoris and semitendinosus muscles. This tendinous complex attaches to the point and sides of the calcaneal tuber. The continuation of the tendon then narrows and continues distad superficial to the long plantar ligament. The tendon of the gastrocnemius, deep to the superficial digital flexor at the hock, inserts on the plantar surface of the calcaneal tuber. An elongated bursa is interposed between the two tendons just above the tarsus. A smaller bursa is present between the SDFT and the calcaneal tuber. These two bursae usually communicate across the lateral surface of the gastrocnemius tendon.[30] An inconstant subcutaneous bursa may develop over the superficial digital flexor at the level of the calcaneal tuber.

Deep to the superficial digital flexor, the long plantar ligament is attached to the plantar surface of the calcaneus. It terminates distally on the fourth tarsal bone and the proximal extremity of the fourth metatarsal bone (Figure 1.38).

Tarsal Joint (Hock Joint)

The distal end of the tibia features deep grooves that constitute the cochlear articular surface. In the standing position, the dorsal articular angle of the hock is around 150°.[16] The joint can be flexed to the extent that the metatarsus contacts the cranial crus. During flexion of the tarsocrural joint, the pes is directed slightly laterad due to the configuration of the joint. Joints between the tarsal bones of the same row, the intertarsal joints, and the tarsometatarsal joint are capable of only a small

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amount of gliding movement, so that nearly all of the hock's range of motion arises from the tarsocrural joint.

A long collateral ligament and three short collateral ligaments bind each side of the hock (Figures 1.38–1.40).[43] The long lateral collateral ligament extends from the lateral malleolus and attaches distally to the calcaneus, talus, fourth tarsal, lateral splint, and cannon bones. The three short lateral collateral ligaments are fused proximally where they attach to the lateral malleolus. The superficial component attaches distoplantarly to both the talus and calcaneus, whereas the middle and deep short lateral collateral ligaments attach solely on the lateral surface of the talus.

The long medial collateral ligament of the hock has less well-defined borders than its lateral counterpart. From its proximal attachment on the medial malleolus, the long medial collateral ligament extends distad and divides into two layers along its dorsal border. The superficial layer passes over the cunean tendon of the cranial tibial muscle and attaches to the fused first and second tarsal bones and the second and third metatarsal bones just distal to the cunean bursa. The deep layer attaches distally to the talus and the central and third tarsal bones. The plantar edge of the ligament attaches to the deep fascia over the sustentaculum tali and the ligament between the second and third metatarsal bones.

The superficial short medial collateral ligament extends from the medial malleolus to the tuberosities of the talus and the ridge between them (Figure 1.39). The middle short medial collateral ligament extends obliquely from the medial tibial malleolus to the sustentaculum tali and central tarsal bone. It lies on the medial surface of the talus between the two tuberosities, varying in position during movement of the joints. The smallest component, the deep short medial collateral ligament, courses from the distal edge of the medial tibial malleolus obliquely to the ridge between the two tuberosities of the talus.

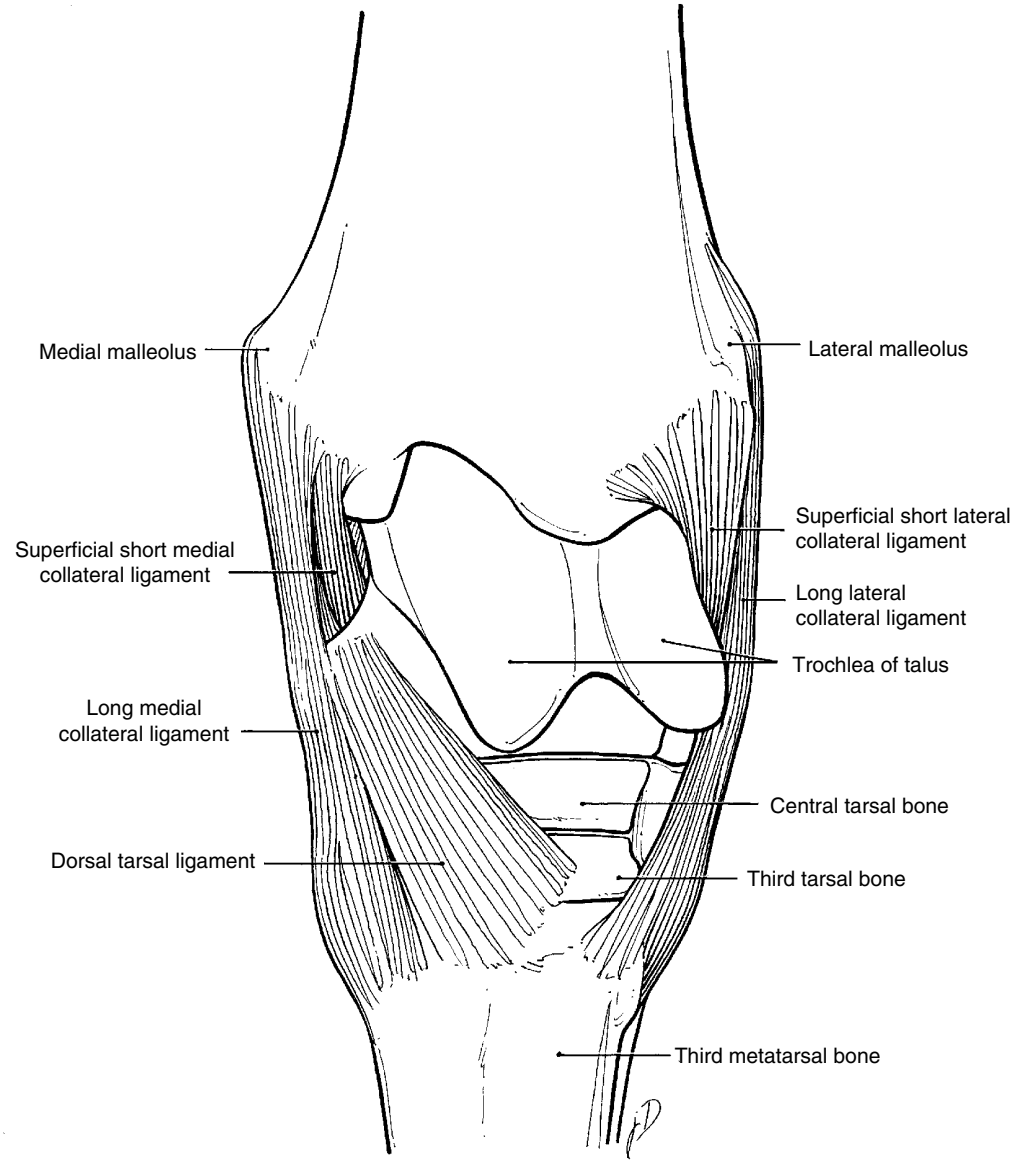


Figure 1.40. Dorsal view of right tarsus.

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A dorsal tarsal ligament fans out distad from the distal tuberosity of the talus, attaching to the central and third tarsal bones and the proximal extremities of the second and third metatarsal bones (Figure 1.40). A plantar tarsal ligament attaches to the plantar surface of the calcaneus and fourth tarsal bone and the fourth metatarsal bone. Smaller, less distinct ligaments join contiguous tarsal bones.

The tarsal joint capsule is thinnest dorsally and thickest in its plantar and distal parts. Cartilage in the capsule covering the flexor groove of the sustentaculum tali provides a smooth surface for the DDFT. Distally the tarsal check ligament of the DDFT takes origin from the fibrous joint capsule.

Three pouches can protrude (most notably with joint effusion) from the large tarsocrural synovial sac where it is not bound down by ligaments: the dorsomedial (largest), medioplantar, and lateroplantar pouches. The tarsocrural sac consistently communicates with the synovial sac associated with the proximal intertarsal joint formed by the talus and calcaneus proximally and the central and fourth tarsal bones distally. The distal intertarsal sac, between the central tarsal and contiguous bones and the distal tarsal row, typically does not communicate with the proximal intertarsal sac, but may communicate with the synovial sac of the tarsometatarsal joint. Communications have been demonstrated in 8.3%–23.8% of cases studied.[38]

Movements of the Tarsocrural Joint

The tarsocrural joint is flexed by contraction of the cranial tibial muscle and the passive pull of the tendinous fibularis (peroneus) tertius. Contraction of the gastrocnemius, biceps femoris, and semitendinosus muscles and the passive pull of the tendinous superficial digital flexor muscle extend the joint. By virtue of its attachments in the extensor fossa of the femur proximally, and on the lateral aspect of the tarsus and dorsal surface of the third metatarsal bone distally, the fibularis tertius passively flexes the tarsocrural joint when the stifle joint is flexed. The superficial digital flexor originates in the supracondyloid fossa of the femur and attaches to the calcaneal tuber. This part of the superficial digital flexor serves to passively extend the tarsocrural joint when the femorotibial joint is extended. The two tendinous, passively functioning elements constitute the reciprocal apparatus (Figure 1.41).

Crus (Leg or Gaskin)

The crus is the region of the hindlimb containing the tibia and fibula, extending from the tarsocrural joint to the femorotibial joint. The proximal end of the fibula articulates with the lateral condyle of the tibia. Distally the fibula narrows to a free end, terminating in the distal one-half to two-thirds of the crus as a thin ligament. An interosseous ligament occupies the space between the two bones. The cranial tibial vessels pass through the proximal part of the ligament. The current preference among anatomists is to replace the Greek word "peroneus" with its Latin equivalent "fibularis" in the naming of crural structures.

Beneath the skin and superficial fascia, a dense crural fascia invests the entire region. The deep crural fascia is continuous with the femoral fascia and with tendons descending from the thigh. It blends with the medial and lateral patellar ligaments and attaches to the medial tibia in the middle of the leg. Caudally, the crural fascia forms the combined aponeuroses of the biceps femoris and semitendinosus muscles that attach with the SDFT to the calcaneal tuber. Deeper layers of the crural fascia invest the muscles of the leg.

Cranial Aspect

The belly of the long digital extensor muscle is prominent beneath the skin on the craniolateral aspect of the crus. It originates in common with the fibularis tertius from the extensor fossa of the femur, the common tendon descending through the extensor sulcus of the tibia (Figure 1.42). The long digital extensor muscle is related on its deep side to the tendinous fibularis tertius and the fleshy cranial tibial muscles. Caudally, it contacts the lateral digital extensor muscle from which it is separated by a distinct intermuscular septum. The superficial fibular nerve courses distad in the groove between the digital extensor muscles and angles craniad toward the hock. The deep fibular nerve courses distad between the two muscles on the cranial surface of the intermuscular septum. At its origin this nerve sends branches to the digital extensor muscles, fibularis tertius, and cranial tibial muscles (Figures 1.42 and 1.43).

Deep to and intimately associated with the fibularis tertius, the cranial tibial muscle covers the craniolateral surface of the tibia, originating from the tibial tuberosity, lateral condyle, and lateral border of the bone and from the crural fascia (Figure 1.42). After it passes through the interosseous space (between the tibia and fibula), the cranial tibial artery courses distad on the tibia deep to the cranial tibial muscle, accompanied by two satellite veins.

Lateral Aspect

The caudal cutaneous sural nerve arises from the tibial nerve (Figure 1.43). Along with the lateral saphenous vein, the caudal cutaneous sural nerve courses laterad across the gastrocnemius muscle. The nerve and vein then descend under the crural fascia and tarsal attachment of the biceps femoris muscle to the distal third of the crus where the nerve penetrates the crural fascia and divides into several branches, one of which courses distad over the hock to the metatarsus (Figures 1.34 and 1.43).

The tibial attachment of the biceps femoris muscle is a broad aponeurosis. It sweeps across the proximal third of the lateral crus to attach to the cranial tibial border. Deep to the belly of the biceps femoris, the common fibular nerve crosses the lateral surface of the lateral head of the gastrocnemius muscle and divides into superficial and deep fibular nerves (Figure 1.43). Caudal to these, the lateral digital extensor muscle originates on the fibula, interosseus ligament, lateral surface of the tibia, and the lateral collateral ligament of the femorotibial joint. The lateral head of the deep digital flexor muscle lies caudal to the belly of the lateral digital extensor.

The lateral head of the gastrocnemius originates on the lateral supracondyloid tuberosity of the femur.

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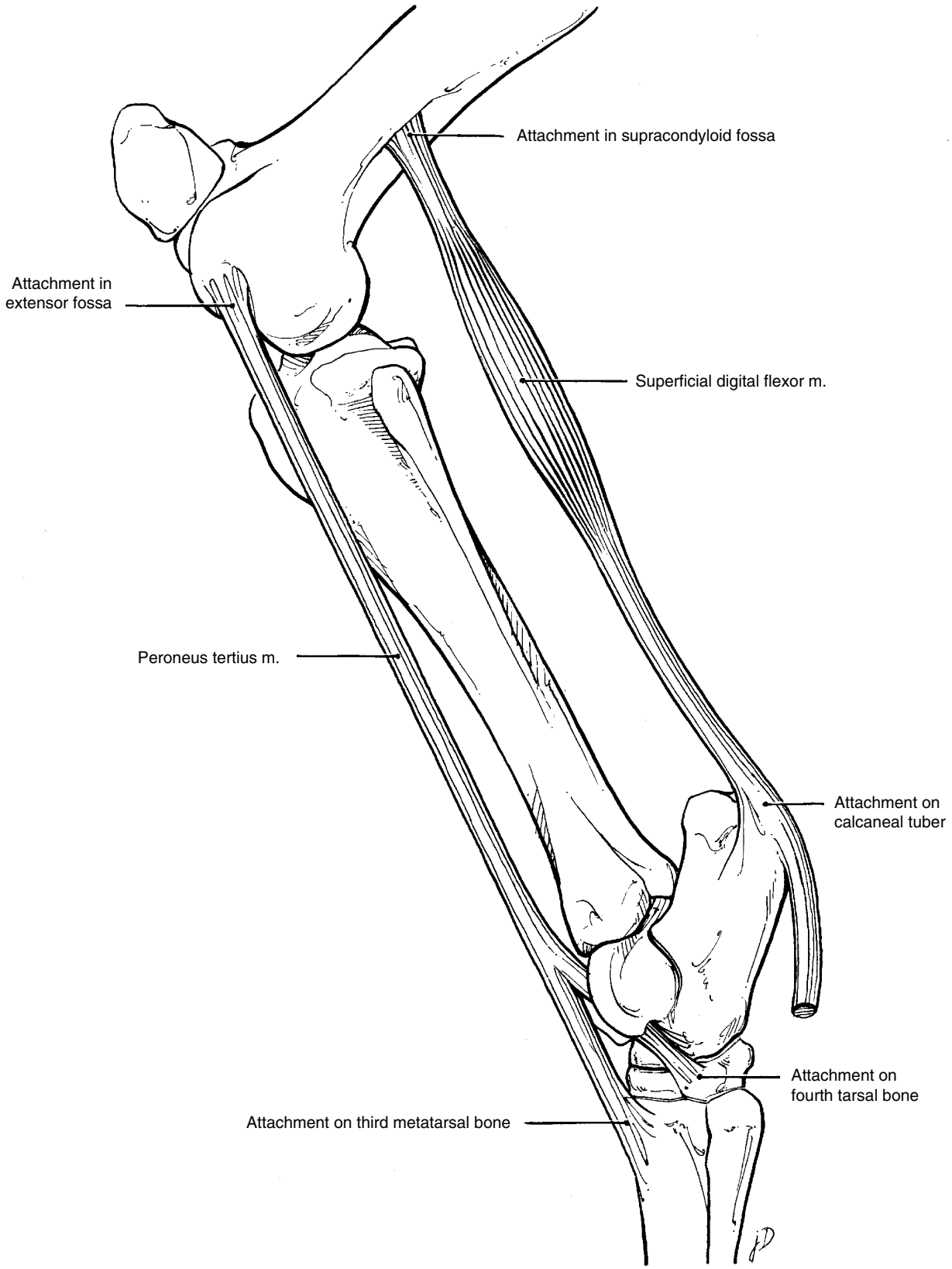


Figure 1.41. Reciprocal apparatus, lateral view of left hindlimb. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

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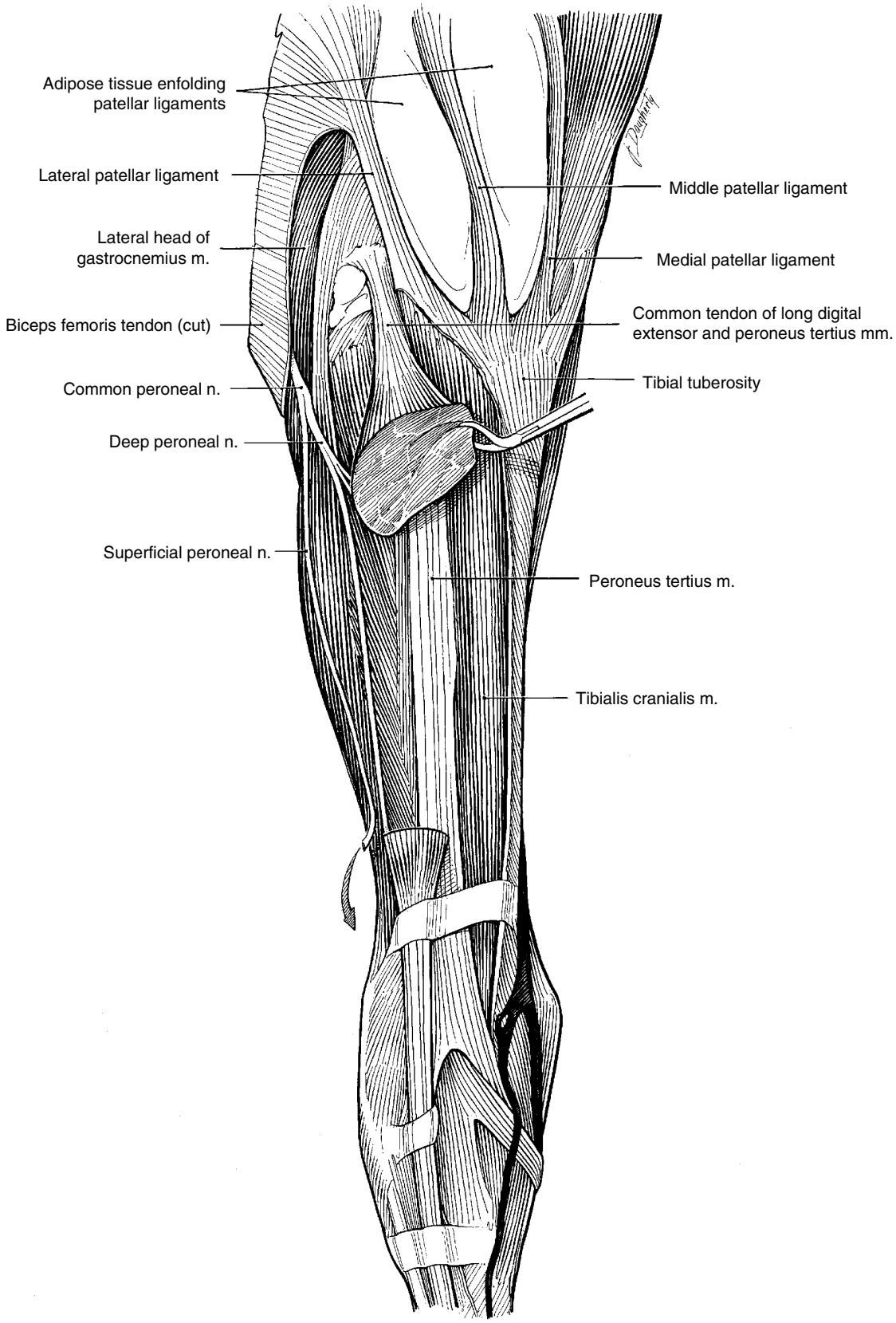


Figure 1.42. Dorsal view of right stifle, crus, and tarsus. The long digital extensor muscle belly has been removed, along with the terminal parts of the superficial fibular (peroneal) nerve (shown by arrow). Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

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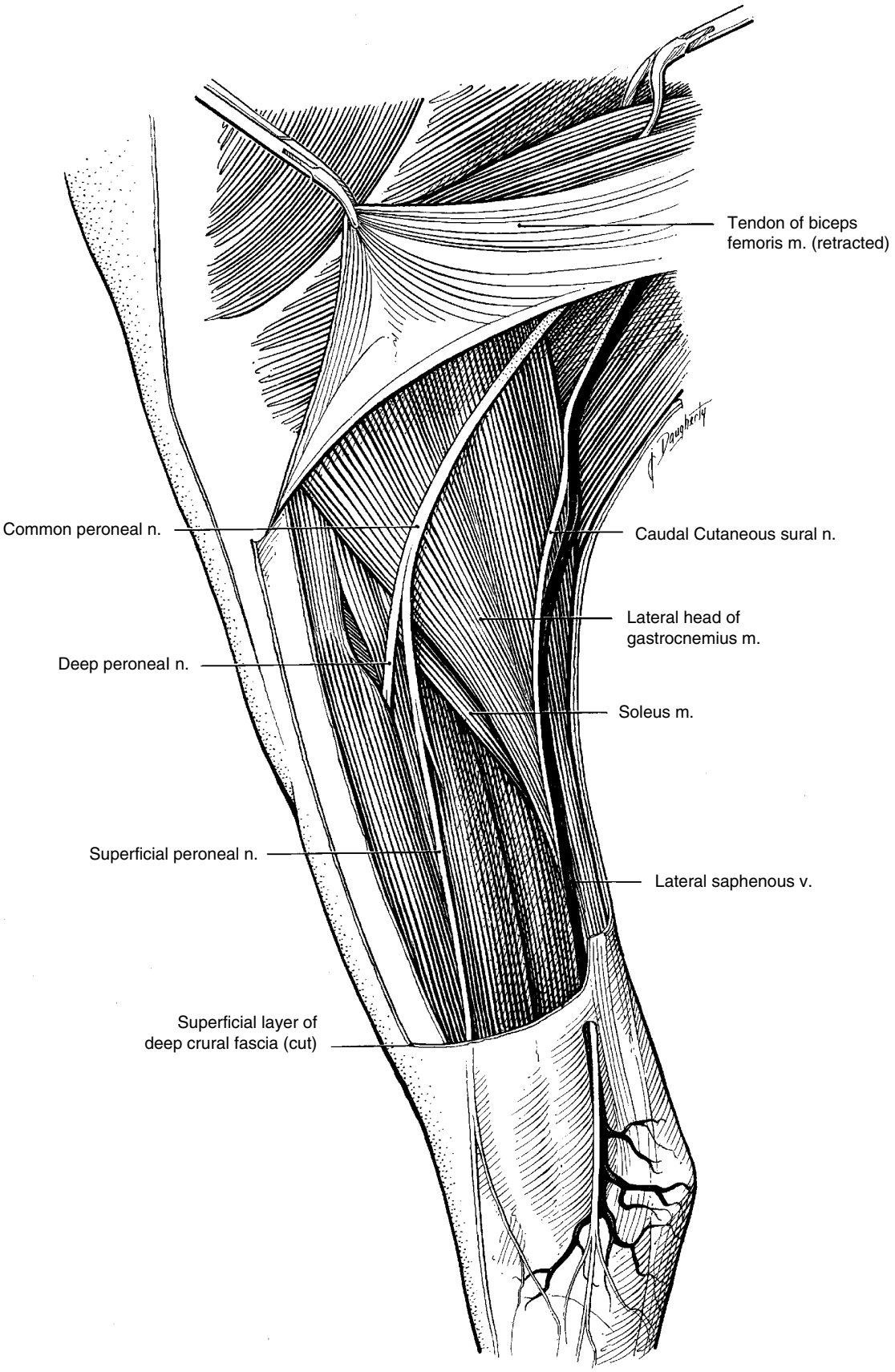


Figure 1.43. Superficial dissection of lateral aspect of left stifle, crus, and tarsus. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

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Under the crural fascia in the proximal half of the crus, the small soleus muscle extends from its origin on the fibula along the lateral aspect of the gastrocnemius muscle to join the gastrocnemius tendon (Figure 1.43).

Medial Aspect

Throughout most of its length, the medial surface of the tibia is subcutaneous (Figure 1.35). Sensation is provided to the medial and cranial aspects of the crus by branches of the saphenous nerve. The distal continuation of the nerve follows the prominent cranial branch of the medial saphenous vein that angles across the medial surface of the tibia. Accompanied by the saphenous artery, the smaller caudal branch of the medial saphenous vein crosses medial to the medial head of the deep digital flexor muscle and joins the cranial branch of the vein superficial to the tibial attachment of the semitendinosus muscle.

Deep to the crural fascia and caudal to the caudal branch of the medial saphenous vein, the tibial nerve descends along with branches of the caudal femoral vessels. The tibial nerve bifurcates into the medial and lateral plantar nerves about 8–10 cm proximal to the point of the calcaneal tuber. These nerves continue distad to the tarsus where they diverge to pursue their independent courses.

The caudal tibial vessels lie deep to the tendon of the medial head of the deep digital flexor as it passes distad. The anastomosis between the caudal tibial and saphenous vessels is located medial to the main tendon of the deep digital flexor (Figure 1.37).

Caudal Aspect

Descending from their origins on the supracondylar tuberosities of the femur, the two heads of the gastrocnemius surround the round, mostly tendinous superficial digital flexor. The latter wraps medially from deep to superficial around the gastrocnemius tendon in the distal third of the crus.

The deep digital flexor muscle possesses three heads with a bewildering variety of names that appear in anatomical texts (Figure 1.45). In the distal third of the crus, the flat tendon of the caudal tibial (the superficial head) joins the larger tendon of the lateral head, whereas the tendon of the medial head (medial digital flexor, long digital flexor) passes over the medial aspect of the hock to join the principal tendon in the proximal metatarsus.

Stifle (Genu)

The stifle is the region that includes the stifle joint (femorotibial joints and femoropatellar joint) and surrounding structures.

Cranial Aspect

Cutaneous innervation of the cranial aspect of the stifle is provided by terminal branches of the lateral cutaneous femoral nerve and lateral branch of the iliohypogastric nerve.

Three patellar ligaments descend from the patella, converging to their attachments on the tibial tuberosity. Between the ligaments and the joint capsule of the femoropatellar joint is an extensive fat pad that wraps around the sides of the ligaments (Figure 1.42). The space between the medial and middle patellar ligaments is greater than the space between the middle and lateral ligaments, reflecting the more widely spaced origin of the medial patellar ligament as it arises from the parapatellar fibrocartilage. The fibrocartilage extends mediad from the patella, giving attachment to the medial patellar ligament. This arcs and descends medial to the medial ridge of the femoral trochlea, attaching distally on the medial side of the tibial tuberosity.

Two bursae lie under the middle patellar ligament. The proximal infrapatellar bursa lies between the proximal part of the ligament and the pointed distal end (apex) of the patella. The distal infrapatellar bursa lies between the ligament and the groove of the tibial tuberosity. Inclining mediad from the lateral aspect of the patella, the lateral patellar ligament serves as an attachment for the biceps femoris muscle and for the fascia lata just before the ligament attaches to the lateral aspect of the tibial tuberosity. The tendon from the biceps femoris continues on to the cranial surface of the patella.

The base, cranial surface, and medial border of the patella, and the parapatellar fibrocartilage and femoropatellar joint capsule all serve as attachments for the insertions of the quadriceps femoris muscle.

Lateral Aspect

The insertional parts of the biceps femoris and the semitendinosus muscles dominate the lateral aspect of the stifle region. The tendon from the cranial part of the biceps femoris inserts on the lateral patellar ligament and the patella; the tendon from the middle division of the muscle sweeps craniodistad to insert on the cranial border of the tibia.

Cutaneous innervation is supplied to this region by branches from several nerves: lateral branches of the iliohypogastric and ilioinguinal nerves, the lateral cutaneous sural nerve originating from the common fibular nerve and passing out between the middle and caudal divisions of the biceps femoris, the caudal cutaneous sural nerve (from the tibial nerve), and terminal branches of the caudal cutaneous femoral nerve (from the caudal gluteal nerve).

Deep to the distal part of the biceps femoris muscle, the lateral femoropatellar ligament extends obliquely from the lateral epicondyle of the femur to the lateral border of the patella (Figure 1.44). The lateral head of the gastrocnemius muscle is crossed by the common fibular nerve and, further caudal, by the caudal cutaneous sural nerve and the lateral saphenous vein (Figure 1.43). As it extends from the lateral epicondyle of the femur to the head of the fibula, the thick lateral collateral ligament passes superficial to the tendon of origin of the popliteus muscle that originates from the lateral epicondyle cranial to the collateral ligament. A pouch from the lateral femorotibial joint capsule lies deep to the tendon. The common tendon of the long digital extensor and fibularis tertius takes origin from the extensor fossa in the craniodistal surface of the lateral epicondyle of the femur. The tendon is cushioned as it extends distad by an elongated pouch from the lateral femorotibial joint capsule.

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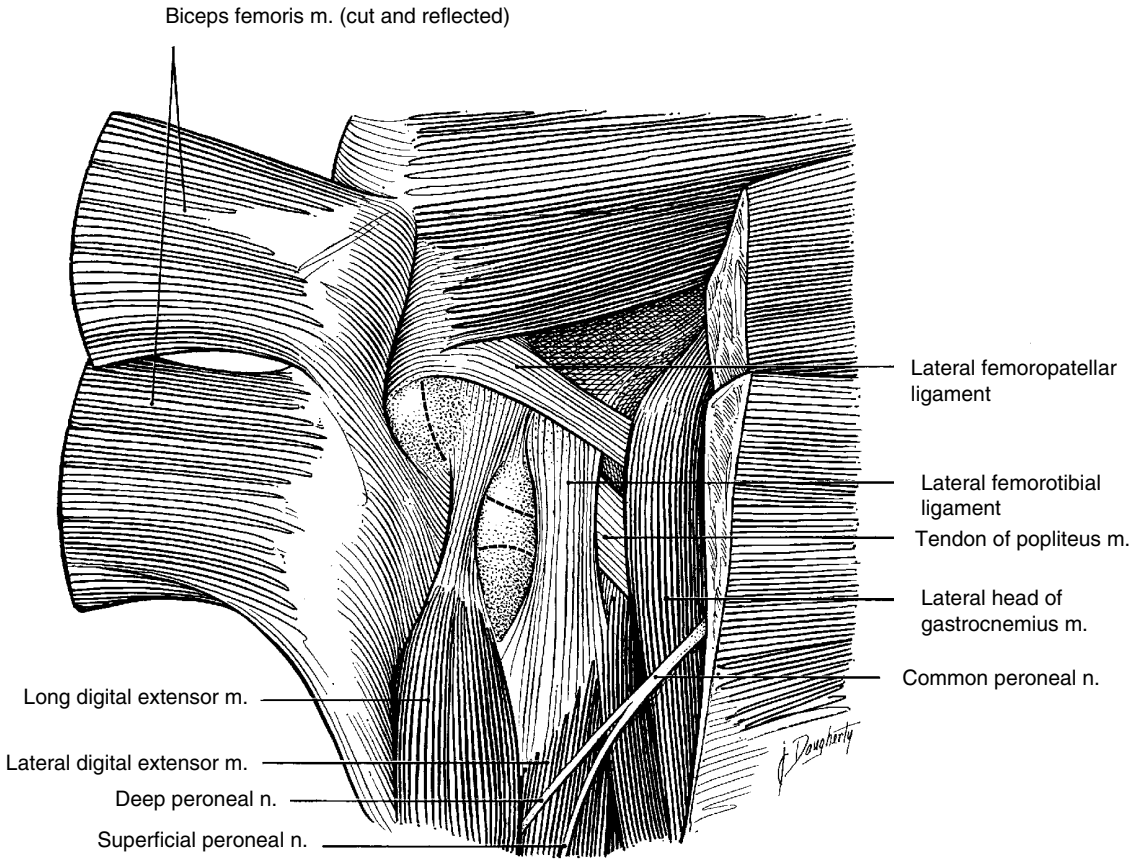


Figure 1.44. Deep dissection of lateral aspect of left stifle with femoral and tibial condylar surfaces indicated by dashed lines. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

Caudal Aspect

The caudal femoral nerve supplies the skin of the caudal aspect of the stifle. The caudal part of the biceps femoris muscle covers the lateral head of the gastrocnemius; a division of the biceps femoris inserts on the cranial border of the tibia, while another continues distad to attach to crural fascia and the calcaneal tuber. The semitendinosus muscle sweeps to its insertion on the cranial border of the tibia and distad toward its tarsal insertion, covering the medial head of the gastrocnemius. The tendons of the two heads of the gastrocnemius combine, and in the proximal part of the crus, the tendon is superficial to the tendon of the superficial digital flexor muscle. Separation of the two heads of the gastrocnemius muscle reveals the tendinous superficial digital flexor muscle that arises in the supracondylar fossa of the femur between the two heads, its initial part embedded in the lateral head (Figure 1.45).

After giving rise to the caudal cutaneous sural nerve, the tibial nerve descends between the two heads of the gastrocnemius along the medial side of the superficial digital flexor. The tibial nerve supplies the gastrocnemius, soleus, superficial digital flexor, deep digital flexor, and popliteus muscles. The femoral artery gives off the distal caudal femoral vessels and continues as the popliteal artery, descending between the two heads of the gastrocnemius (Figure 1.46). Distal to the stifle joint, the popliteal vessels divide into cranial and caudal tibial vessels. The larger cranial artery, the main blood supply to the pes, deviates laterad into the interosseous space between the tibia and fibula; the smaller caudal tibial artery continues distad between the tibia and the popliteus muscle. Satellite veins of the same name accompany these vessels.

The triangular popliteus muscle extends mediodistad from its origin on the lateral epicondyle of the femur (Figure 1.44). The tendon of origin passes deep to the lateral collateral ligament of the stifle joint, partly enveloped in its course by an extension of the femorotibial joint capsule. The popliteus broadens and inserts on the medial part of the caudal tibia, lying alongside the medial head of the deep digital flexor (Figure 1.45).

Medial Aspect

Skin and fascia on the medial aspect of the stifle are supplied by the saphenous and lateral cutaneous femoral nerves. The region is traversed by the medial saphenous vein and the saphenous artery and nerve. Cranially, the vastus medialis of the quadriceps femoris muscle attaches to the parapatellar fibrocartilage, medial border of the patella, and medial patellar ligament. The strap-like sartorius muscle attaches to the medial patellar ligament and the tibial tuberosity. Caudal to the sartorius, the gracilis muscle also attaches to the medial patellar ligament and to the medial collateral ligament

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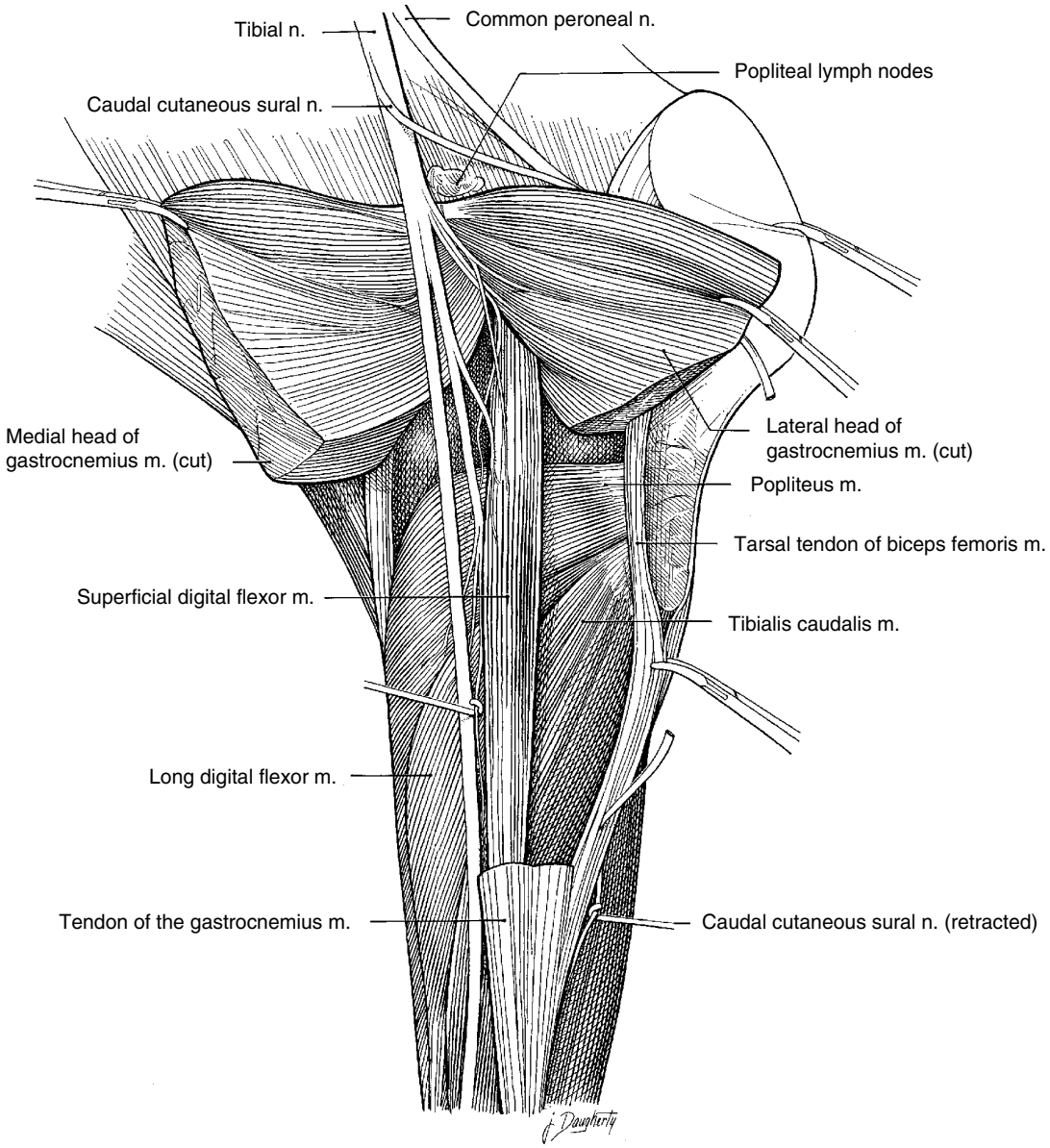


Figure 1.45. Dissection of caudal aspect of right stifle and crus. Please note that the term "fibularis" has superseded "peroneus" (fibular rather than peroneal), although both are widely used.

of the femorotibial joint and the crural fascia (Figure 1.47).

The medial collateral ligament of the femorotibial joint extends from the medial epicondyle of the femur to just distal to the margin of the medial tibial condyle (Figure 1.47). In its course, it detaches fibers to the medial meniscus. The adductor muscle inserts on the ligament and the medial epicondyle. The medial femoropatellar ligament is thinner than its lateral counterpart. It extends from proximal to the medial epicondyle of the femur to the parapatellar fibrocartilage, blending with the femoropatellar joint capsule in its course.

Stifle Joint

The stifle is the "true knee." It comprises two joints, the femoropatellar and femorotibial joints, which together form a hinge joint. The synovial space of the stifle is partitioned into three distinct sacs: the femoropatellar sac and right and left femorotibial joint sacs.[34]

Femoropatellar Joint

The patella is a sesamoid bone between the termination of the quadriceps femoris muscle and the three patellar ligaments constituting the tendon of insertion. The articular surface of the patella is much smaller than the trochlear surface of the femur; the larger gliding surface of the trochlea accommodates the proximal–distal movements of the patella. Contact between the patella and trochlea changes as the patella moves on its gliding surface. During full extension and "locking" of the stifle, the patella is rolled onto to its narrow distal articular surface (resting surface). The narrow craniodorsal surface of the proximal part of the trochlea offers a complimentary resting surface for the patella in this position.[37]

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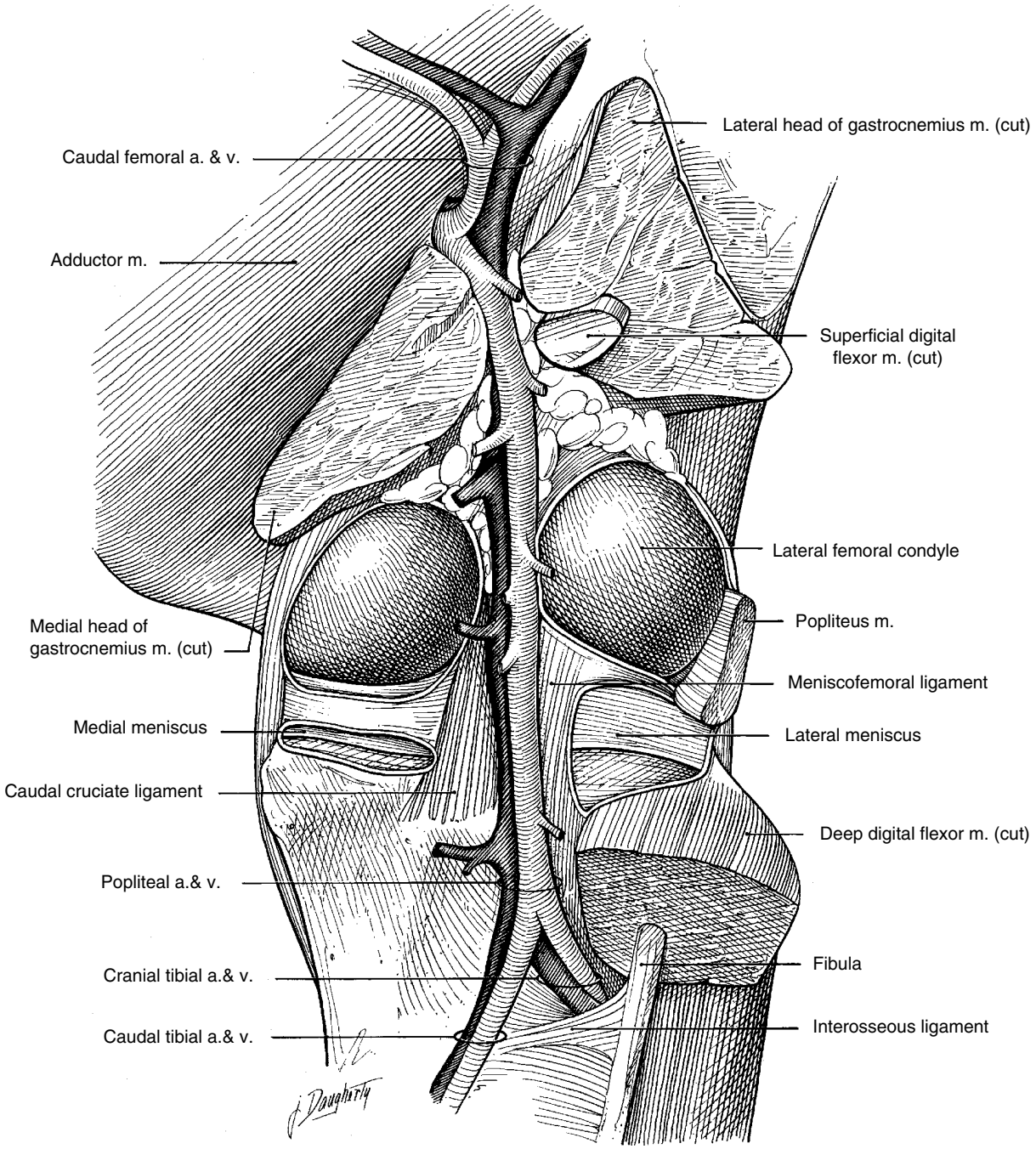


Figure 1.46. Deep dissection of caudal aspect of right stifle. The joint capsule of the femorotibial joint has been opened.

A voluminous joint capsule attaches to the edges of the femoral trochlea, and its patellar attachment lies close to the edge of the patellar articular surface. A large pouch from the joint capsule protrudes proximad under a fat pad and the distal part of the quadriceps femoris muscle. The vastus intermedius of the quadriceps femoris attaches, in part, to the femoropatellar joint capsule, acting to tense the capsule during extension of the femoropatellar joint.

Femorotibial Joint

The fibrous part of the joint capsule is thick caudally, thin cranially. The cruciate ligaments of the femorotibial joint lie between the joint capsule's medial and lateral synovial sacs. These two sacs rarely communicate with one another, although the medial sac communicates with the femoropatellar joint sac in a majority (65%) of horses.[34] Two fibrocartilaginous menisci intervene between the femoral and tibial articular surfaces. An extension of the lateral synovial sac encloses the tendon of origin of the popliteus muscle, and another protrudes distad under the common tendon of origin of the long digital extensor and fibularis tertius muscles.

The two fibrocartilaginous menisci are crescent shaped, thicker peripherally and thinner along the concave edge. Their proximal surfaces are concave to accommodate the convex femoral condyles. Distally they conform to the articular surfaces of the tibial condyles.

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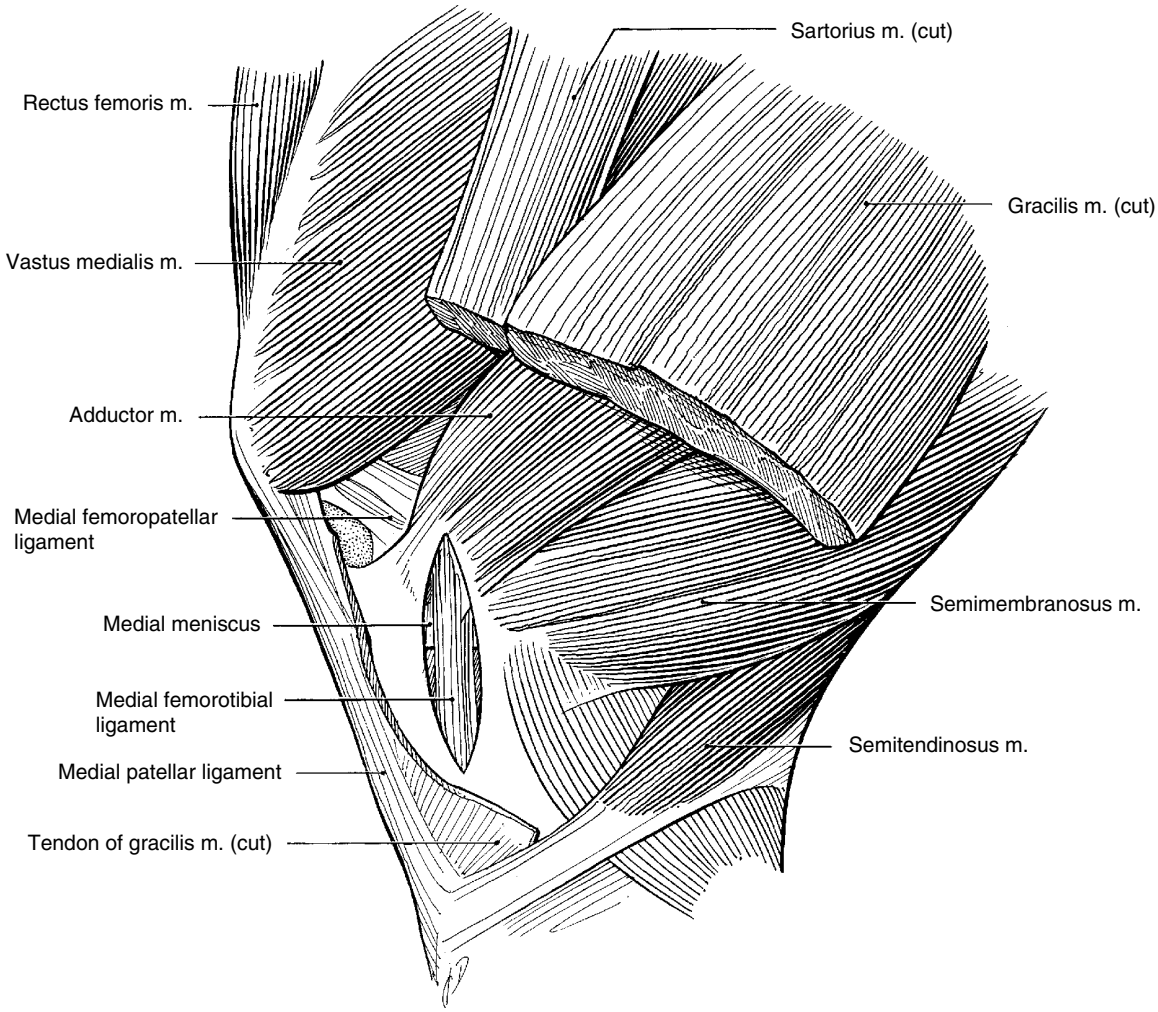


Figure 1.47. Deep dissection of medial aspect of left stifle. Tendon of adductor muscle is incised to reveal medial collateral ligament of the stifle.

Cranial and caudal ligaments anchor each meniscus to the tibia, and a meniscofemoral ligament attaches the caudal aspect of the lateral meniscus to the caudal surface of the intercondyloid fossa of the femur.

In addition to the support rendered by medial and lateral collateral ligaments, the femur and tibia are joined by the two cruciate ligaments that cross one another in the intercondyloid space between the two synovial sacs of the femorotibial joint. The caudal cruciate ligament, the more substantial of the two, extends from the cranial part of the intercondyloid fossa of the femur to the popliteal notch of the tibia, crossing the medial aspect of the cranial cruciate ligament. From its attachment on the caudolateral side of the intercondyloid fossa, the cranial cruciate ligament attaches to a central fossa between the articular surfaces of the condyles.

The stifle is supplied principally by the descending genicular artery that originates from the femoral artery and descends toward the medial aspect of the stifle joint deep to the sartorius and vastus medialis muscles.

Movements of the Stifle Joint

In the standing position, the caudal angle of the stifle joint is around 150°.[16] Extension of the stifle joint through action of the quadriceps femoris, tensor fasciae lata, and cranial division of the biceps femoris muscles is limited by tension from the collateral and cruciate ligaments. Flexion of the joint by the semitendinosus, middle division of the biceps femoris, popliteus, and gastrocnemius muscles is limited only by the caudal muscle masses. During flexion the crus is rotated slightly mediad, and the femoral condyles and menisci move slightly caudad on the tibial condyles.

When a horse shifts its weight to rest on one hindlimb, the supporting limb flexes slightly as the contralateral relaxed limb is brought to rest on the toe. The pelvis is tilted so that the hip of the supporting limb is higher. The stifle on the supporting limb is locked in position as the medial patellar ligament and parapatellar cartilage are pulled proximad and mediad to engage the medial ridge of the femoral trochlea. The locked position achieved by this configuration together with the support rendered by the other components of the stay apparatus minimizes muscular activity in the supporting limb while the relaxed contralateral hindlimb is resting. A very small amount of muscle tone in the vastus medialis is necessary to stabilize the stifle in the locked position.[38]

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Thigh and Hip

Lateral Aspect

Cutaneous innervation is supplied to the lateral aspect of the thigh and hip by the lateral branches of the iliohypogastric and ilioinguinal nerves, the caudal cutaneous femoral nerve, and the dorsal branches of the lumbar and sacral nerves.[17]

From caudal to cranial the superficial muscles of the lateral thigh and hip are the semitendinosus, biceps femoris, gluteus superficialis, gluteus medius, and tensor fasciae lata. Both the semitendinosus and biceps femoris have ischiatic (pelvic) and vertebral origins. The semitendinosus attaches to the first and second caudal vertebrae and fascia of the tail, and the biceps femoris attaches to the dorsal sacroiliac ligament and the gluteal and tail fasciae. A prominent longitudinal groove marks the site of the intermuscular septum between the semitendinosus and the biceps femoris muscles.

The strong gluteal fascia gives origin to the long caudal head and the cranial head of the superficial gluteal (gluteus superficialis) muscle. The two heads of the superficial gluteal muscle come together in a flat tendon that attaches to the trochanter tertius of the femur.

The large middle gluteal (gluteus medius) muscle forms most of the mass of the rump. The middle gluteal muscle is massive, giving the rump its rounded shape. It takes origin from the aponeurosis of the longissimus lumborum muscle, the gluteal surface of the ilium, the tuber coxae and tuber sacrale, the sacrotuberal and dorsal sacroiliac ligaments, and the gluteal fascia. Distally the muscle attaches to greater trochanter and the intertrochanteric crest. A smaller deep part of the middle gluteal, the gluteus accessorius, arises entirely from the ilium. Its tendon passes over greater trochanter on its way to attach on the crest distal to the trochanter. The large trochanteric bursa lies between the tendon and the cartilage covering the convexity (Figure 1.48).

The small deep gluteal (gluteus profundus) muscle is deep to the caudal part of the gluteus medius, arising from the ischiatic spine and body of the ilium and attaching on the medial edge of the convexity of the greater trochanter (Figure 1.50). This muscle covers the hip joint and parts of the articularis coxae, a small fusiform muscle that occasionally has two heads. A bursa is commonly present under the tendon of insertion of the deep gluteal.[30]

The tensor fasciae latae muscle arises from the tuber coxae and fans out distally to insert into the fascia lata. An intermuscular septum attaches the caudal part of the muscle to the cranial head of the superficial gluteal. The fascia lata attaches to the patella and the lateral and middle patellar ligaments (Figures 1.48 and 1.49).

On the caudal side of the proximal part of the femur lie three short muscles that arise from their origins on the ischium, pubis, ilium, and wing of the sacrum to insert in the trochanteric fossa. These are the gemelli, external obturator, and internal obturator muscles, which are rotators of the hip joint. The nearby quadratus femoris muscle extends from the ventral aspect of the ischium to a line on the femur near the distal part of the lesser trochanter where it produces hip extension.

A broad sheet of dense white fibrous connective tissue, the sacrotuberous (sacrotuberal) ligament (a.k.a. broad pelvic ligament), forms most of the lateral wall of the pelvic canal, attaching dorsally to the sacrum and first two caudal vertebrae and ventrally to the ischiatic spine and ischiatic tuber (Figure 1.50). The ventral edge of the sacrotuberous ligament creates two openings along the dorsal edge of the ischium: the lesser and greater ischiatic foramina that allow passage of neurovascular bundles to the soft tissues of the rump.

Branches of the cranial gluteal vessels and nerve pass through the greater ischiatic foremen to supply the gluteal muscles, tensor fasciae latae, and articularis coxae. The caudal gluteal vessels and nerve perforate the sacrotuberal ligament dorsal to the sciatic nerve. The caudal gluteal nerve divides into two trunks. The dorsal trunk supplies the biceps femoris, middle gluteal, and long head of the superficial gluteal; after supplying a branch to the semitendinosus, the ventral trunk continues as the caudal cutaneous femoral nerve. The latter passes outward between the biceps femoris and semitendinosus to branch subcutaneously over the lateral and caudal surfaces of the thigh and hip. Muscles in this region are supplied by the caudal gluteal vessels.

In this region the internal pudendal artery courses on the deep face of the sacrotuberous ligament. It will terminate in branches that supply the urogenital organs and the perineal region. Iliolumbar vessels (from the cranial gluteal vessels) course laterad between the iliacus muscle and the ilium, supplying branches to the iliopsoas and longissimus lumborum. The vessels then go around the lateral border of the ilium and supply branches to the middle gluteal and tensor fasciae latae.

The large, flat sciatic nerve passes through the greater ischiatic foramen and courses ventrocaudad on the sacrotuberale ligament (Figure 1.50). Turning distad, the nerve passes over the gemelli, the tendon of the internal obturator, and the quadratus femoris, supplying branches to these muscles. A large branch is detached that supplies the semimembranosus, the biceps femoris and semitendinosus, and adductor medially and the biceps femoris laterally. The sciatic nerve terminates by dividing into common fibular and tibial nerves.

Medial Aspect

The medial aspect of the thigh receives cutaneous innervation from a mosaic of sensory nerves, including lateral cutaneous femoral nerve (craniomedial thigh); medial branches of the iliohypogastric, ilioinguinal, and genitofemoral nerves (medial thigh); and branches from the caudal cutaneous femoral nerve (caudomedial thigh).[17]

Accompanied by the small saphenous artery and the saphenous nerve, the large medial saphenous vein pursues a subcutaneous course proximad on the cranial part of the gracilis muscle and then between the gracilis and sartorius muscles to join the femoral vein. The broad gracilis muscle covers most of the medial aspect of the thigh, attaching proximally to the prepubic tendon, adjacent surface of the pubis, accessory femoral ligament, and middle of the pelvic symphysis. The narrow sartorius muscle takes origin from the tendon of the psoas minor and ilial fascia and descends toward its insertion in the stifle that blends with the aponeurosis of the gracilis.

The pectineus muscle lies deep to the gracilis. It originates on the cranial border of the pubis, the prepubic tendon, and accessory femoral ligament and inserts on

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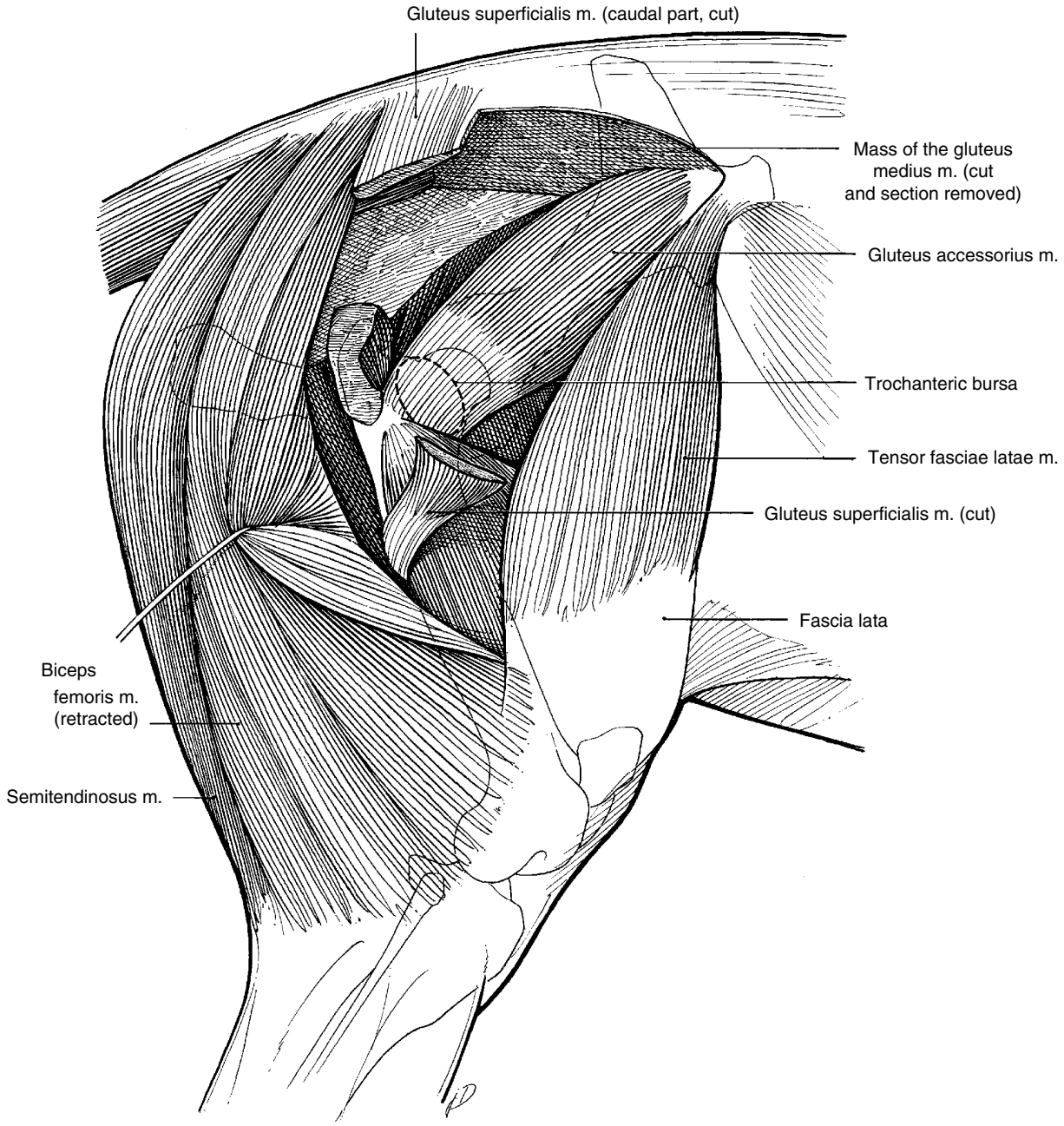


Figure 1.48. Lateral dissection of right thigh and hip. Most of the superficial and middle gluteal muscles have been removed.

the medial femur. The femoral canal, containing the neurovascular bundle supplying the pelvic limb, is bordered caudally by the pectineus, cranially by the sartorius, laterally by the vastus medialis and iliopsoas, and medially by the femoral fascia and cranial edge of the gracilis. The canal contains the femoral artery and vein, the saphenous nerve, and an elongated group of lymph nodes of the deep inguinal lymphocenter. Within the canal the saphenous nerve detaches a motor branch to the sartorius muscle (Figure 1.51).

Caudal to the pectineus and vastus medialis, the thick adductor muscle extends from the ventral surface of the ischium and pubis to the caudal surface of the femur, the medial femoral epicondyle, and the medial collateral ligament of the femorotibial joint. The obturator nerve passes through the cranial part of the obturator foramen and external obturator muscle and branches to supply the external obturator, adductor, pectineus, and gracilis muscles (Figure 1.51). Branches from the obturator artery (from the cranial gluteal artery) supply the muscles in this region.

Cranial Aspect

The quadriceps femoris, articularis coxae, and sartorius muscles lie in the cranial part of the thigh and hip. In addition, the iliacus muscle crosses the cranial aspect of the hip where the muscle is joined by the psoas major, creating the conjoined iliopsoas muscle. Their common tendon inserts on the lesser trochanter. The psoas major

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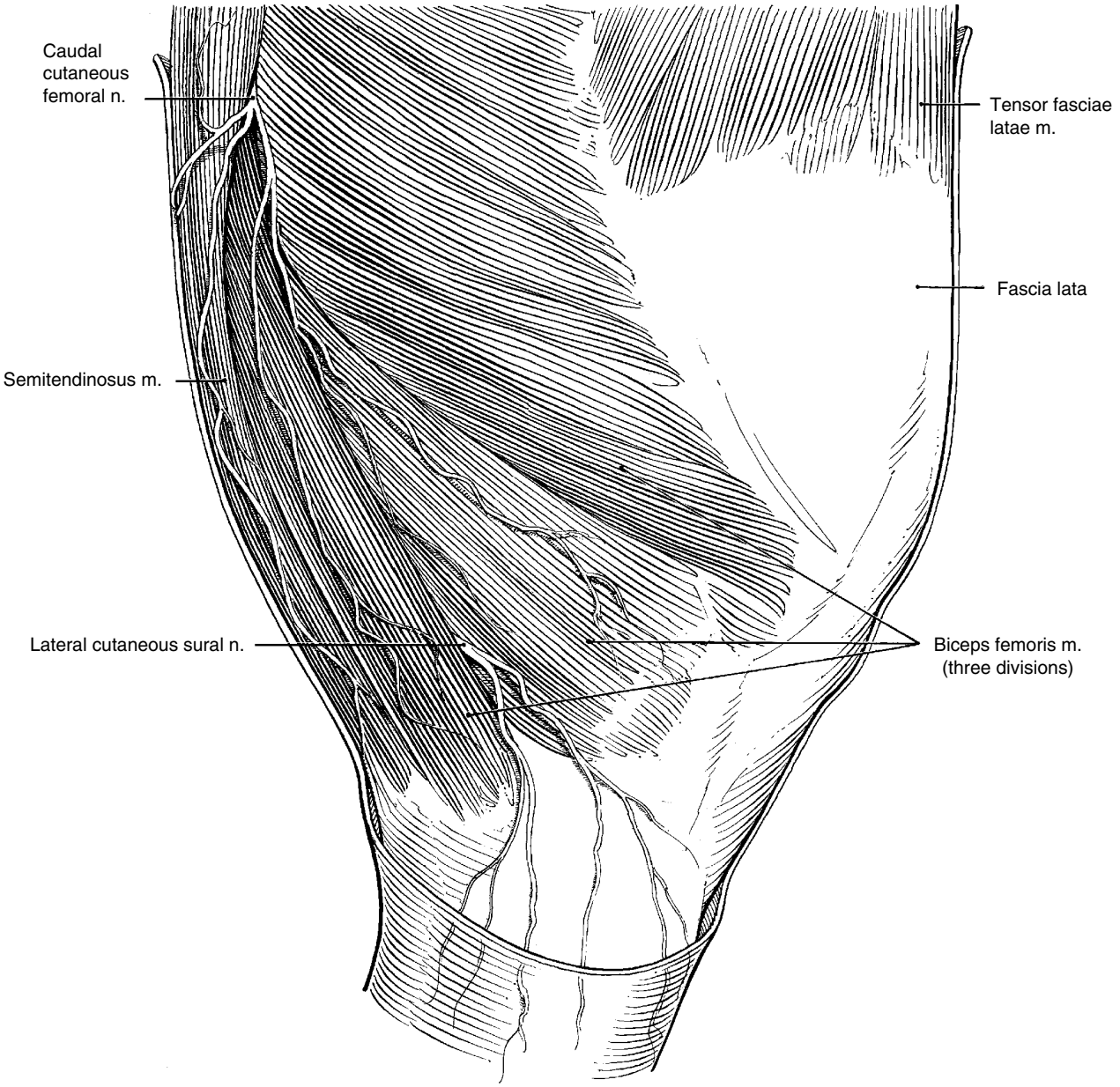


Figure 1.49. Lateral view of right stifle and thigh.

arises from the last two ribs and the lumbar transverse processes; the iliacus comes from the wing of the sacrum, ventral sacroiliac ligaments, ilium, and tendon of the psoas minor muscle.

Three heads of the quadriceps femoris muscles (vastus lateralis, vastus intermedius, and vastus medialis) take origin from the shaft of the femur. The fourth head, the rectus femoris, originates via a pair (medial and lateral) of tendons from the ilium craniodorsal to the acetabulum (Figure 1.50). A bursa is located under the lateral tendon.[30] All four heads of the quadriceps femoris attach to the patella. Bursae occur commonly under the insertions of the rectus femoris, vastus lateralis, and vastus medialis.

The femoral nerve passes distad between the iliopsoas and sartorius muscles. It supplies branches to the iliopsoas (which also receives segmental innervation from lumbar nerves) and to all heads of the quadriceps femoris. Near the insertion of iliopsoas, the femoral nerve gives rise to the saphenous nerve.

Caudal Aspect

Innervation to the caudal skin of the thigh and hip is provided principally by the caudal cutaneous femoral nerve (Figure 1.49). The main muscle mass is that of the semitendinosus and the semimembranosus with the caudal division of the biceps femoris related to these laterally and the gracilis medially. The long head of the semimembranosus attaches to the caudal border of the sacrotuberous ligament. The thicker short head attaches to the ventral part of the ischiatic tuber. The thick, roughly three-sided belly of the semimembranosus ends on a flat tendon that attaches to the medial femoral epicondyle.

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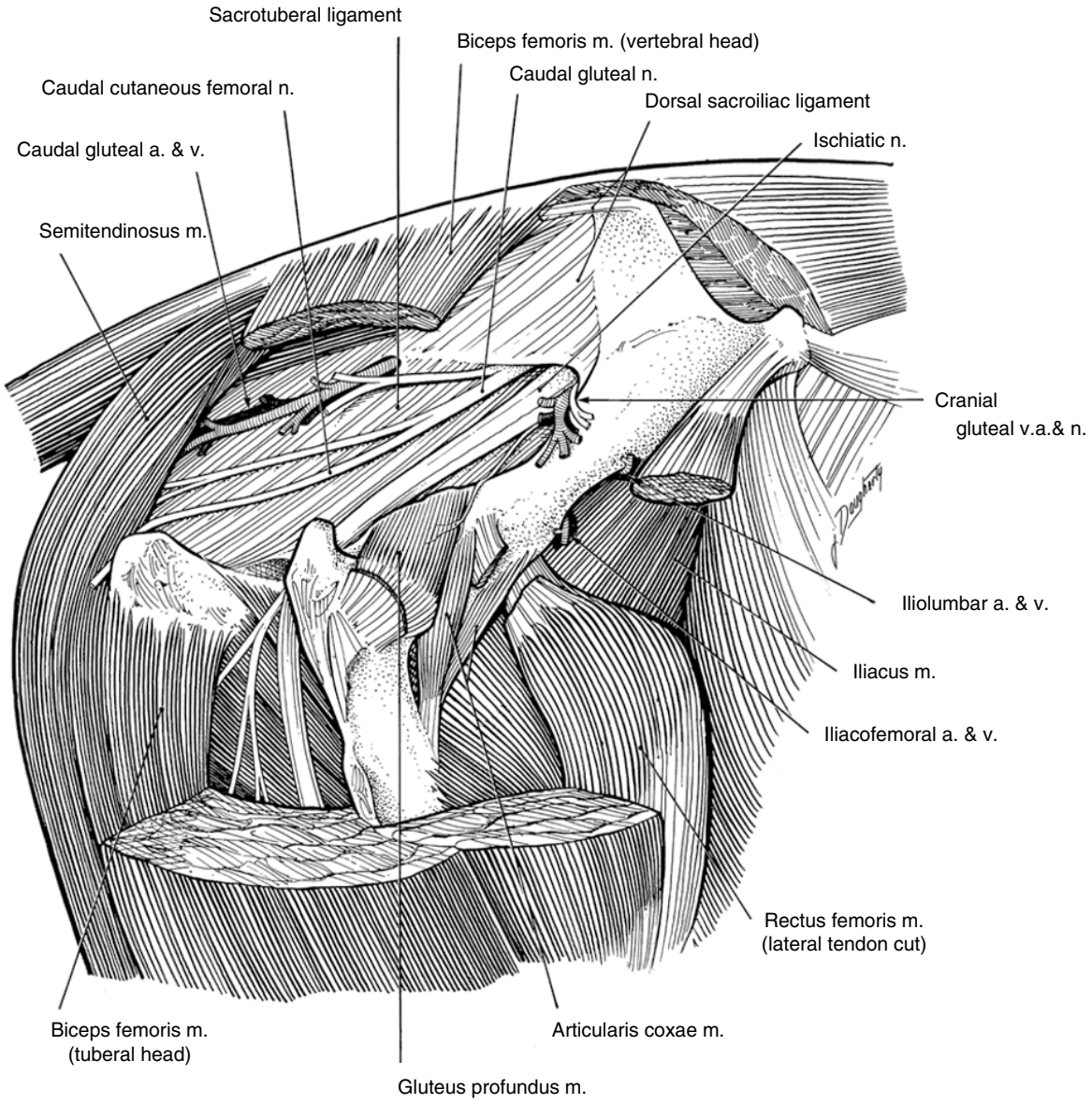


Figure 1.50. Deep dissection of right hip. Lateral view. Superficial and middle gluteal muscles and the vertebral head of biceps femoris removed.

Blood Supply to the Thigh

The external iliac artery gives off the deep femoral artery just prior to passing through the femoral ring to continue as the femoral artery. The deep femoral courses between the sartorius and iliopsoas muscles and then between the latter and the pectineus. After supplying branches to the deep inguinal lymphocenter, the deep femoral artery gives off the large pudendoepigastric trunk. This divides into the caudal epigastric artery and the external pudendal artery. The deep femoral artery continues ventral to the pubis as the medial circumflex femoral artery. This supplies the iliopsoas, pectineus, external obturator muscles, adductor, and semimembranosus. Satellite veins accompany the arteries.

The femoral artery continues distad through the femoral canal accompanied on its caudal side by the femoral vein and cranially by the saphenous nerve. Within the canal the femoral artery gives off the lateral circumflex femoral artery that enters the quadriceps femoris, passing between the vastus medialis and rectus femoris.

The femoral artery directly supplies branches to muscles in this region. At the distal end of the femoral canal, the saphenous artery leaves the femoral artery and passes to the subcutaneous tissues between the gracilis and sartorius muscles. It courses caudodistad, related caudally to the much larger medial saphenous vein. The saphenous nerve accompanies the vessels as they course over the tendon of the gracilis. At the level of the proximal crus, the saphenous artery and medial saphenous vein each divide into cranial and caudal branches. In its course, the saphenous artery supplies the sartorius, gracilis, and adductor muscles as well as fascia and skin.

The next branch of the femoral artery is the nutrient artery of the femur, and then the large descending genicular

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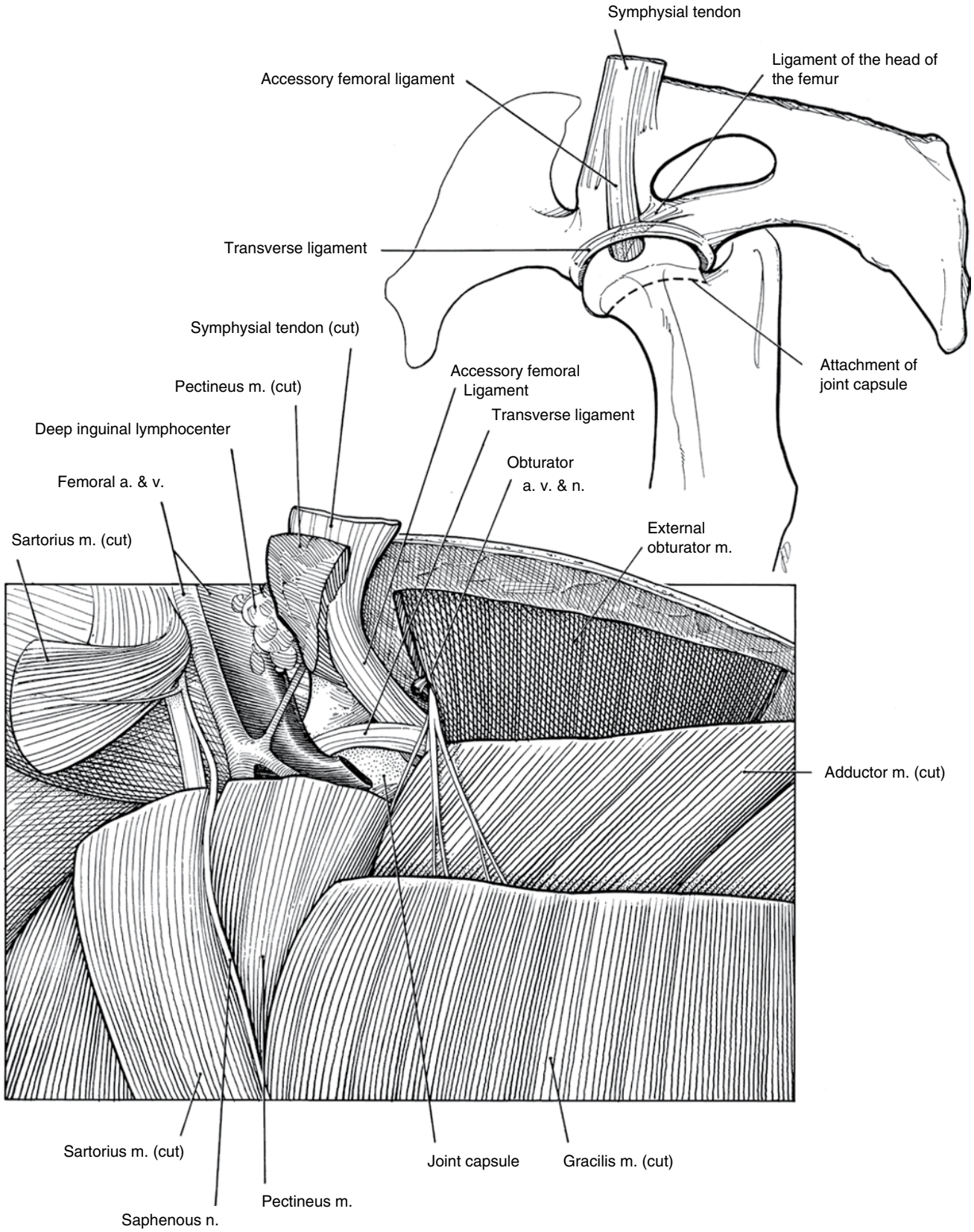


Figure 1.51. Deep dissection of right hip. Ventromedial view.

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artery is detached from the cranial wall of the femoral artery. In the distal third of the thigh, the descending genicular artery courses distocraniad between the sartorius and the adductor, supplying these muscles and terminating in branches to the stifle.

The femoral artery gives off its last branch, the (distal) caudal femoral artery, and continues between the medial and lateral heads of the gastrocnemius as the popliteal artery (Figure 1.46). The caudal femoral artery pursues a short course caudad, giving off muscular branches to the superficial digital flexor and gastrocnemius. Ascending branches from this substantial artery supply thigh and calf muscles. A descending branch of the caudal femoral artery runs distocaudad over the lateral head of the gastrocnemius along with the lateral saphenous vein. The many branches of the caudal femoral artery make anastomotic connections between the obturator artery proximally and the saphenous artery distally.

Hip (Coxofemoral) Joint

The acetabulum of the os coxae is formed where the ilium, ischium, and pubis meet. The lunate surface of the acetabulum, a cup-shaped cavity arcing around a deep nonarticular fossa, articulates with the head of the femur. A fibrocartilaginous rim, the acetabular labrum, increases the articular surface of the acetabulum. The transverse acetabular ligament bridges the labrum across the medially located acetabular notch, binding two ligaments as they emerge from the fovea capitis of the femoral head (Figure 1.51). The ligament of the head of the femur comes from the fovea and attaches in the pubic groove on the ventral aspect of the pubis. The thick accessory femoral ligament passes out through the acetabular notch to blend into the prepubic tendon. It provides partial origin to the gracilis and pectineus muscles.

The capacious joint capsule of the hip attaches to the acetabular labrum and on the neck of the femur a few millimeters from the margin of the femoral head (Figure 1.51). The synovial membrane within the joint wraps around the intracapsular ligaments. An outpocketing of the synovium passes out through the acetabular notch to lie between the accessory femoral ligament and the pubic groove. The fibrous joint capsule is intimately attached to the fascia of the external obturator and deep gluteal muscles. Fat covers the capsule dorsally. The articularis coxae muscle is related to the lateral aspect of the hip joint, detaching some fibers to the joint capsule. During flexion of the hip joint, the articularis coxae tense the joint capsule.

Movements of the Hip Joint

While the hip joint is a ball-and-socket joint, it is capable only of very limited movement beyond flexion and extension. Abduction of the thigh is restricted by the ligament of the head of the femur and the accessory femoral ligament. Adduction is checked by the attachments of the gluteal muscles on the femur. In the normal standing position, the caudolateral part of the head of the femur lies outside the acetabulum. The hip joint is slightly flexed in this position. The range of motion between extreme flexion and extension is only 60°.[16]

Flexor muscles of the hip joint are the gluteus superficialis, tensor fasciae latae, rectus femoris, iliopsoas, sartorius, and pectineus. Extensor muscles of the hip joint are the gluteus medius, biceps femoris, semitendinosus, semimembranosus, adductor, and quadratus femoris. Muscles adducting the thigh include the gracilis, sartorius, adductor, pectineus, quadratus femoris, and obturatorius externus. Slight abduction is exerted on the thigh by all three gluteal muscles. The thigh is rotated laterad by the iliopsoas, external and internal obturators, and the gemelli. Medial rotation is accomplished through the combined action of the adductor and gluteus profundus muscles.

Pelvis

The equine pelvis, like that of other animals, comprises ilium, ischium, and pubis; these bones are individually identifiable in the young but have fused by 10–12 months of age.[16] The acetabulum is formed through contributions from all three bones of the pelvis.

The wing-shaped ilium presents two prominences, visible landmarks on the horse. The dorsally directed tuber sacrale inclines mediad toward its fellow, so that the two sacral tubers come within 2–3 cm over the first sacral spinous process. The ilial wing sweeps ventrolaterad as the tuber coxae, creating the point of the hip. Caudally, the ischial tuberosity presents as a laterally directed ridge to which muscles of the thigh attach.

The pubis and ischium from each side meet ventrally at the pelvic symphysis. In the young animal fibrocartilage joins the bones. Later in life, a synostosis is formed as the cartilage ossifies in a cranial to caudal sequence.

Lymphatic Drainage

Two lymphocenters are involved in the lymphatic drainage of the pelvic limb. The popliteal lymphocenter consists of 3–12 small popliteal lymph nodes embedded about 5 cm deep between the biceps femoris and semitendinosus muscles adjacent to the tibial nerve (Figure 1.44). They may be absent in some horses. The popliteal lymph nodes receive afferent lymphatics from the distal pelvic limb. Efferents drain to the deep inguinal lymphocenter.

The deep inguinal lymphocenter is a mass of lymph nodes 8–12 cm long within the femoral canal. It consists of 16–35 individual lymph nodes. In addition to receiving lymphatic vessels from the popliteal lymphocenter, the lymph nodes of the deep inguinal lymphocenter (Figure 1.50) receive vessels from the caudal abdominal wall and superficial inguinal lymph nodes. Efferent vessels from the deep inguinal lymphocenter are afferent to the medial iliac lymph nodes, located retroperitoneally near the origin of the external iliac arteries.[16]

Stay Apparatus of the Pelvic Limb

The quadriceps femoris muscle and the tensor fasciae latae act to pull the patella, parapatellar cartilage, and medial patellar ligament proximad to the locked position over the medial trochlear ridge of the femur when the limb is positioned to bear weight at rest (Figure 1.52). Through the components of the reciprocal apparatus (cranially,

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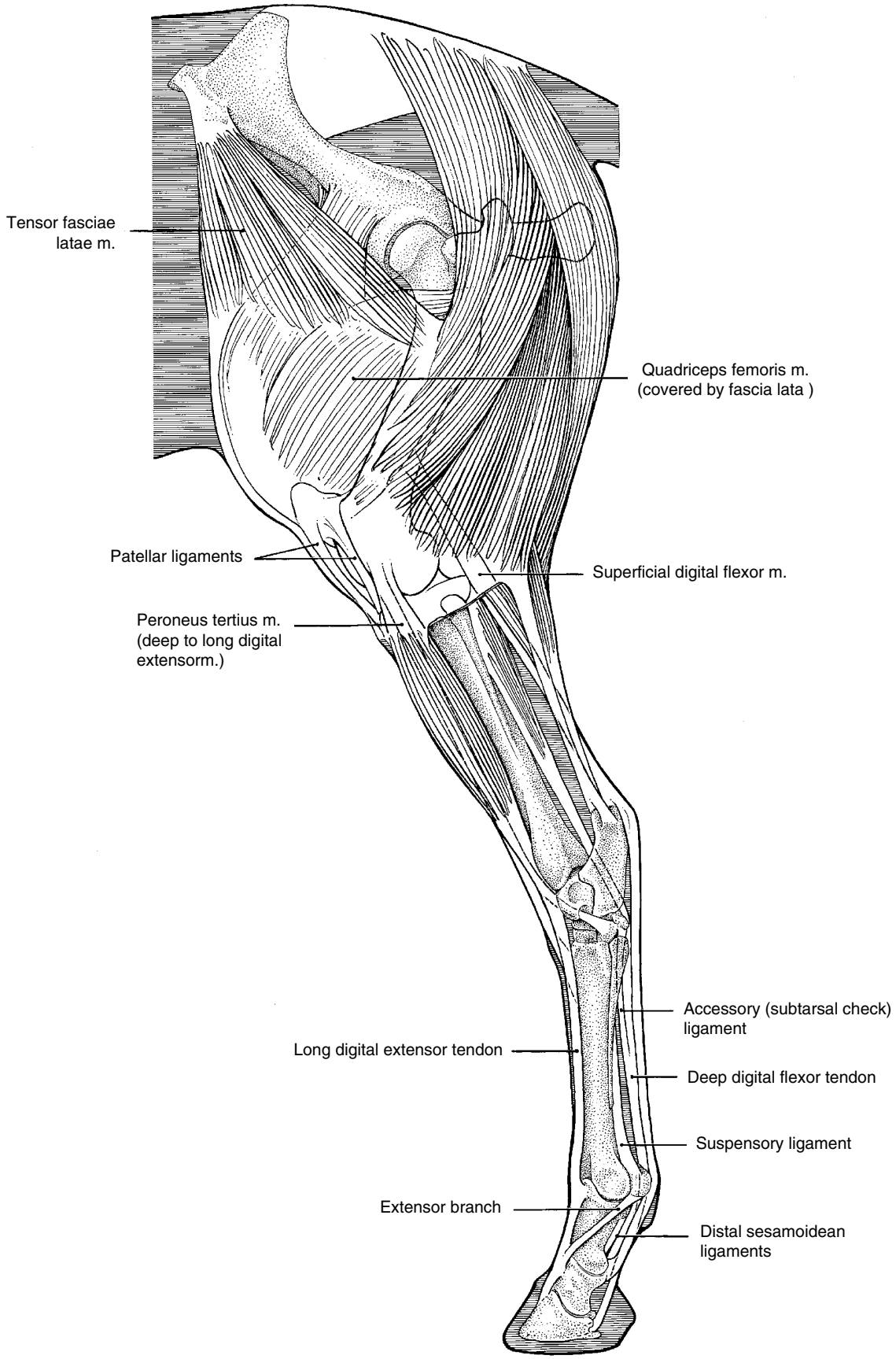


Figure 1.52. Stay apparatus of the pelvic limb.

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fibularis tertius and, caudally, superficial digital flexor), the tarsus is correspondingly locked in extension. Even during sleep, small amount of muscular tone in the quadriceps muscle assures stability of this locked configuration, preventing flexion of the stifle and tarsocrural joints.[39]

Distal to the hock the elements of the stay apparatus are analogous to those of the thoracic limb. The superficial digital flexor is connected proximally to the calcaneal tuber (effectively, its "check ligament"), and the deep digital flexor is anchored via its accessory (tarsal check) ligament to the thick plantar part of the tarsal fibrous joint capsule. Prevention of overextension of the fetlock and interphalangeal joints during the fixed, resting position is accomplished through the support rendered by the digital flexor tendons and the suspensory apparatus (suspensory ligament, proximal sesamoid bones, and their ligaments).

AXIAL COMPONENTS

Vertebral Column

The vertebral formula of the horse is 7 cervical, 18 thoracic, 6 lumbar, 5 sacral, and an inconsistent number of caudal vertebrae (ranging from 15 to 21). There is some individual variation in numbers of other vertebrae, most commonly in the number of lumbar vertebrae where five or seven are sometimes seen (there is an increased incidence of five lumbar vertebrae in Arabian horses[41]). The typical vertebra possesses a ventrally placed, roughly cylindrical body whose cranial and caudal ends articulate with adjacent vertebrae at the intervertebral disc (Figure 1.53). A bony vertebral arch attaches to the body and surrounds the spinal cord. The aperture created within a given vertebrae by the dorsal

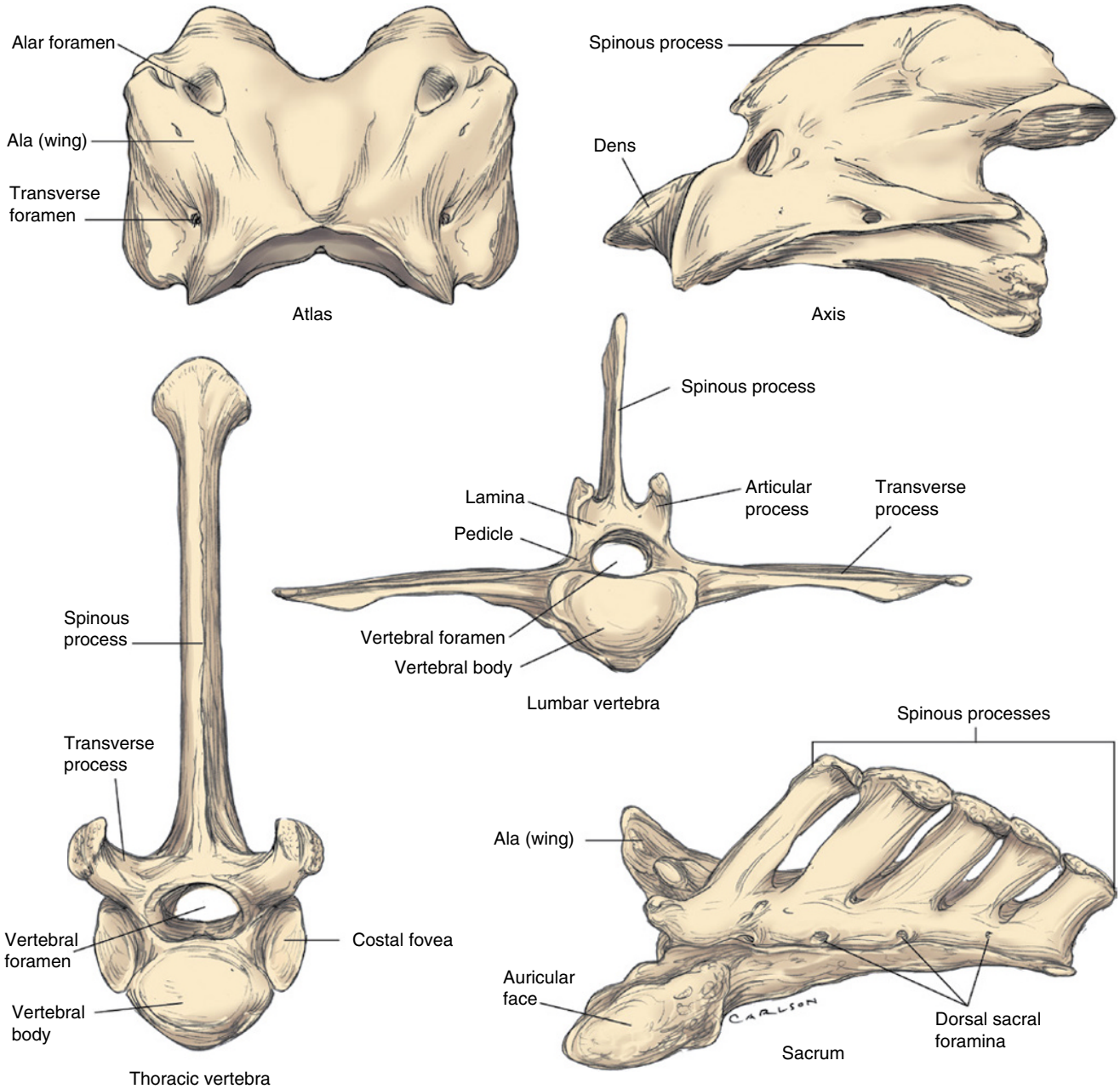


Figure 1.53. Vertebrae.

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aspect of the body and the medial and ventral parts of the arch is the vertebral foramen; where vertebral foramina of adjacent vertebrae are aligned to admit the spinal cord, the resulting passageway is called the vertebral canal. The vertebral canal is widest in the caudal cervical–cranial thoracic region, where it accommodates the cervical enlargement of the spinal cord. A second dilation of the canal occurs in the lumbar region where the lumbosacral enlargement of the cord resides.

The vertebral arch comprises the pedicles and laminae, which together create the "roof" over the spinal cord. The pedicles are the vertical attachments to the vertebral body. The dorsal part of the arch is created by the right and left laminae. The arch is characterized by vertebral notches, indentations on the cranial and caudal aspects of the pedicle. When individual vertebrae are articulated, the cranial vertebral notch of one vertebra abuts the caudal vertebral notch of another, creating an intervertebral foramen through which the spinal nerve emerges from the vertebral canal.

The vertebral arch features other bony processes that bear synovial joints between adjacent vertebrae and that serve as sites of attachment for epaxial muscles. Each vertebra has a single dorsal midline spinous process and two transverse processes that arise near the point at which the pedicle attaches to the body. The dorsal contour of the equine thorax and loin is largely determined by the relative size and prominence of the spinous processes of thoracic and lumbar vertebrae. Arising adjacent to the spinous process are a pair of cranial articular processes and a pair of caudal articular processes.

Cervical Vertebrae

The first two cervical vertebrae are highly modified to meet their specialized function in permitting movement of the head. The first vertebra is the atlas. It lacks the cylindrical body characteristic of other vertebrae, instead taking the form of a bony ring comprising dorsal and ventral arches. The spinous process is likewise absent. The transverse processes are modified into the wings of the atlas. These are robust, bent in a ventrolateral direction, and strongly concave ventrally. Their craniolateral edges form a prominent palpable ridge caudal to the ramus of the mandible. The dorsal aspect of the wing of the atlas bears three foramina: the transverse foramen, the alar foramen, and the lateral vertebral foramen. The cranial aspect of the atlas possesses two deeply concave cranial articular foveae, which form a synovial joint (the atlanto-occipital joint) with the occipital condyles. The caudal articular foveae are also concave and participate in the synovial atlantoaxial joint.

The second cervical vertebra is the axis. The body of the axis is long, and its cranial extremity is modified into a scoop-like projection called the dens, which features a rounded ventral articular surface that articulates with the floor of the atlas. The caudal extremity of the axis' body is deeply concave where it articulates with the body of the third cervical vertebra. The spinous process of the axis is tall and long, modified into a thick midline plate. The transverse processes are small and caudally directed.

The third through seventh vertebrae are similar to one another and follow the basic pattern of most vertebrae. They are progressively shorter from cranial to caudal. Cervical vertebrae three, four, and five bear a distinct ventral crest on their bodies. This crest is diminished in size on the sixth and absent from the seventh cervical vertebra. Articular processes on these cervical vertebrae are large, with prominent oval fovea for articulation between vertebral arches. The cranial articular processes present their foveae in the dorsomedial direction; caudal articular processes have complementary foveae directed in the ventrolateral direction. Transverse processes are broad, each with two thick tubercles for muscular attachment. Transverse processes of the sixth cervical vertebra are especially robust, while those of the seventh are somewhat diminished relative to the other cervical vertebrae. The dorsocaudal aspect of the seventh cervical body features a costal fovea that participates in the synovial articulation of the head of the first rib with the seventh cervical and first thoracic vertebrae. The spinous process of the seventh cervical vertebra is tall compared with other cervical vertebrae.

Thoracic Vertebrae

There are usually 18 thoracic vertebrae in the horse, although on occasion there may be one more or one less than typical. The bodies of the thoracic vertebrae tend to be short with a small vertebral arch dorsally. The spinous processes are relatively tall, with the first four or five increasing in height and more caudal spinous processes gradually decreasing in height until at the level of the 12th thoracic vertebra, after which they are the same height as those of the lumbar vertebrae. The tall spinous processes of those first 12 vertebrae constitute the withers. The dorsal apex of the spines is somewhat expanded and in young horses surmounted by cartilage. The cartilage is replaced by bone as the horse ages, with the cartilages associated with the prominence of the withers persisting the longest at 10 years or more. The anticlinal vertebra is defined as the one whose spinous process is perpendicular to the long axis of the vertebral column. The spinous processes of more cranial vertebrae incline caudad, while those of more caudal vertebrae incline craniad. In the horse, the anticlinal vertebra is usually the 16th and occasionally the 14th.

The vertebral bodies possess cranial and caudal costal foveae for articulation with the heads of ribs, excepting the last thoracic vertebra, which features only cranial costal foveae. Transverse processes are irregular, largest in the cranial thoracic vertebrae and gradually decreasing in size toward the lumbar region. Mammillary processes appear in the caudal thoracic region. These are directed craniad and arise primarily from the transverse processes. In the most caudal of the thoracic vertebrae, they arise in common from the transverse and cranial articular processes and may for this reason be called mamilloarticular processes.

Lumbar Vertebrae

There are usually six lumbar vertebrae, although five and seven have also been reported. The cylindrical bodies of the lumbar vertebrae are somewhat flattened dorsoventrally, especially the last three. Except for the seventh and sometimes the sixth lumbar vertebrae, a ventral crest is prominent. The spinous processes project

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slightly craniad. The vertebral arches tend to overlap dorsally, except at the L5–L6 and L6–S1 interspaces where the larger interarcuate spaces are much larger and clinically accessible. The cranial and caudal articular processes articulate in an approximation of the sagittal plane, an orientation that allows for a very slight degree of flexion and extension of the vertebral column but prevents lateral flexion. The transverse processes of the lumbar vertebrae are large and blade-like. They project laterad. The caudal aspect of the fifth transverse process articulates with the cranial aspect of the sixth. The caudal aspect of the sixth transverse process features a large concave facet through which it articulates with the sacrum.

Sacrum

The equine sacrum is a single bone formed through fusion of embryologically distinct sacral vertebrae, generally five of these, with four, six, and seven sacral vertebrae also being reported.[41] Fusion is usually complete by 5 years of age. The sacrum is triangular and gently curving so as to present a slightly concave ventral aspect. Intervertebral foramina are transformed by the fusion of adjacent vertebrae into a row of four dorsal sacral foramina and four ventral sacral foramina, through which pass dorsal and ventral branches, respectively, of the sacral spinal nerves. The spinous processes remain individually distinct and incline slightly caudad. The second through fifth spinous processes end in slight enlargements that are not uncommonly bifid.

The first sacral vertebra gives rise to the wings of the sacrum. Their articular surfaces face dorsolateral to articulate with the auricular surface of the ilium. The ventral aspect of the first sacral vertebra is slightly rounded, forming the promontory of the sacrum, the point from which the conjugate diameter (the dorsal–ventral distance) of the pelvic canal is measured.

Caudal Vertebrae

Although there is considerable individual variation, the average horse has 18 caudal vertebrae. Only the first three or so have vertebral arches, the remaining being represented by cylindrical bodies only. The first caudal vertebra is not uncommonly fused with the sacrum, especially in old horses.

Vertebral Articulations

Excluding the atlantoaxial joint (a pivot joint), the joints of the vertebral column all permit small amounts of flexion, extension, lateral flexion, and limited rotation. These movements are especially limited through thoracic and lumbar regions, but the cervical vertebral column is capable of more extensive movement. Intervertebral discs of fibrocartilage are interposed between adjacent vertebral bodies. Further stabilization is provided to the vertebral column by (1) the continuous dorsal and ventral longitudinal ligaments on their respective surfaces of the vertebral bodies; (2) a supraspinous ligament that passes along the dorsal aspect of the spinous processes of thoracic, lumbar, and sacral vertebrae; and (3) interspinal ligaments that pass between adjacent spinous processes. In the thoracic region, intercapital ligaments pass transversely between the heads of contralateral ribs over the dorsal aspects of the intervertebral discs. Articulations between articular processes on vertebral arches are true synovial joints. In the cervical region, these constitute broad plates, oriented in a nearly horizontal plane to permit significant lateral bending. Articular facets on the cranial articular processes face dorsomediad, while the complementary facets on the caudal articular processes face ventrolaterad. Synovial joints also exist between the transverse processes of the fifth and sixth lumbar vertebrae and between the transverse processes of the sixth lumbar vertebra and the wings of the sacrum.

Sacroiliac Region

The axial skeleton and appendicular skeleton of the hindlimb are united at the sacroiliac joint (Figure 1.54). This planar joint is created by the auricular face of the wings of the sacrum, which are oriented dorsolaterad, and the auricular face of the wings of the ilia, which angle ventromediad. This joint is histologically synovial but is capable of only extremely limited gliding movement. Its principle purpose is most likely absorption of some of the concussive forces transmitted through the appendicular skeleton to the vertebral column. The joint capsule is close-fitting and is substantially reinforced by

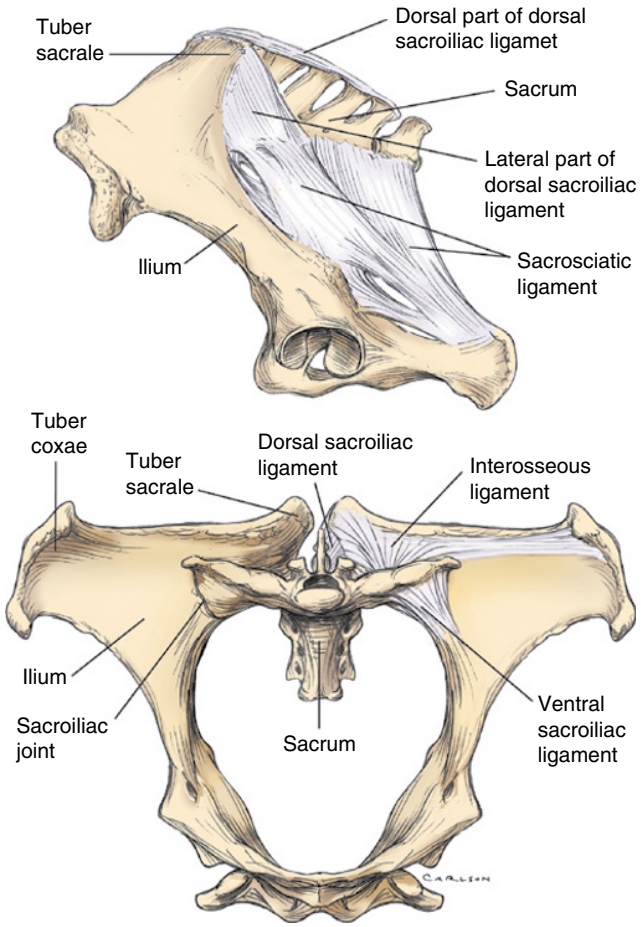


Figure 1.54. Sacroiliac joint. Lateral (top) and cranial (bottom) views.

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a series of sacroiliac ligaments that contribute markedly to the overall stability of the joint and probably act to transfer most of the weight of the trunk to the pelvic limbs. These ligaments can be summarized as the ventral sacroiliac ligament, dorsal sacroiliac ligament, and interosseous ligament.

The ventral sacroiliac ligament surrounds the joint and fills the space between the ilium and the ventral aspect of the wing of the sacrum. The dorsal sacroiliac ligament presents two distinct portions. One (dorsal or short part) arises from tuber sacrale and inserts on the spinous processes of the sacral vertebrae. The other (lateral or long part) arises from tuber sacrale and the caudal edge of the ilial wing and inserts along the lateral aspect of the sacrum. From here it blends ventrad into the broad sacrosciatic ligament that fills the space between the pelvis and sacrum. The interosseous ligament (interosseous sacroiliac l.) consists of strong, vertically oriented fibers between the ventral part of the wing of the ilium and the dorsal aspect of the wing of the sacrum.

Ligamentum Nuchae

The topline of the neck is in part determined by the presence of the ligamentum nuchae (nuchal ligament), which in horses extends from its cranial attachments on the external occipital protuberance to the spinous process of the third or fourth thoracic vertebra. Both parts of the nuchal ligament (funicular and laminar) are paired. The rope-like funicular part is connected to sheets that compose the laminar portions. These midline elastic sheets arise from the second through fifth cervical vertebrae and insert on the spines of the second and third thoracic vertebrae.[24] Bursae are consistently found between the funicular part of the nuchal ligament and the atlas and between the nuchal ligament and the second thoracic spine. These are the bursa subligamentosa nuchalis cranialis and the bursa subligamentosa supraspinalis, respectively. A third bursa (bursa subligamentosa nuchalis caudalis) is inconsistently found between the nuchal ligament and the spine of the axis.[13,16]

Muscles of the Trunk and Neck

Muscles of the torso (neck, trunk, and tail) may be roughly divided into those dorsal to the transverse processes (epaxial muscles) and those ventral to the transverse processes (hypaxial muscles). The dorsal branches of the spinal nerves innervate the epaxial muscles while hypaxial muscles receive their innervation from the ventral branches.

The epaxial muscles are extensors of the vertebral column and are themselves divided into three parallel bundles of fascicles: from lateral to medial these are the iliocostalis system, the longissimus system, and the transversospinalis system (Figure 1.55). The iliocostalis system (named for its attachments to the ilium and ribs) does not extend into the neck. The others continue into the cervical region and are associated with additional distinct muscles. Of these, the splenius muscle is most superficial (Figure 1.56). The splenius possesses capital and cervical parts. Both arise from the third, fourth, and fifth thoracic spines and from the funicular part of the nuchal ligament, and they insert on the nuchal crest of the skull, the wing of the atlas, and the transverse processes of the third, fourth, and fifth cervical vertebrae. The splenius extends the neck and elevates the head, and it is largely the rhythmic contraction of this powerful muscle that creates the thrusting movements of the neck during the gallop.[27]

Deep to the splenius are a number of shorter muscles extending from the cervical vertebrae to the skull; these "specific activators of the head" are important in giving the head and neck its considerable range of motion.

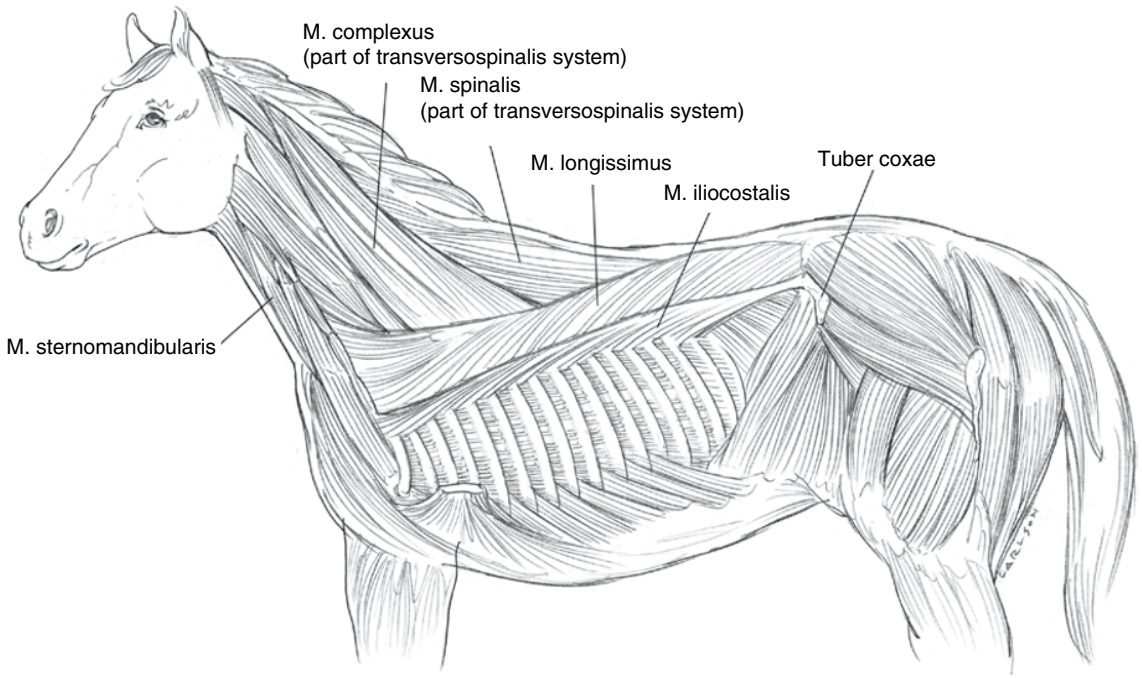


Figure 1.55. Deep muscles of trunk.

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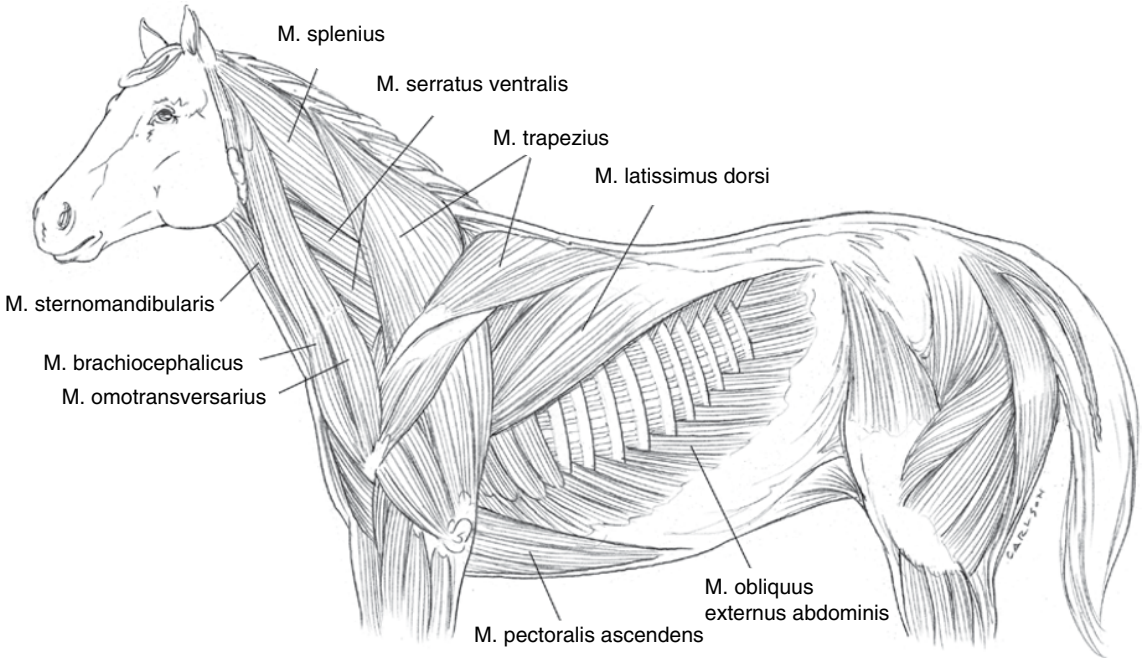


Figure 1.56. Superficial muscles of trunk. Cutaneous muscles have been removed.

Among those dorsal to the transverse processes are the cranial and caudal parts of the obliquus capitis muscle. The cranial part bridges between the nuchal crest and the cranial border of the wing of the atlas. The caudal part is a large, powerful rotator of the atlantoaxial joint. It attaches to the cranial border of the wing of the atlas and the vertebral arch of the axis.

Deep to the oblique muscles are a series of straight muscles that link the skull to the atlas and axis. The major part of rectus capitis dorsalis attaches on the caudal aspect of the occipital bone and the spinous process of the axis; its minor portion lies dorsal to the dorsal atlanto-occipital membrane, bridging between occipital bone and the wing of the atlas. Rectus capitis ventralis runs from the base of the skull to the ventral arch of the atlas. Rectus capitis lateralis attaches from the wing of the atlas to the paracondylar process. These last two muscles are relatively weak in the horse.

The longus capitis muscle bridges between the transverse processes of C2 through C4 or C5 and the ventral skull.

Activators of the head that are above the transverse processes will extend the atlanto-occipital and atlantoaxial joints; those ventral to the transverse processes flex those joints. When acting unilaterally, all of these muscles have the ability to turn the head laterally.

The longissimus group is described as having lumbar, thoracic, cervical, atlantal, and capital portions. It is most robust in the lumbar region, where it gives a well-conditioned horse's back its typical rounded appearance.

Hypaxial muscles of the trunk (the psoas minor, quadratus lumborum, and the four abdominal muscles on each side) act to flex the vertebral column during the gallop. Epaxial muscles extend the vertebral column. When they contract unilaterally, both hypaxial and epaxial muscles create lateral movement of the trunk and neck.

In the ventral neck, the equine m. longus colli is particularly well developed relative to other domestic species. The cervical portion of this muscle arises from the transverse processes and bodies of the third through sixth cervical vertebrae in paired bundles that converge to an insertion on the preceding vertebral bodies, sometimes bridging more than one intervertebral space. The most cranial attachment is on the ventral aspect of the atlas. The thoracic portion of the longus colli arises on the lateral vertebral bodies of thoracic vertebrae one through six, passing craniad to insert on the transverse processes of cervical vertebrae six and seven.

The omohyoideus muscle is well developed in the horse. It arises from an aponeurosis in the fascia near the shoulder joint. Its muscle belly is closely attached to the deep side of the brachiocephalicus until the neck's midpoint, where the omohyoideus becomes evident as a distinct muscle as it passes craniad deep to the sternocephalicus muscle. Near its insertion on the lingual process, it fuses with its partner from the contralateral side and with the sternohyoideus.

The right and left sternomandibularis muscles of the horse are fused on midline near their origin on the manubrium. Near mid-neck, the two halves separate, moving from their position ventral to the trachea to a more lateral location. As the muscle approaches its insertion on the sternomandibular tuberosity on the ramus of the mandible, it narrows to a distinct tendon that is visible in the cranial neck just caudal to the caudal border of the mandible. The tendon of insertion is classically considered one side of Viborg's triangle.

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CHAPTER

Examination for Lameness

HISTORY, VISUAL EXAM, AND CONFORMATION

Gary M. Baxter and Ted S. Stashak

INTRODUCTION

Lameness is an indication of a structural or functional disorder in one or more limbs or the axial skeleton that is evident while the horse is standing or at movement.[29] Lameness can be caused by trauma (single event or repetitive work), congenital or acquired anomalies, developmental defects, infection, metabolic disturbances, circulatory and nervous disorders, or any combination of these. The diagnosis of lameness requires a detailed knowledge of anatomy, an understanding of kinetics and kinematics, and an appreciation for geometric design and resultant forces. It is important to differentiate between lameness resulting from pain and nonpainful alterations in gait, often referred to as "mechanical lameness," and lameness resulting from neurologic (nervous system) dysfunction.[8,35] Lameness due to pain is most common in the horse.

A complete lameness examination helps to differentiate among the many types of lameness problems in the horse. The objectives of a lameness examination are to determine:

1. Whether the horse is lame
2. Which limb or limbs are involved
3. The site or sites of the problem
4. The specific cause of the problem
5. The appropriate treatment
6. The prognosis for recovery

The steps to perform a routine or traditional lameness examination include:

1. Complete history including signalment and use
2. Visual exam of the horse at rest
3. Palpation of the musculoskeletal system including hoof tester examination of the feet
4. Observation of the horse in motion (usually at a straight walk and trot/lope followed by circling)
5. Observation of the horse under saddle or in work if necessary
6. Manipulative tests such as flexion tests
7. Diagnostic anesthesia if necessary
8. Diagnostic imaging

Palpation of the limbs and axial skeleton and hoof tester examination of the feet are usually performed prior to exercising the horse. However, some clinicians prefer to observe the horse at exercise prior to palpation of the musculoskeletal system. Diagnostic anesthesia and imaging often follow to document the location of the pain, specific cause of the problem, extent of injury, and prognosis for recovery.

ADAPTIVE STRATEGIES OF LAME HORSES

Horses adapt to lameness with compensatory movements of specific body parts.[11,12,39] With most lameness conditions, the horse attempts to "unload" the lame limb during weight-bearing or the stance phase of the stride. In a kinetic study of forelimb lameness, the vertical force peak had the highest sensitivity and specificity for lameness classification.[12,24] Horses accomplish this by abnormal movement of a body part (head nod or pelvic hike), weight shifting (to the contralateral or diagonal limb or torso), change in joint angles (lack of fetlock extension), and alterations in foot flight.[39] For example, hyperextension of the fetlock has been shown to be an indirect

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Figure 2.1. Chronic hindlimb lameness that has resulted in a wide flat foot on the sound left hindlimb and a narrow, upright hoof on the lame right hindlimb.

measure for the vertical ground reaction force and is reduced in the lame limb at the stance phase proportionally to the degree of lameness.[12,32,39] In addition, in horses with chronic lameness, the limb with the flatter hoof exhibits higher vertical loads because it is the non-lame limb (Figure 2.1).[39] Detecting these compensatory movements is an integral part of diagnosing lameness in the horse. The most consistent compensatory movements that are observed are the vertical displacement and acceleration of the head in forelimb lameness and of the sacrum and tuber coxae in hindlimb lameness.[11,25,39] Although overlap of these movements can occur (head movement with hindlimb lameness and pelvic movement with forelimb lameness), they tend to occur primarily in moderate to severe lameness. In addition, more subtle lameness causes fewer compensatory changes, making lameness diagnosis more difficult, and problems within the axial skeleton (back) may also alter the movement of the limbs.[19,39] Although some of these gait changes may be difficult to appreciate during visual examination of a lame horse, it should be remembered that the primary adaptive strategy of the horse is to redistribute load to compensate for pain in a limb without causing an overload situation in other limbs.[39]

CLASSIFICATION OF LAMENESS

There are a variety of ways to classify lameness in the horse. In most cases there is a primary or baseline lameness that contributes to the most obvious gait abnormalities. Compensatory, secondary, or complementary lameness results from overloading of the other limbs as a result of the primary lameness.[33] Lameness may also be classified according to when it occurs (or is best observed) within the stride. The different classifications of lameness are defined below:

1. Supporting limb lameness: Apparent when the foot first contacts the ground or when the limb is supporting weight (stance phase). Injury to bones, joints, soft tissue support structures (e.g. ligaments and flexor tendons), and the foot are considered causes of this type of lameness. This is by far the most common type of lameness identified in the horse.
2. Swinging limb lameness: Evident when the limb is in motion. A variety of pathologic changes may be the cause, and the majority of these problems are thought to involve the upper limbs or axial skeleton.[35]
3. Mixed lameness: Evident both when the limb is moving (swing phase) and when it is supporting weight (stance phase). Mixed lameness can involve any combination of structures affected in swinging or supporting limb lameness.
4. Primary or baseline lameness: Most obvious lameness or gait abnormality that is observed before flexion or manipulative tests.[7,33] This can be complicated by lameness in multiple limbs, but in most cases the lameness that is the worst is considered the primary lameness. Evaluation of the primary lameness should be performed initially before scrutinizing complementary lameness problems.
5. Compensatory or complementary lameness: Pain in a limb can cause uneven distribution of weight on another limb or limbs, which can produce lameness in a previously sound limb. It is common to have complementary lameness produced in a forelimb as a result of lameness in the opposite forelimb.[35] Also, a lameness in a hindlimb can mimic a forelimb lameness on the ipsilateral side (right hind and right fore). Quantitative lameness evaluations suggest that an apparent lameness in the forelimb and hindlimb on the same side often suggests a primary hindlimb lameness and a false compensatory forelimb lameness.[25] Additionally, lameness in one hindlimb may contribute to lameness in the opposite hind.

Even minor changes in weight-bearing can produce complementary lameness at high speeds, especially over long distances. The feet, suspensory ligament, sesamoid bones, hocks, and flexor tendons seem to be affected most commonly.[35] Complementary lameness in the same limb usually results when excess stress occurs to an otherwise healthy structure in an attempt to protect a painful region in that limb. For example, a horse with navicular syndrome often lands toe first, potentially contributing to toe bruising and/or osteitis.

By observing the gait from a distance, one can usually determine whether the lameness is supporting limb, swinging limb, or mixed. Some conditions that cause supporting limb lameness may cause the horse to alter the movement of the limb to protect the foot when it lands. This can be mistaken for swinging limb lameness. Because of this and other adaptive strategies that occur in lame horses, some clinicians feel that mixed lameness occurs most commonly in horses.[33]

Character of the Stride

The character of the stride of a limb is important to the diagnosis of lameness. When observing the stride, the following characteristics should be observed:

The phases of the stride: The stride consists of a cranial phase and a caudal phase. The cranial phase of the stride is in front of the footprint of the opposite limb, and the caudal phase is behind it (Figure 2.2). With lameness, the cranial or caudal phases may be shortened. If the cranial phase is shortened, there may be a compensatory lengthening of the caudal phase, and vice versa.

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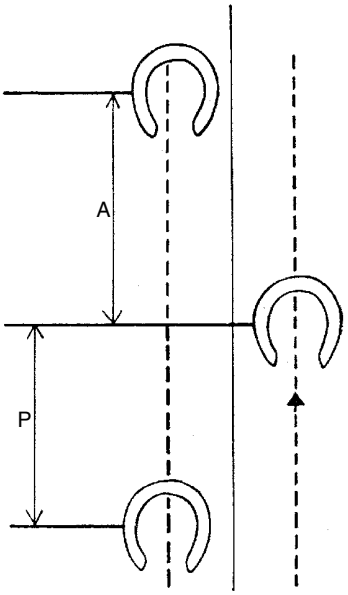


Figure 2.2. Phases of the stride. (A) The cranial phase of the stride is the half of the stride in front of the print of the opposite foot. (P) The caudal phase of the stride is the half of the stride in back of the print of the opposite foot.

Alterations in phase of the stride are best viewed from the side of the horse. Potential causes for a shortened cranial phase of the stride include navicular syndrome, joint problems such as osteoarthritis (OA), shoulder problems, and bone spavin. Causes of shortened caudal phase of the stride may include digital annular ligament desmitis and tendonitis or tenosynovitis involving the flexor tendons.[35]

The arc of foot flight: The arc of foot flight is usually changed when there is pain anywhere in the limb. The arc of one foot should be compared with that of the opposite member when viewed from the side. In some cases, the arc is changed in both forefeet (bilateral navicular syndrome, laminitis, etc.) or in both hindfeet (bilateral bone spavin). In the hindlimb, the arc may be changed enough to cause the toe to drag when the limb is advanced because of reduced flexion of the hock or stifle joints. Most horses exhibiting a decrease in height of the foot flight arc also show alteration in their phase of stride. For subtle alterations in the hoof flight arc of the hindlimb, it is sometimes helpful to observe the horse from the rear to compare the extent and duration that the horse presents the solar surfaces of both feet to the observer. In general, the less sole that can be seen, the lower the arc of foot flight in the limb.[35]

How the foot lands: When a painful condition is present in the foot, the horse usually indicates the pain by placing its weight opposite to the pain. For example, with pain in the heel region, the horse may only toe-touch. If the lesion is in the lateral portion of the sole, the weight is carried on the medial side of the foot, and vice versa.

Joint extension: The degree of extension of the fetlock (how close the back of the fetlock approaches the ground) during weight-bearing is a sensitive indicator of how much weight the horse is willing to bear on that limb. Reduction in fetlock extension during weight-bearing usually indicates the lame limb (Figure 2.1). This sign can be observed at a walk and/or a trot.

Joint flexion angles: Joint flexion angles are best viewed from the side and may or may not be associated with alterations in the hoof flight arc and phase of stride. One limb is compared with the other and the degree of flexion is assessed. In some cases, horses compensate with an increased flexion of the unaffected limb.

Head and pelvic movement: Horses use their pelvis or hemipelvis with hindlimb lameness similar to how they use their head and neck with forelimb lameness. Both are used as a method to "unweight" the lame limb, and the amount of movement of each is often proportional to the degree of lameness. Movement of the head and pelvis (or hemipelvis) is best observed from the side and should be correlated with gait changes.[7]

Other Lameness Factors

It is generally considered that the majority of lameness problems in horses occur in the forelimb due to the increased weight-bearing on the forelimbs (60%–65%) and the shock of landing that the forelimbs absorb during movement.[7,35] The hindlimbs propel the horse during movement and carry less of the overall weight. This results in greater concussion to the structures of the forelimbs. However, breed and use can alter this typical relationship. For instance, horses that pull carts or perform events such as dressage, cutting, and reining, which place greater stress on the hindquarters, often have a higher percentage of hindlimb lameness. Many hindlimb lameness problems involve the hock and/or stifle. Horses within a discipline that do different activities may also have different limbs that are affected most commonly. For instance, horses used for heading in team roping have more problems in the right forelimb, and horses used for heeling have more bilateral hindlimb lameness problems.[13] Although there is considerable overlap, common lameness conditions that are associated with the specific type of work should be suspected first (Table 2.1). See Chapter 9 for further information on occupation-related lameness conditions.

It is also believed that approximately 95% of lameness in the forelimb occurs distal to the carpus.[35] Close evaluation of the distal limb should be performed in any horse with forelimb lameness before considering an upper limb problem, unless a more proximal condition is obvious. In the hindlimb, approximately 80% of lameness problems are thought to involve the hock or stifle.[35] Therefore, after preliminary examination of the foot and distal limb, the hock and stifle should be closely evaluated until conditions of those two regions can be eliminated.

Multiple other factors that may contribute to lameness should also be considered. Horses that are improperly or irregularly shod may become lame. Surfaces that are too soft, too hard, slippery, or rocky may aggravate conformational imperfections or may be the outright cause of lameness. Improper conditioning resulting in muscle fatigue is considered to be a common cause of performance-related injuries. Young horses in work may develop lameness problems more easily than older horses due to the immaturity of their musculoskeletal system. With the emphasis on racing and showing young

Table 2.1. Occupation-related lameness problems (See Chapter 9 for further information.)

Occupation	Lameness Conditions
TB and QH racehorse (Fatigue-associated repetitive overuse)	Foot bruising, quarter cracks, heel pain Forelimb fetlock synovitis and fractures Carpal synovitis and fractures Bucked shins in young horses Fatigue/stress fractures—MC/MT, P1, humerus, tibia, pelvis SDFT tendonitis Suspensory injuries Coffin joint synovitis/OA—QH Proximal suspensory desmitis—QH Catastrophic fractures—sesamoids, P1, MC/MT, and humerus Palmar/plantar osteochondral disease (POD)
STD racehorse (hindlimb > forelimb)	Front feet bruising and corns Hindlimb fetlock synovitis and fractures Carpal synovitis and fractures (C3) Fatigue/stress fractures—similar to TB and QH but tibia most common Suspensory injuries especially PSD, SDFT tendonitis Hock, stifle, and sacroiliac problems
Endurance horse	Muscle disorders including tying-up and muscle spasm, cramps, and strains Forelimb and hindlimb suspensory desmitis Foot bruising, P3 osteitis, and laminitis Fetlock synovitis/OA SDFT tendonitis
Show/pleasure horse	Navicular syndrome/disease Coffin joint synovitis/OA Forelimb and hindlimb PSD Distal tarsitis or bone spavin Lumbar and sacroiliac problems
Western performance horses (Cutting, reining, roping, barrel racing, rodeo, gymkhana, and ranch horses)	Navicular syndrome/disease including DDFT injuries Phalangeal fractures (primarily P2) Pastern ringbone Fetlock and carpal synovitis/OA Distal tarsitis or bone spavin Stifle synovitis/OA Forelimb and hindlimb PSD Thoracolumbar myositis Hindlimb muscle strains
Jumping/dressage/eventing	Navicular syndrome/disease including DDFT injuries Suspensory branch injuries Forelimb and hindlimb PSD Fetlock synovitis/OA SDFT tendonitis Distal tarsitis or bone spavin Stifle injuries Thoracolumbar myositis—back problems Sacroiliac problems
Draft horses	Hoof cracks and laminitis Subsolar abscesses, canker, and thrush Ringbone Sweeny Bone and bog spavin Stifle synovitis/OA Shivers and PSSM
Gaited horses (Hindlimb > Forelimb)	Distal tarsitis or bone spavin Stifle synovitis/OA Thoracolumbar myositis/back problems DSLD

TB, Thoroughbred; QH, Quarter Horse; STD, Standardbred.
P1, first phalanx; P2, second phalanx; P3, third phalanx; MC/MT, metacarpus/metatarsus; C3, third carpal bone; PSD, proximal suspensory desmitis; SDFT, superficial digital flexor tendon; OA, osteoarthritis; PSSM, polysaccharide storage myopathy; DSLD, degenerative suspensory ligament desmitis.

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horses, many lameness problems may be produced that would not occur in older, more mature horses.

SIGNALMENT AND USE

Patient age and use are important considerations when determining potential lameness conditions. For example, an aged crossbred horse used for ranch work, occasional rodeo performance, and trail riding will have a higher incidence of problems associated with the forefeet, low-motion joints (e.g. pastern and distal tarsal joints), and ligaments. In contrast, a young racehorse will often have lameness problems associated with high-motion joints (e.g. carpus and fetlock), sprain/strain of flexor support structures, and stress-related fractures. Horses used for competitive trail or endurance riding often sustain a higher incidence of sprain/strain injuries, tendonitis, and stress-related fractures to the phalanges. Young cutting horses appear to be prone to stifle issues, and any young horse just beginning training may become lame from developmental orthopedic-related problems.

HISTORY (ANAMNESIS)

A detailed medical history should be obtained on every horse. Records should include specific information regarding the duration and intensity of the lameness, specific symptoms, the activity immediately preceding the lameness, and any previous treatments or therapies employed. The following questions should be asked:

1. What activity does the horse perform and did the lameness occur during this activity?
2. How long has the horse been lame? (Acute vs. chronic injury.)
3. Has the horse been rested or exercised during the lameness period? (The horse may not exhibit the same lameness if it has been rested.)
4. Has the lameness worsened, stayed the same, or improved? (May indicate severity of the problem.)
5. Was the cause of the lameness observed? (This should include the character of the lameness at the onset.)
6. Does the horse warm out of the lameness?
7. When do you notice the lameness most consistently?
8. What treatments have been given, and have they helped? (Response to treatment may help predict the prognosis of the case but may also mask the severity of lameness and give a false impression of recovery.)
9. When was the horse last shod or trimmed? (Lameness directly after shoeing may suggest that the feet were trimmed too short or a nail was driven into or near sensitive tissue.)
10. What are the abnormalities that the owner or trainer observe or feel when they ride the horse or watch the horse go?

VISUAL EXAMINATION AT REST

A careful visual examination is made with the horse standing squarely on a flat surface at rest.[8,35] This should be done at a distance and then up close, viewing the horse from all directions. From a distance, the body type is characterized (stocky vs. slender); conformation is noted; and body condition and alterations in posture, weight shifting, and pointing are also noted. It is important to look for changes in contour of the limbs and asymmetry between limbs.

Under normal circumstances, the forelimbs bear equal weight and are opposite each other. With bilateral forelimb involvement, the weight may be shifted from one foot to the other, or both limbs may be placed too far out in front of the horse. In the hindlimbs, it is normal for the horse to shift its weight from one limb to the other. If the horse consistently rests one hindlimb and refuses to bear weight on it for a length of time or cannot be forced to bear weight on it at all, lameness in that hindlimb should be considered. Even small alterations in limb positioning and posturing may suggest a potential problem and should be investigated during the lameness examination.

At close observation, each limb and muscle group should be observed and compared to its opposing member for symmetry. Feet are observed for abnormal wear, hoof cracks, imbalance, size, and heel bulb contraction (Figure 2.3). All joints and tendons and their sheaths are visually inspected for swelling, and the muscles of the limbs, back, and rump are observed for swelling and atrophy. Comparing one side to the other is most important. Each abnormal finding should be ruled out as a cause of lameness during exercise and palpation examination. For the forelimbs, the limb with the narrowest (smallest) foot and highest heel with varying degrees of extensor muscle atrophy is usually the lame or lamest (if the problem is bilateral) limb.[7] The foot is smaller due to chronic alteration in weight-bearing, and the muscle atrophy results from a reluctance to extend that limb. For the hindlimb, atrophy of the middle gluteal and/or gracilis muscles usually indicates the lame limb (Figures 2.4 and 2.5). Generally, if one tuber sacrale is higher than the other and/or the pelvis appears tilted, the horse will usually have an asymmetrical gait (Figure 2.6).



Figure 2.3. Horse with two different front feet: low heel and long toe on the left fore and a more upright hoof conformation on the right front. Unlike many horses with this hoof conformation, this horse was lame on the left forelimb.

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Figure 2.4. Example of atrophy of the inner and outer thigh muscles of the left hindlimb that occurred secondary to an upper hindlimb lameness.



Figure 2.5. Example of atrophy of the gluteal muscles (left hindlimb) that often accompanies pelvic fractures in horses.



Figure 2.6. Rear view of the pelvis of a horse with a history of an acute onset of right hind lameness. There is asymmetry to the sacroiliac region visible and pain on firm palpation of the right tuber sacrale.

CONFORMATION

Simultaneously while performing a visual examination on any horse presented for lameness, the conformation of the horse, especially the limbs, should be assessed. It is important to have a basic understanding of conformation and how abnormalities in conformation may contribute to alterations in limb movement and lameness. Conformation refers to the physical appearance and outline of a horse as dictated primarily by bone and muscle structures.[22,29] It is often a subjective assessment based on what a particular breed may consider to be "ideal."[10] However, it can be difficult to specifically define *ideal* or *normal* conformation because the guidelines depend on the classification, type, breed, and intended use of the horse.[29,30] Despite these difficulties, it is well recognized that certain conformation traits may predispose to lameness and these should be observed during the initial visual exam of any lame horse. For instance, poor forelimb conformation was found to be a predisposing factor for young Standardbred racehorses that developed lameness in the middle carpal joint.[37] The trick becomes determining which abnormal conformational traits are contributing to lameness in any given horse because many conformational traits are not always related to performance and unsoundness.[21,30] One study that evaluated conformational traits of Thoroughbred yearlings found only weak associations between performance and conformation that could not be accounted for by the very strong relationship between pedigree and conformation.[28] More such studies that attempt to evaluate the association between limb conformation and specific lameness conditions are needed to truly determine the effect of conformation on lameness in performance horses.

Ideally, conformation assessment of a lame horse should relate to function,[9] but objective studies relating specific conformational traits to performance or lameness problems can be difficult to perform.[10,21,23] To complicate things more, while many lameness problems occur in the lower limbs, the causative factors may be located in the upper limbs or body; therefore, overall conformation must be considered together with limb conformation. Objective techniques to assess conformation as it relates to movement and lameness in horses are needed to truly quantify its contribution to lameness. Furthermore, conformation is often a subjective assessment, and improved techniques to quantify conformation as it relates to lameness are needed.

Types and Breeds

Horses are broadly classified as draft horses, light horses, or ponies. Classifications are further divided by type according to overall body style and conformation

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and the work for which the horse is best suited. Light (riding and driving) horses are by far the most common and can be described as one of several types: pleasure horse, hunter, stock horse, sport horse, animated (show) horse, and racehorse.

A "breed" is a group of horses with common ancestry and usually strong conformational similarities. In most cases, a horse must come from approved breeding stock to be registered with a particular breed. If a horse is not eligible for registration, it is considered to be a "grade" or "crossbred" horse. Several breeds can have similar makeup and be of the same type. For example, most Quarter horses, Paint horses, and Appaloosas are considered to be stock horse types. In addition, racing Quarter horses often have considerable Thoroughbred background. Some breeds contain individuals of different types within the breed; Thoroughbreds can be of the race, hunter, or sport horse type. In addition, crossbreeding horses of different breeds to attain characteristics for a specific sport is not uncommon.

Systematic Evaluation of Conformation

Although breed characteristics may vary, the process of evaluating any horse should be similar. A general systematic assessment of the horse's four functional sections is made (head and neck, forelimbs, trunk, and hindlimbs), giving each area approximately equal importance (Figure 2.7).[36] The horse's specific conformational components or traits are then evaluated in more detail.

The evaluation begins by viewing the horse from the near (left) side in profile and assessing balance by comparing the forehand (head, neck, and forelimbs) to the hindquarters (hindlimbs and croup). Attention should be paid to the curvature and proportions of the top

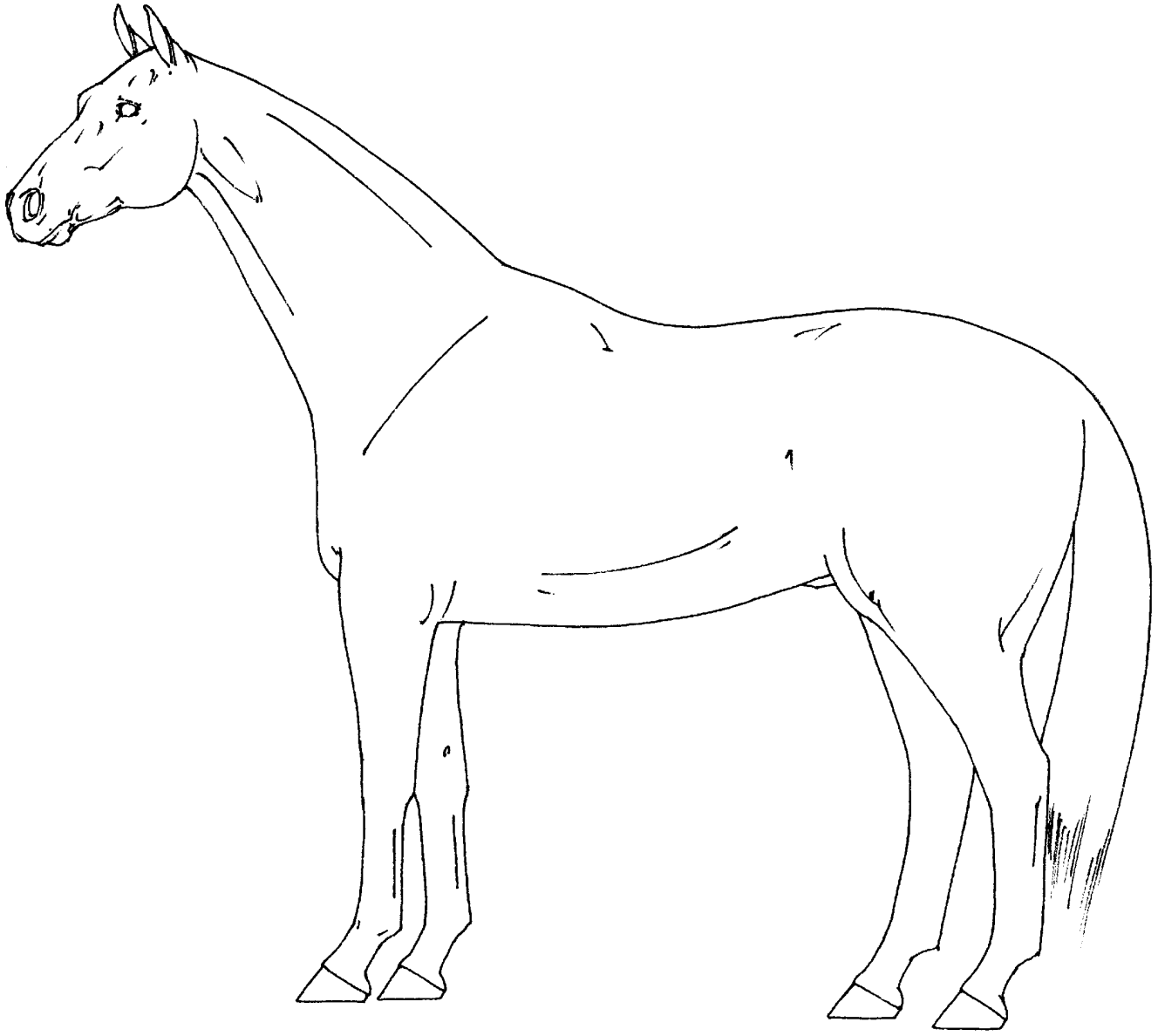


Figure 2.7. Normal horse. The body and limbs should be well proportioned.

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line, and observations made from poll to tail and down to the gaskin. The attachment of the appendicular skeleton (limbs) to the axial skeleton (head and trunk) should be observed with emphasis placed on limb angles. There is a strong relationship between long bone lengths and wither heights at differing ages, suggesting that horses should be proportional regardless of their size.[1]

From the front of the horse, the limbs and hooves are evaluated for straightness and symmetry. The depth and length of the muscles in the forearm and chest are observed. Evaluation of balance, top line, and limb angles is confirmed by viewing the off (right) side of the horse.

From directly behind the tail, the straightness and symmetry of the back, croup, point of the hip, and buttock are evaluated along with the hindquarters and the hindlimbs. This is also the best vantage point for evaluating back muscling, alignment of the vertebral column, and (provided the horse is standing square) left-to-right symmetry.

The observer should then make another entire circle around the horse, this time stopping at each quadrant to look diagonally across the center of the horse. From the rear of the horse, the observer should look from the left hindlimb toward the right forelimb and from the right hindlimb toward the left forelimb. This angle will often reveal abnormalities in the limbs and hooves that were missed during the side, front, and rear examinations. The horse is then viewed from the front in a similar diagonal approach. While observing the horse, it is helpful to obtain an overall sense of correctness of each of the four functional sections: head/neck, forelimbs, trunk (barrel), and hindlimbs.

Head and Neck

The size or shape of the head is often breed specific, but does not appear to influence performance or lameness. The neck acts as a lever to help regulate the horse's balance while moving; therefore, it should be long and flexible with a slight convex curve to its top line.[9,21] Jumping horses have been found to benefit from longer necks, probably because they make it easier to maintain balance over the fence.[21]

Forelimbs

Kinematic/kinetic studies have confirmed that at the beginning of the stance phase, the distal portion of the forelimb is subjected to more stress from weight-bearing forces than the distal portion of the hindlimb.[5,6] The forelimbs tend to "bounce," whereas the hindlimbs "slide."[21] The forelimbs are considered to support approximately 60%–65% of the horse's body weight, so they should be well muscled and conformed normally.[9,36]

Trunk (Barrel)

The horse should have adequate heart girth and width (spring) to the ribs to house the vital organs. The back should be well muscled and strong so that the horse is able to carry the weight of its internal organs and the rider and saddle.

Hindlimbs

The hindlimbs are the source of power for propulsion and stopping.[36] The hindlimb muscling should be appropriate for the type, breed, and use of the horse. The croup and points of the hip and buttock should be symmetrical, and the limbs should be straight and angled correctly.

CONFORMATION COMPONENTS AND TRAITS

Balance

Balance refers to the relationship between the forehand and hindquarters, between the limbs and the body, and between the right and the left sides of the body (Figures 2.7–2.9). It is a subjective assessment based on the overall conformation of the horse. A well-balanced horse is thought to move more efficiently, thereby experiencing less stress on the musculoskeletal system. However, one study that measured multiple conformational traits in racehorses found that 25% of all parameters showed a significant difference between the left- and right-sided measurements.[40]

The center of gravity is a theoretical point in the horse's body around which the mass of the horse is equally distributed. It is located at a point of intersection of a vertical line dropped from the highest point of the withers and a line from the point of the shoulder to the point of the buttock. The center of gravity is usually just behind the xyphoid and two-thirds the distance down from the top line of the back[36] (Figure 2.9).

Although the center of gravity remains relatively constant when a well-balanced horse moves, most horses must learn to rebalance their weight (and that of the rider and tack) when ridden. To pick up a front foot to step forward, the horse must shift its weight toward the rear. The amount of this weight shift depends on the horse's conformation, the position of the rider, the gait, the degree of collection, and the style of the performance. The higher the degree of collection, the more the horse must step under the center of gravity with the hindlimbs.

If the forehand is proportionately larger than the hindquarters, particularly if it is associated with a downhill top line, the center of gravity tends to shift forward. This causes the horse to travel heavy on his front end, setting the stage for increased concussion, stress, and lameness. When the forehand and hindquarters are balanced and the withers are level with or higher than the level of the croup, the horse's center of gravity is located more toward the rear. Such a horse can carry more weight with his hindquarters, thus moving in balance and exhibiting a lighter, freer motion with the forehand than the horse with withers lower than the croup. However, hip height was consistently 2–3 cm greater than wither height in young growing Thoroughbreds, suggesting that level top lines may only develop with maturity, if at all.[38] Even with a level top line, if the forehand is heavily muscled in comparison to the hindquarters, the horse may travel heavy on the forehand.

A balanced horse has approximately equal lower limb length and depth of body. The lower limb length (chest floor to the ground) should be equal to the distance from the chest floor to the top of the withers

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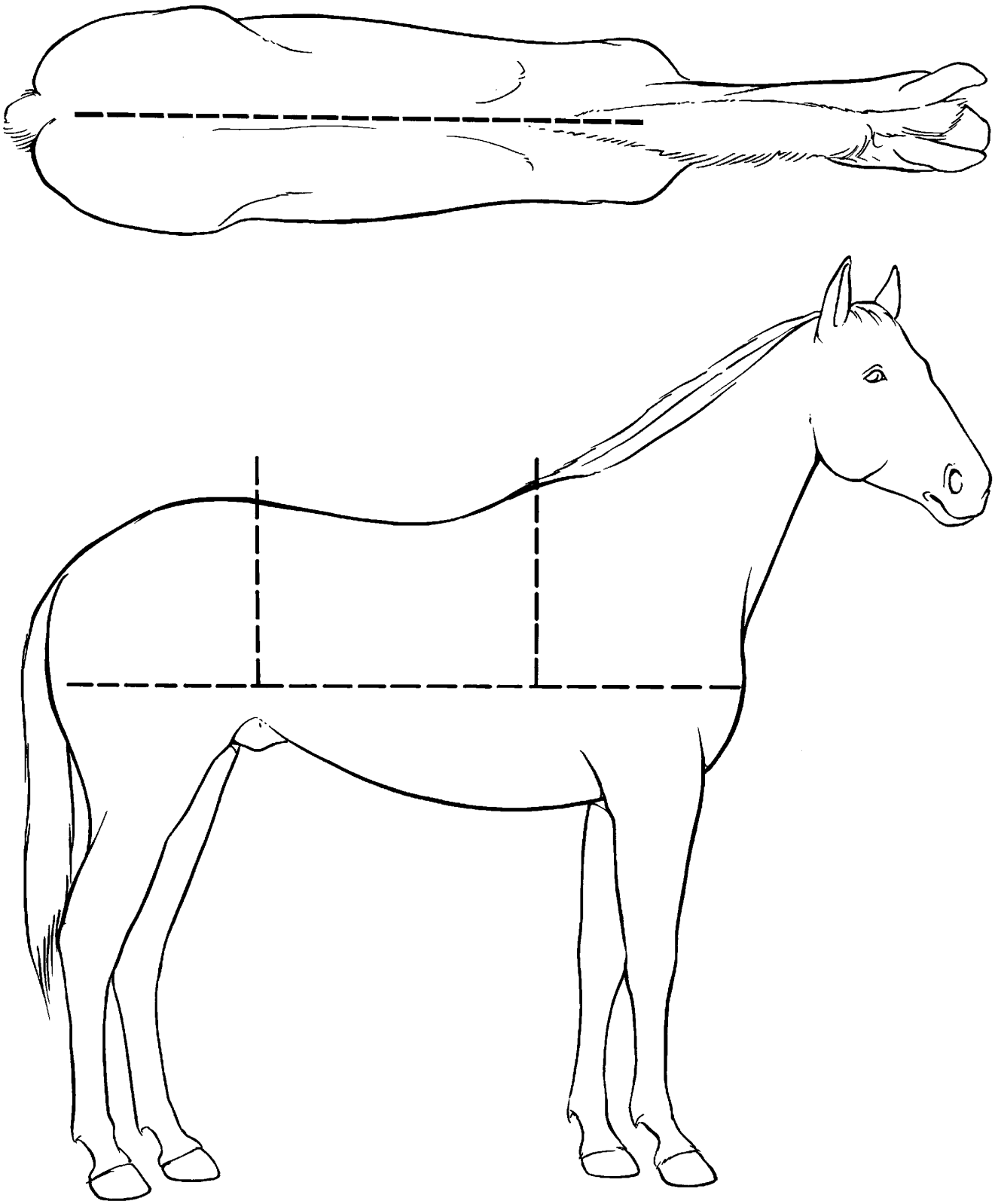


Figure 2.8. Axial alignment. A line drawn from the center of the withers through the center of the back should roughly divide the body into two equal halves. Bottom: Balance is evaluated by dividing the body into three equal parts.

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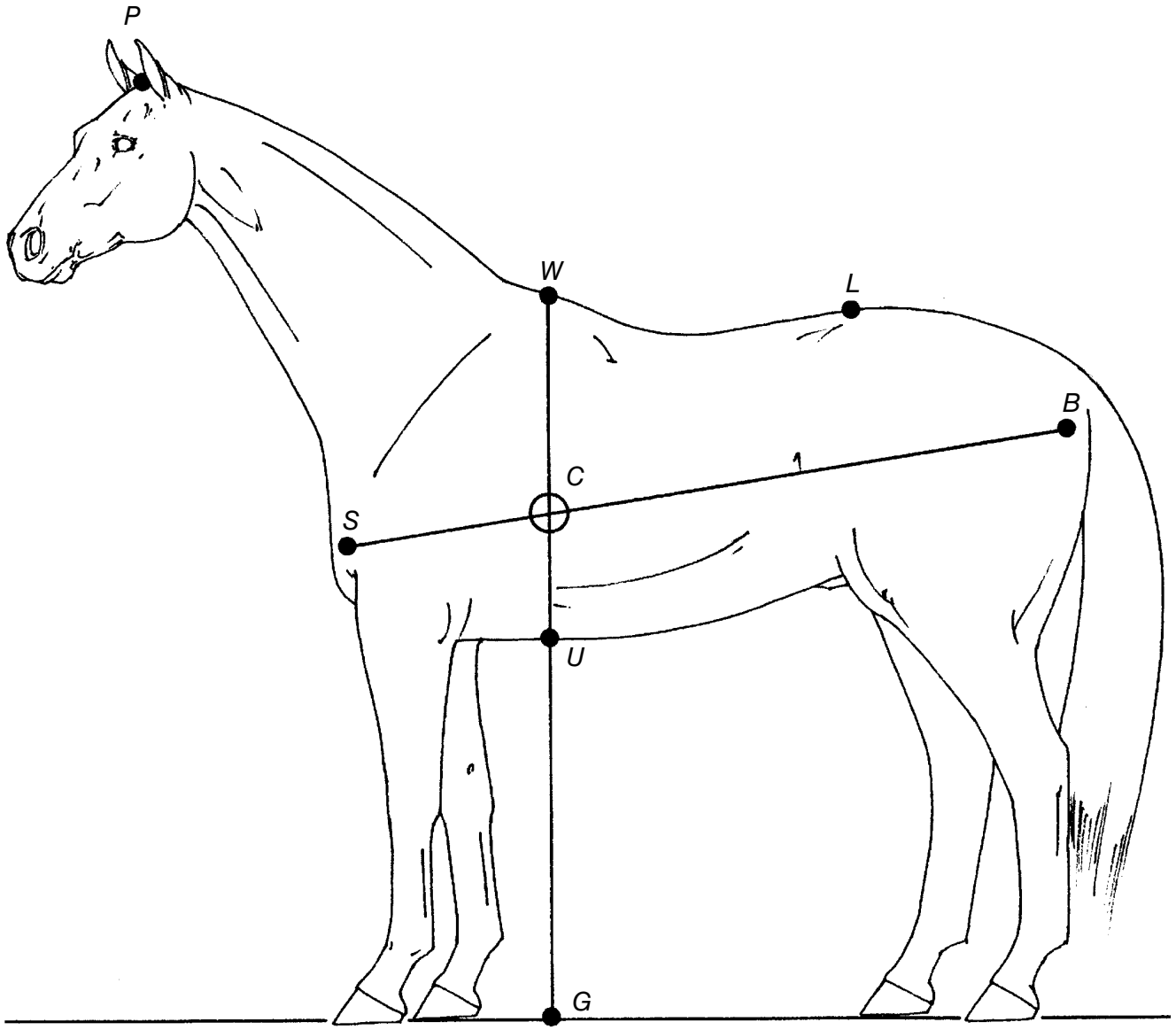


Figure 2.9. Proportions. P = poll, W = highest point of withers, L = caudal loin, B = point of buttock, S = point of shoulder, C = center of gravity, U = underline, G = ground, WU = depth of body, UG = lower limb length, WG = height and overall limb length, SB = length of body, PW = length of neck, WL = length of back, LB = length of hip. *Source*: Reprinted with permission from Hill and Klimesh.[20]

(Figure 2.9). Proportionately shorter lower limbs are associated with a choppy stride. The horse's height or overall limb length (point of the withers to the ground) should approximate the length of the horse's body (the point of the shoulder to the point of the buttock) (Figure 2.9). A horse with a longer body than its height often experiences difficulty in synchronization and coordination of movement. A horse with limbs proportionately longer than the body may be predisposed to forging, overreaching, and other gait defects. Overall, the right side of the horse should be symmetrical with the left side.

Proportions and Curvature of the Top Line

The ratio of the top line's components, curvature of the top line, strength of loin (longissimus dorsi muscles in the lumbar region), sharpness of withers, slope to the croup, and length of the underline in relation to the length of back all affect a horse's movement (Figures 2.8 and 2.9).[36]

The neck is measured from the poll to the highest point of the withers (Figure 2.9). The back measurement is taken from the withers to the caudal extent of the loin located in front of the pelvis. The hip length is measured from the caudal loin to the point of the buttock (Figure 2.9).

A neck that is shorter than the back tends to decrease a horse's overall flexibility and balance. A back that is much longer than the neck tends to hollow (flex down or is more concave). A very short hip, in relation to the neck or back, is associated with lack of propulsion and often a downhill configuration. A general rule of thumb is that the neck length should be greater than or equal to

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the back length and that the hip should be at least two-thirds the length of the back (Figure 2.9).

The shape of the neck is determined by the "S" shape formed by the seven cervical vertebrae. The neck should have a graceful shape that rises up out of the withers, not dipping ventral (downward) in front of the withers. The thickest point in the neck should be at the ventral limits. Ewe-necked horses often have a long, ventral curve to the neck that appears to attach low on the chest. The attachment of the neck muscles to the shoulders should be smooth. The dorsal (upper) neck length (poll to withers) should be twice the ventral (lower) neck length (throat latch to chest). This is dictated to a large degree by the slope of the shoulder. A horse with a steep shoulder has an undesirable ratio (approaching 1:1) between the dorsal and ventral neck lengths.[36]

The withers should blend gradually into the back, ending ideally at about the midpoint of the back. In one study all circumference measurements and the majority of the length measurements in a group of National Hunt racehorses were significantly correlated with the height of the horse at the withers.[40] The back behind the withers should resemble a natural place for a saddle, beginning with prominent withers located above or behind the heart girth. The neck and forearm muscles and the ligamentum nuchae should attach at the highest point of the withers. There should not be a prominent dip in the muscles in front of or behind the withers (Figure 2.10). Low (mutton) withers limit a horse's ability to raise its back as it lowers and extends its neck. A horse with a well-sloped shoulder usually has correctly placed withers.

The longissimus muscles that run along the spine should be flat and appear strong rather than sloped and weak. The back muscles aid to counteract the gravitational pull from the weight of the horse's internal organs and to support the rider's weight.

The loin is located along the lumbar vertebrae from the last thoracic vertebrae to the lumbosacral junction (Figure 2.9). The loin should be well muscled and relatively short. Horses termed "long-backed" often have an acceptable back length but a long, weak loin. A horse with a weak and/or long loin and loose coupling (flanks) tends to have a hollow back. (The coupling is the area behind the ribs and in front of a vertical line dropped from the point of the hip). A horse that has a chronic hollow back may be predisposed to focal lumbar pain, pinched nerves, or vertebral damage.[36] In addition, kinematic studies have documented that even subtle hindlimb lameness can alter movement of the thoracolumbar region, potentially contributing to secondary musculoskeletal pain in the region.[19]

The loin and coupling transfer the motion of the hindquarters up through the back and forward to the forehand; therefore, they should be strong and well connected. A short, heavily muscled loin may have great strength, power, and durability but may lack the flexibility of a more moderately muscled loin. A lumpy appearance in the loin may indicate abnormal alignment (subluxations) of the vertebrae or damage to the supraspinous ligament.[36]

The croup is measured from the lumbosacral junction to the tail head. The croup should be fairly long, because this is associated with a good length to the hip and a desirable forward placement of the lumbosacral articulation. The top line (the back) should be short in relation to the underline. Such a combination indicates strength plus desirable length of stride.

Substance

Thickness, depth, and breadth of bone, muscle, and other tissues are described as "substance." Muscle



Figure 2.10. Marked ventral curvature or lordosis of the back is considered to be undesirable because it may predispose to back problems.

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substance is described by the type, thickness, and length of the muscle and the position of attachment. Other substance factors include weight of the horse, height of the horse, size of the hoofs, depth of the heart girth and flank, and spring of rib.

"Spring of rib," which is best viewed from the rear, refers to the curvature of the ribs. In addition to providing room for the heart, lungs, and digestive tract, a well-sprung rib cage provides a natural, comfortable place for a rider's legs. A slab-sided horse with a shallow heart girth is difficult to sit upon properly; an extremely wide-barreled horse can be stressful to the rider's legs.

"Substance of bone" refers to adequacy of the bone to the horse's weight ratio. Traditionally, the circumference around the cannon bone just below the carpus serves as the measurement for substance of bone. For riding horses, an adequate ratio is approximately 0.7 inches of bone for every 100 lbs of body weight.[36] Using this rule, a 1,200-lb. horse should have an 8.4-inch cannon bone circumference.

Correctness of Angles and Structures

The correct alignment of the skeletal components provides the framework for muscular attachments. The length and slope to the shoulder, arm, forearm, croup, hip, stifle, and pasterns should be appropriate and work well together. There should be a straight alignment of bones when viewed from the front and rear, large clean joints, high-quality hoof horn, adequate height and width of heel, concave sole, and adequate hoof size.

Forelimbs

Cranial View

Both forelimbs should be of equal length and size and should appear to bear equal weight when the horse is standing squarely. A line dropped from the point of the shoulder (middle of the scapulohumeral joint) to the ground should bisect the limb. The manner in which the shoulder blade and arm (humerus) are conformed and attach to the chest often dictates the alignment of the lower limb. Whether the toes point in or out is often related to upper limb structures, which is why it may be counterproductive to alter a limb's alignment through radical hoof adjustments. The toes should point forward, and the width of the feet on the ground should be the same as the width at the origin of the limbs in the chest (Figure 2.11).

The medial/lateral (ML) slope of the humerus is evaluated by finding the point of the shoulder and a spot in front of the point of the elbow on each side. The four points are then connected visually. If the resulting box is square, the humerus lies in an ideal position for straight lower limbs. If the bottom of the box is wider, the horse may toe in and travel with loose elbows and paddle. If the bottom of the box is narrower, the horse will probably toe out, have tight elbows, and wing in.

The muscles of the forearm (antebrachium) should extend to the knee, tapering gradually rather than ending abruptly a few inches above the knee. This is believed to allow the horse to use its front limbs in a smooth, sweeping, forward motion. The pectoral muscles should also extend far down onto the limb. The pectoral and

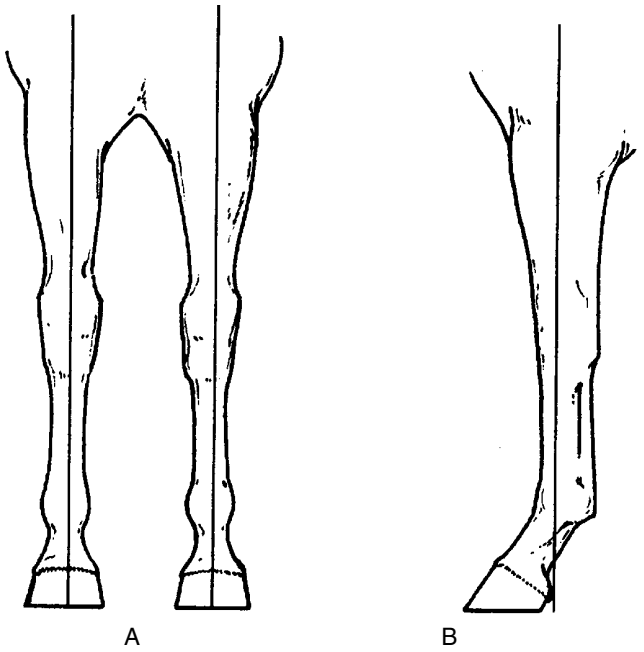


Figure 2.11. Cranial and lateral views of normal forelimbs. (A) A line dropped from the point of the shoulder joint should bisect the limb. (B) A line dropped from the tuber spinae of the scapula should bisect the limb down to the fetlock and end at the heel bulbs.

the forearm muscles help a horse to move its limbs laterally and medially and to elevate the forelimb. It is believed by some horseman that long forearms and short cannon bones are conducive to speed,[26] but this has not been documented.[29]

The carpal joints should be balanced and should not deviate toward, or away from, one another. The cannon bone should be centered under the carpus and not to the lateral side (bench knees). Deviations from a straight limb may cause strain on the collateral support structures and asymmetrical loading (compression) of the hinge joints in the forelimb.

Lateral View

When viewed from the side, limbs should exhibit a composite of moderate angles so that shock absorption is efficient (Figure 2.12). The shoulder angle is measured along the spine of the scapula, from the point of the shoulder to the point of the withers. The angle of the scapula and shoulder tend to increase (become more upright) as horses mature from foals to adults.[1] The shorter and straighter the shoulder, the shorter and quicker the stride, and the more stress and concussion that is transmitted to the limb.[21] Studies in dressage horses and show jumpers have found that a more horizontal scapula was related to a higher level of performance.[22,27] Also important is the angle the shoulder makes with the arm, which should be at least 90°. Horses with a more horizontally positioned scapula or a more flexed shoulder joint show more maximal extension of the elbow joint relative to the angle at initial ground contact.[5] This prolongs the stance phase. A long sloping scapula is ergonomically efficient and is thought to place the rider more to the rear of the horse, resulting

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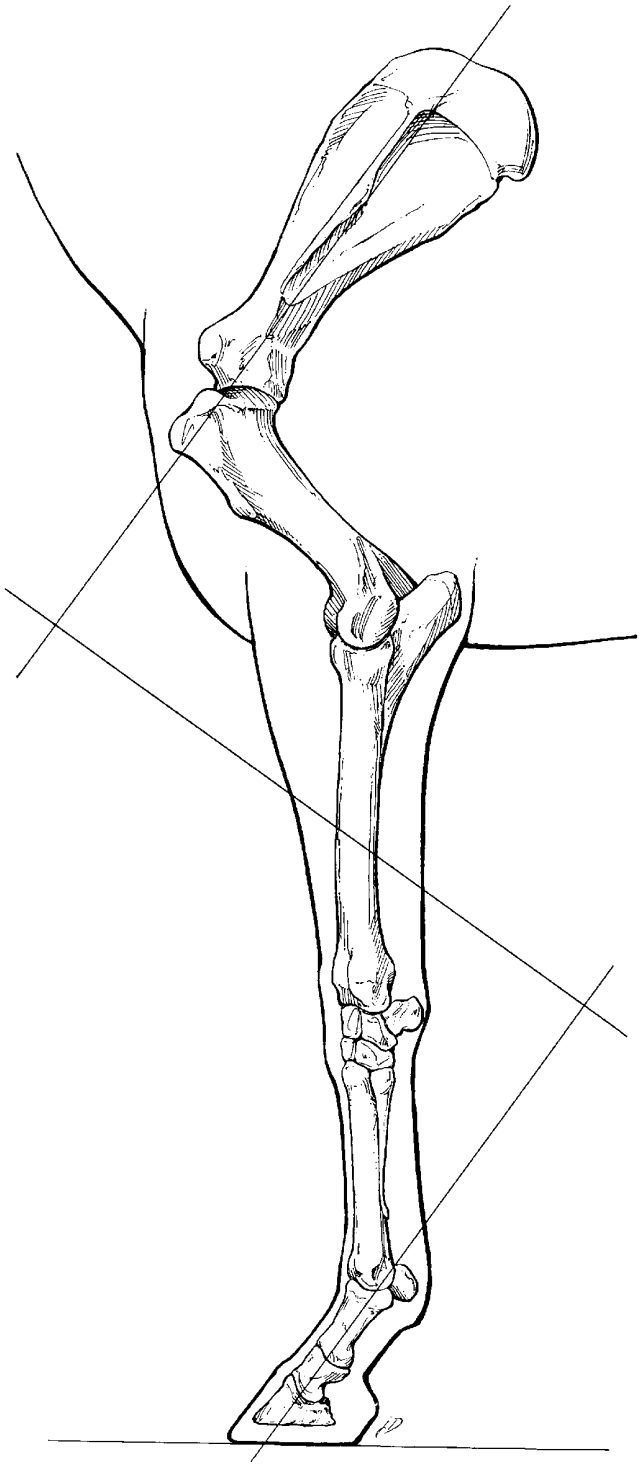


Figure 2.12. The angle of the shoulder usually influences the angle of the pastern.

in improved balance.[21] The scapulohumeral (shoulder) joint is supported entirely by the muscles and tendons surrounding it. Because this muscle support is so important to proper movement, a horse should have well-developed muscles in this region.

The length of the humerus (from the point of the shoulder to the point of the elbow) can affect stride length (Figure 2.12). A long humerus is associated with a long, reaching stride and good lateral ability; a short humerus is related to a short, choppy stride and poor lateral ability. A long humerus corresponds to a long triceps muscle that facilitates a larger range of elbow movements.[21] In general, the steeper the angle of the humerus, the higher the action; the closer the angle is to horizontal, the lower the action.

The angle formed by the humerus and radius and ulna at the elbow joint should be between 120° and 150°.[36] A more flexed elbow together with a horizontal scapula results in a longer stance duration, which is thought to improve gait quality with more collection in the forelimbs.[21,23] Straighter conformation (lesser angulation) at this joint may result in a short, choppy gait and increased concussion on the distal limb.

Ideally the limb should form a straight column from the elbow joint to the fetlock. This conformation will disseminate the axial compression forces to all bony surfaces equally. With malalignment, axial compressive forces become focused on one side, and tensional forces are created opposite to it, increasing the stress and strain on musculoskeletal structures.[36]

The carpus (knee) is a complex joint and functions in flexion, absorption of concussion, and extension. Flexion primarily occurs at the radiocarpal and middle carpal joints, and extension occurs by a locking mechanism while the horse is weight bearing during the extension phase of the stride. The carpal bones should be in good axial alignment with the radius and third metacarpal bone and of sufficient size to support downward concussive forces. The carpus should appear straight and not deviate forward or backward (Figure 2.13).

Fetlock joints should be large enough and angled to permit free movement. A more extended fetlock joint resulted in more maximal extension, which correlated with a good gait in the forelimbs in one study.[6] It has also been found that a straighter hindlimb fetlock joint angle results in a longer stride and swing duration.[6] A study on Swedish Warmblood horses found the mean front fetlock angle to between 146° and 155° and the mean hind fetlock angle between 153° and 161°.[22]

This is considerable variation in the literature regarding what the "normal" hoof-pastern axis of the forefoot should be. Previous reports of angles less than 50° are currently considered to be too low, and normal angles are thought to be approximately 54°.[4,29,36] Although these angles may vary considerably, the angle of the dorsal hoof wall should be similar to the angle of the pastern.[9] Exceptionally long, sloping pasterns or a broken-back hoof-pastern axis are thought to increase strain to flexor support structures of the fetlock and phalanges (Figure 2.14). Short, upright pasterns or a broken-forward hoof-pastern axis are thought to cause greater concussive stresses to the fetlock, phalangeal joints, and foot.[4,9,29]

The hoof should be appropriately sized for the size for the horse, well shaped (more round than egg shaped), and symmetric. It should have high-quality hoof horn, adequate height and width of heel, a concave sole, and a robust frog. The bulbs of the heel should lie vertically below the central axis of the cannon bone in the sagittal plane (Figure 2.11B). Normally trimmed hooves usually impact the ground heel first or flat-footed.[4,36] Studies have documented that lower hoof angles predispose

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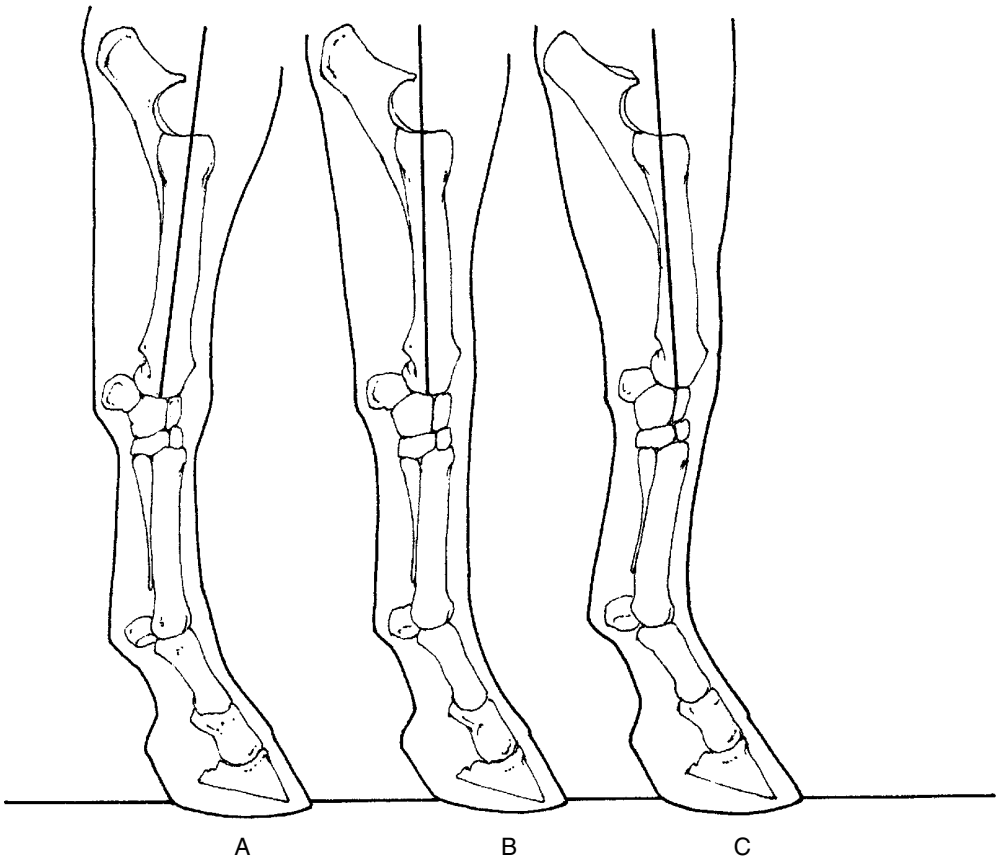


Figure 2.13. (A) Calf-kneed (backward deviation of the knee). (B) Normal. (C) Buck-kneed (forward deviation of the knee).

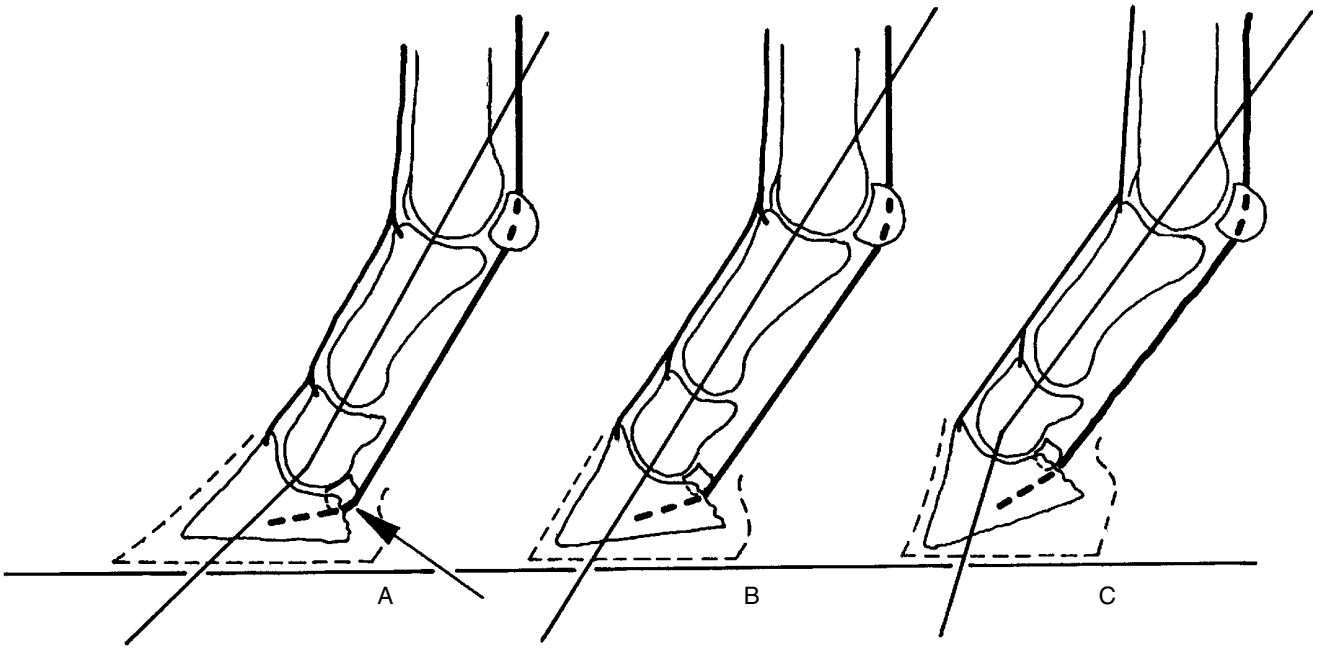


Figure 2.14. (A) Broken-back pastern axis contributes to increased pressure in the navicular region (arrow). (B) Normal or straight pastern axis. It is considered normal for the coffin bone to be 3°–5° above the horizontal. (C) Broken-forward pastern axis. *Source*: Reprinted with permission from Hill and Klimesh.[20]

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racehorses to musculoskeletal injuries and contribute to a multitude of lameness problems in the palmar aspect of the foot.[26,29,31]

Faults in Conformation of the Forelimbs

Base-Narrow

In base-narrow conformation, the distance between the center of the feet on the ground is less than the distance between the center of the limbs at their origin in the chest when viewed from the front (Figure 2.15). This is often seen in horses with large chests and well-developed pectoral muscles, such as the Quarter horse. This conformation may be accompanied by a toe-in (pigeon-toed) or toe-out (splay-footed) conformation. Base-narrow conformation inherently causes the horse to bear more weight on the outside of the foot than on the inside.

Base-Wide

In base-wide conformation, the distance between the center of the feet on the ground is greater than the distance between the center of the limbs at their origin in the chest when viewed from the front (Figure 2.16). This condition is found most commonly in narrow-chested horses and may be accompanied by toe-out (splay-footed) position of the feet. Base-wide, toe-out conformation usually causes winging of the limb to the inside (Figure 2.17). Base-wide conformation may force the horse to land on the inside of the foot, increasing weight-bearing forces on the inside of the foot and entire limb.

Toe-In or Pigeon-Toed

Toe-in is the position of the feet in which the toes point toward one another when viewed from the front (Figures 2.18 and 2.19). Its point of origin can be as high as the chest or as low as the fetlock down. It is often accompanied by a base-narrow conformation but rarely is present when the horse is base-wide. In the

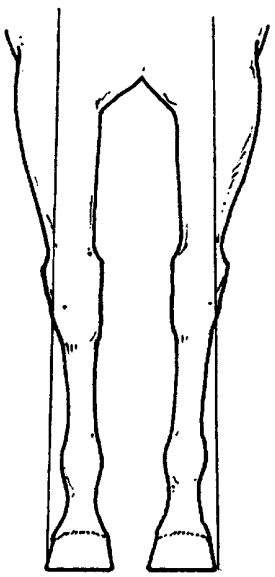


Figure 2.15. Base-narrow conformation. The distance between the center lines of the limbs is greater than the distance between the center lines of the feet on the ground.

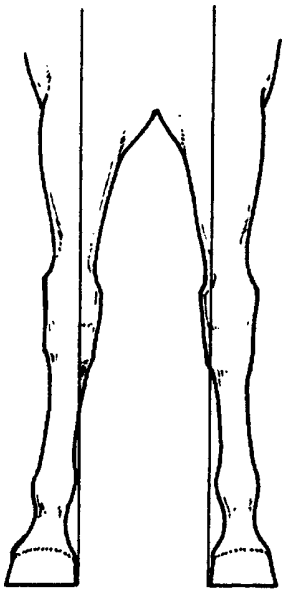


Figure 2.16. Base-wide conformation. The distance between the center lines of the feet is greater than the distance between the center lines of the limbs at the chest.

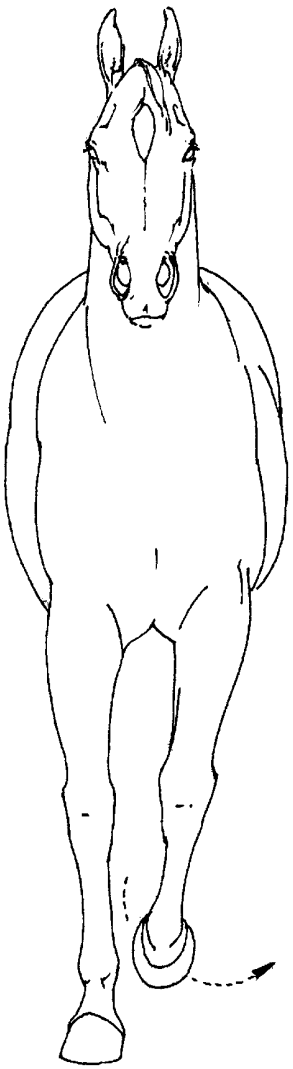


Figure 2.17. Winging, which may cause interference, is caused by a toe-out position of the foot.

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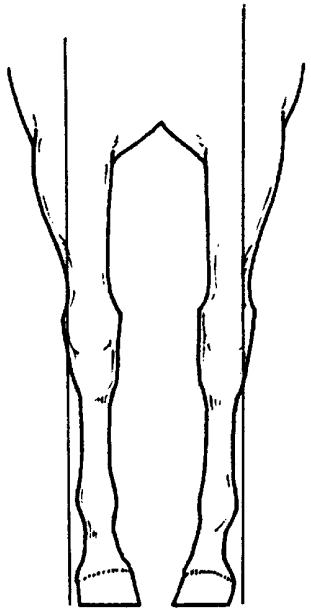


Figure 2.18. Base-narrow, toe-in conformation.



Figure 2.19. Horse with a slight varus deviation of the distal limb. Slight toe-in or pigeon-toed conformation is relatively common and does not appear to contribute to lameness in many cases.

young foal, the condition may be seen as the result of an angular limb deformity. Proper trimming and corrective shoeing of the feet may prevent worsening of the condition, especially in growing horses. One study documenting variations in conformation in Swedish Warmblood horses found a 50% frequency of mild toe-in conformation in elite sport horses, suggesting that minor deviations do not impair soundness or performance.[22]

Toe-Out or Splay-Footed

In a toe-out or splay-footed conformation, the toes point away from one another when viewed from the front (Figure 2.20). The point of origin is usually at the

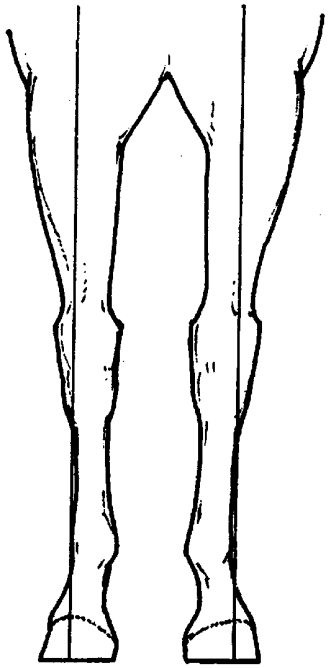


Figure 2.20. Base-narrow, toe-out conformation.

chest, but the condition can be associated with outward rotation of the fetlock. It may be accompanied by either base-wide or base-narrow conformation. As with a toe-in conformation, it may be controlled or partially corrected by corrective trimming or shoeing. The flight of the foot goes through an inner arc when advancing, contributing to interference with the opposite forelimb (Figure 2.17).

Base-Narrow, Toe-In Conformation

Base-narrow, toe-in conformation causes excessive strain on the lateral collateral support structures of carpus, fetlock, and phalangeal joints (Figure 2.18). It often causes paddling and appears to be a common conformational abnormality.

Base-Narrow, Toe-Out Conformation

Base-narrow, toe-out conformation is one of the worst types of conformation in the forelimb (Figure 2.20). The closely placed feet, combined with a tendency to wing inwardly from the toe-out position, commonly cause limb interference or plaiting. The hoof breaks over the inside toe, swings inward, and lands on the outside wall, contributing to increased strain on the limb below the fetlock. The base-narrow limb position places the weight on the outside wall, as with base-narrow, toe-in conformation.

Base-Wide, Toe-Out Conformation

When a horse is base-wide, the feet usually toe out. The base-wide conformation places the greatest stress on the inside of the limb (Figure 2.21). The foot usually breaks over the inside toe, wings to the inside, and lands on the inside hoof wall. Interference is common, similar to any type of toe-out conformation. Some horses, especially those with base-narrow, toe-out conformation, tend to place one foot directly in front of the other.

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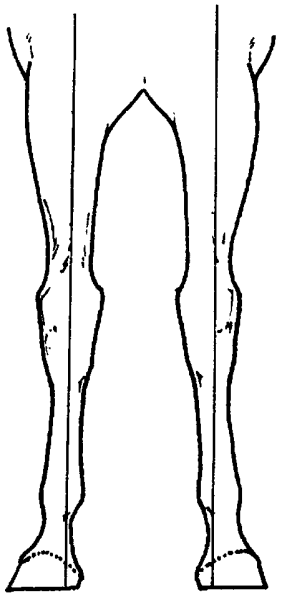


Figure 2.21. Base-wide, toe-out conformation.

This is an undesirable characteristic referred to as "plaiting," because it can produce interference and stumbling when the advancing limb contacts the one placed in front of it. Plaiting tends to be seen more commonly in the hindlimbs than the forelimbs.

Palmar (Backward) Deviation of the Carpus (Hyperextended Knees, Calf Knees)

Backward deviation of the carpus (back at the knee) is thought to place increased stress on the palmar soft tissue structures and increase compression on the dorsal aspect of the carpus (Figure 2.13). Backward knee conformation is thought to make horses working at speed more susceptible to carpal injuries, but this is not widely accepted. Hyperextension of the carpus is not the only reason for carpal injuries; long toes were associated with carpal problems in Thoroughbred racehorses in one study.[31] Additionally, injury to the palmar soft tissue structures of the carpus may contribute to this conformation, suggesting that it may not be a strictly genetic influence.

Dorsal (Forward) Deviation of the Carpus (Bucked Knees or Knee Sprung)

This condition may also be called "goat knees" or "over in the knees" (Figure 2.13). It is generally believed that "bucked knees" is a less serious problem than the calf-knee condition and may protect against carpal disease in racehorses.[29] However, severe dorsal deviation may be more dangerous for the rider because the horse's knees are on the verge of buckling forward. Forward deviation of the carpus may be caused by contraction of the carpal flexors (i.e. ulnaris lateralis, flexor carpi ulnaris, and flexor carpi radialis) in young horses and may place increased strain on the extensor carpi radialis and the suspensory ligament.[36] Congenital forms are nearly always bilateral and may be accompanied by a forward knuckling of the fetlocks. The condition is often present at birth and usually disappears by 3 months of age if it is not severe. One study documenting the variations in conformation in Swedish Warmblood horses found that considerably more elite sport horses were "buck kneed" than "calf kneed," while the reverse was true in riding school horses.[22]

Lateral Deviation of the Carpus (Bowlegs, Carpal Varus, or Bandy-Legged Conformation)

Carpal varus is an outward deviation of the carpus (or inward deviation of the distal limb) when viewed from the front of the horse (Figure 2.22A). It may be accompanied by a base-narrow, toe-in conformation. This condition increases tension on the lateral surface of the carpus and compression on the medial surface of the carpus and carpal bones (Figure 2.18). This deviation may be correctable in young horses, but growth of the knee is thought to reach a plateau at approximately

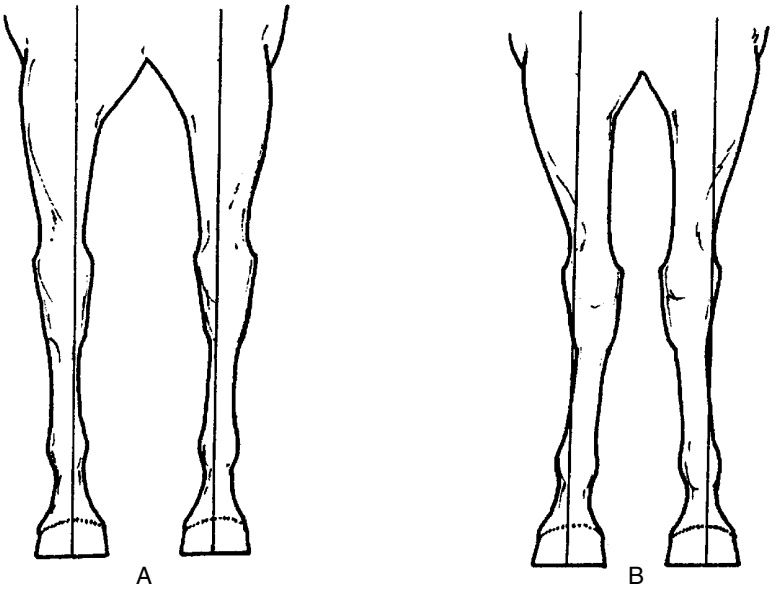


Figure 2.22. Examples of abnormal carpal conformation as compared to Figure 2.11. (A) Carpal varus or bowlegs. (B) Carpal valgus or knock knees. Some degree (<5°–7°) of carpal valgus is considered to be within normal limits in most horses.

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140 days of age in Thoroughbreds, suggesting that most carpal deviations (lateral or medial) should be corrected before this age.[38] However, carpal and fetlock conformation changed greatly in Thoroughbred foals up to 18 months of age in one study suggesting that the window of growth may be longer than previously thought.[34]

Medial Deviation of the Carpus (Knock Knees, Carpus Valgus)

Medial angular deviation of the carpus (or lateral deviation of the distal limb) can result from abnormalities of the distal metaphysis, physis, and epiphysis of the radius; from abnormal development and alignment of the carpal bones; or from carpal joint laxity (Figure 2.22B). This deviation contributes to increased tensional strain on the medial collateral ligaments of the carpus and compression forces on the lateral (concave) surface of the carpus. Varying degrees of stresses are also transmitted to the joints proximal and distal to the carpus. Varying degrees of outward rotation of the cannon bone, fetlock, and foot may accompany this entity. Mild carpal valgus (5°–7°) may have a protective mechanism against carpal injuries, as the odds for carpal fracture and effusion decreased with an increase in carpal angle in Thoroughbred racehorses.[2] However, in a study of National Hunt racehorses, carpal valgus conformation was associated with an increased risk for SDF tendon injury.[41] In the same study, valgus deformations of the carpus, fetlock, and tarsus were all associated with an increased risk of injury and considered to be detrimental to performance.[41]

Bench Knees (Offset Knees)

Bench knee is a conformation in which the cannon bone is offset to the lateral side and does not follow a straight line down from the radius when viewed from the front (Figure 2.23). Increased weight-bearing on the medial splint bone is thought to occur in bench-kneed horses. This is thought to contribute to increased stress on the interosseous ligament predisposing to "splints." In a study documenting the conformational abnormalities in 356 Warmblood horses, more than 50% were bench kneed.[22] The combination of bench knees and carpal valgus is common, and offset knees has been associated with fetlock problems in racing Thoroughbreds.[2]

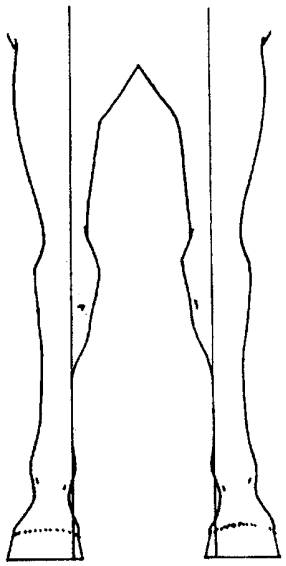


Figure 2.23. Offset knees (bench knees), which are characterized by the cannon bones being set too far laterally.

Short Upright Pastern

A short upright pastern is believed to increase concussion and injuries to the fetlock and phalangeal joints, the navicular bone region, and soft tissue structures within the metacarpus (Figure 2.24).[29,36] If the angle of the hoof is more upright than that of the pastern, it is referred to as a broken-forward hoof-pastern axis.[21] This type of conformation is often associated with a base-narrow, toe-in conformation and is often seen in horses with short limbs and a powerful body and limb musculature. Additionally, a straight shoulder (more vertical) usually accompanies a short upright pastern.

Long Sloping Pastern

A long sloping pastern is characterized by a normal or subnormal angulation of the forefoot (less than 50°–54°) with a pastern that is too long for the length of the limb (Figure 2.25). If the angle of the hoof is more acute (lower) than that of the pastern, it is referred to as a broken-backward hoof-pastern axis.[21] It is often seen in horses with long toes and/or low heels and may

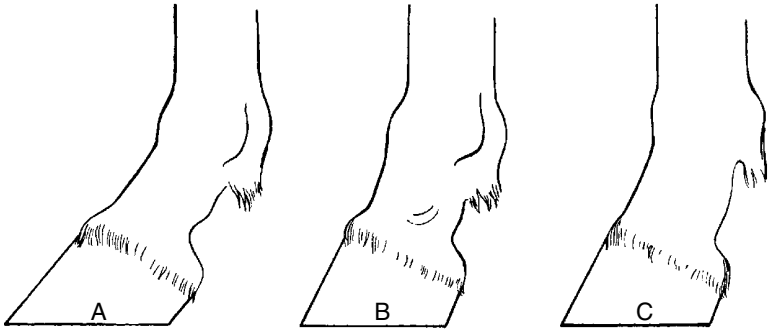


Figure 2.24. Examples of pastern conformation. (A) A normal angulation of hoof wall and pastern. (B) A short upright pastern. (C) A long upright pastern.

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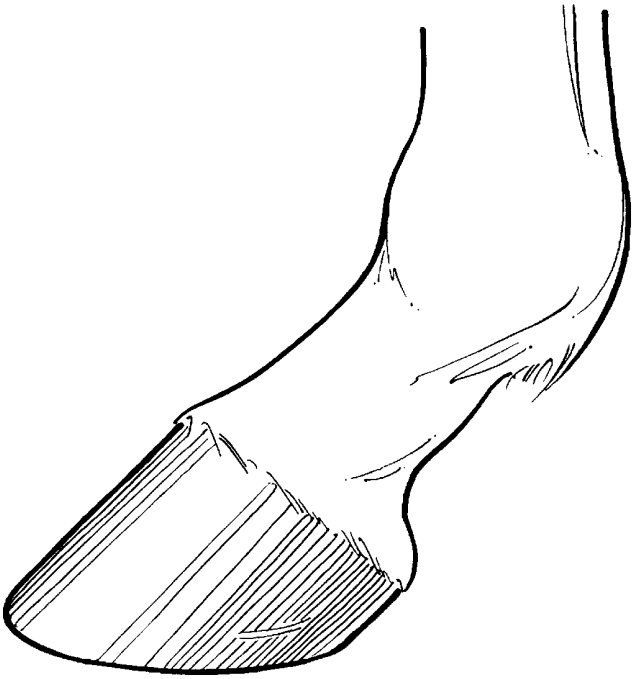


Figure 2.25. Example of a long sloping pastern that is often associated with a broken-back hoof-pastern axis. A long toe and a low heel often contribute to this conformation. The weight-bearing surface of the foot is well forward of the metacarpus/metatarsus.

predispose a horse to injury of the navicular region, flexor tendons, sesamoid bones, and suspensory ligament (Figure 2.25).[36] Long pasterns were also found to increase the odds of Thoroughbred racehorses fracturing a front limb,[2] and the risk of SDF tendon injury increased with an increase in fetlock joint angle in a study of National Hunt racehorses.[41]

The Hindlimbs

The hindlimbs constitute the "engine" of the horse regardless of its intended use. Endurance horses typically have longer, flatter muscles, stock horses have shorter, thicker muscles, and all-around horses have moderate muscling. Lack of hindlimb musculature or a disparity between the forelimb vs. hindlimb muscle mass may suggest chronic hindlimb lameness problems.

Lateral View

When viewed from the side, the hindlimbs should exhibit a composite of moderate angles so that shock absorption will be efficient (Figure 2.26). A line from the point of buttock to the ground should touch the hock and end slightly behind the bulbs of the heels. A hindlimb in front of this line is often standing under (Figure 2.27) or sickle hocked (Figure 2.28); a hindlimb behind this line is often post-legged (Figure 2.29) or camped out (Figure 2.30). The hindquarter should be symmetric and well connected to the barrel and the lower limb. The gluteal muscles should tie well forward into the back, and the hamstrings should extend down low into the Achilles tendon of the hock.



Figure 2.26. Normal hindlimb conformation as viewed from the side. A line dropped from the point of the buttock (tuber ischii) should follow the cannon bone.

The relationship of the length of the bones, the angles of the joints, and the overall height of the hindlimb often dictate the type of action and amount of power produced. The length and slope to the pelvis (croup) are measured from the point of the hip to the point of the buttock. A flat, level croup is associated with hindlimb action that occurs behind the hindquarters rather than underneath it. A "goose rump" is a very steep croup that places the hindlimbs so far under the horse's belly that structural problems may occur due to the overangulation. Somewhere between these two extremes is ideal. In general, a more flexed hip causes a more protracted position of the hindlimb at the stance phase and a more horizontal femur during the swing phase. The coxal angle (angle between the ilium and ischium) was recently shown to have a positive effect on performance in National Hunt racehorses, and the risk of pelvic fractures decreased with an increasing coxal angle.[3,41]

Probably the most important individual conformational detail in sport horses is the femur. A long and forwardly sloping femur places the hindlimbs more under the horse, which allows the horse to keep its balance more easily because the limb is closer to the center of gravity.[21,22] A forwardly sloping femur has also been shown to have a positive effect on soundness. A long femur is thought to result in a longer stride with more reach. A short femur is associated with the short, rapid

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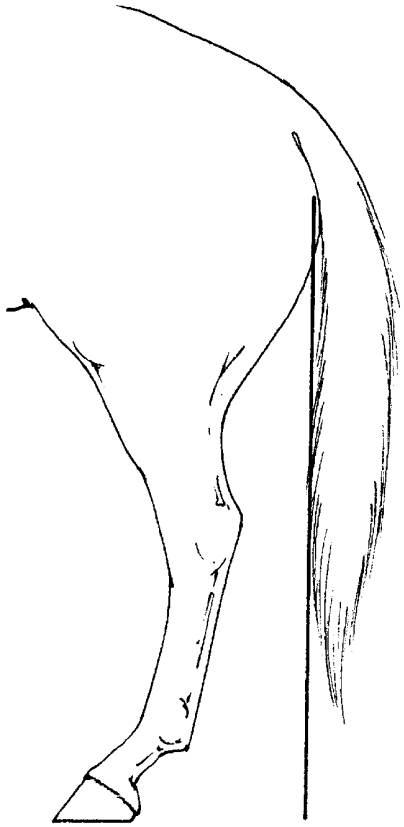


Figure 2.27. Standing under behind (compare with Figure 2.26).

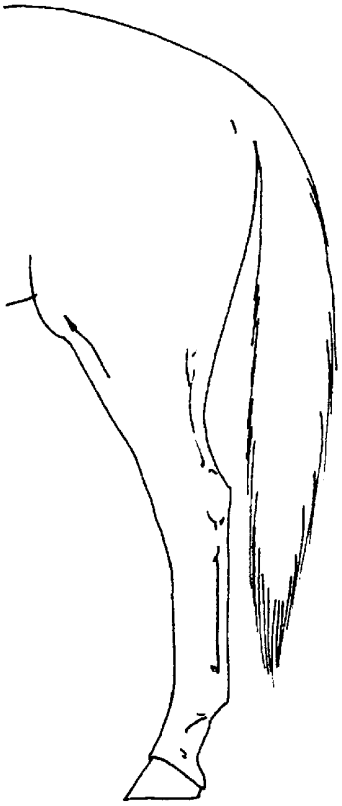


Figure 2.29. Post-legged refers to too little angulation of the hock and stifle joints. The hock is very straight and angles of more than 165°–170° are considered to be abnormal.

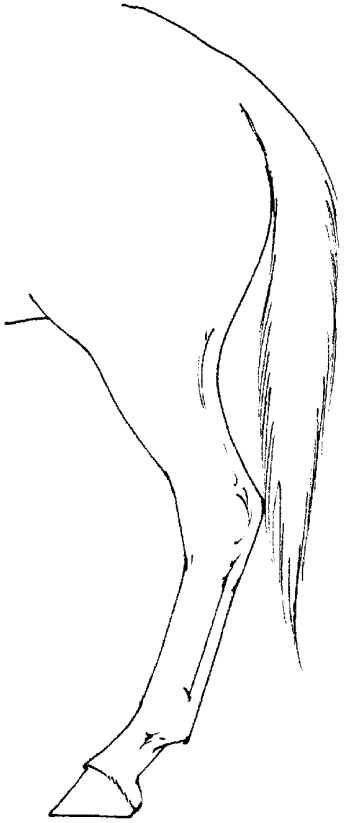


Figure 2.28. Sickle hocks. The angle of the hock looks excessive but is actually less than normal. Hocks with an angle of less than 150°–153° are considered to be sickle.

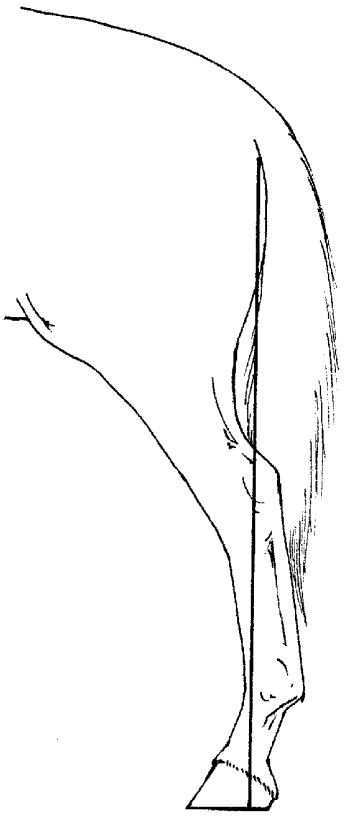


Figure 2.30. Camped out behind (compare with Figure 2.26).

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stride characteristic of a sprinter. The gaskin length (stifle to hock) should be shorter than the femur length (buttock to stifle). A gaskin length longer than the femur length tends to be associated with cow hocks and sickle hocks.

Hindlimbs with less angulation (open angles, straighter hindlimbs when viewed from the side) usually have a shorter overall limb length and produce efficient movement suitable for hunters or racehorses. Generally, hindlimbs with more angulation (closed joints) have a longer overall limb length and produce a more vertical, folding action necessary for collection characteristic of dressage horses.[36] An overall limb length that is too long may contribute to either a camped-out or sickle-hocked conformation. Elite Warmblood horses were found to have larger hock angles (straighter) than other groups of horses.[22] Horses with lameness and back problems usually have significantly smaller hock angles than sound horses.[21]

Caudal View

From the rear, both hindlimbs should be symmetrical, have the same length, and bear equal weight (Figure 2.31). A left-to-right symmetry should be evident between the peaks of the croup (tuber sacrale), the points of the hip, the points of the buttock, and the midline position of the tail. A study in Standardbred trotters found that hindquarter asymmetry (tuber sacrale at unequal heights) and associated factors had a negative effect on performance (Figure 2.32).[14] In addition, poor development of the epaxial muscles in the thoracolumbar region and asymmetry of the hindquarter musculature but not asymmetry of the tuber sacrale were common findings in sport horses with documented sacroiliac disease.[16] The widest point of the hindquarters should be the width between the stifles. A line dropped from the point of the buttock to the ground should essentially bisect the limb; however, the

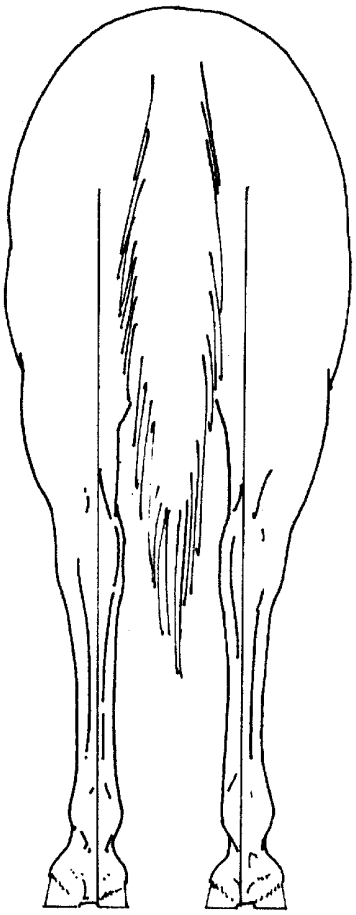


Figure 2.31. Normal hindlimbs as viewed from the rear. A line dropped from the point of the buttock (tuber ischii) should bisect the limb.

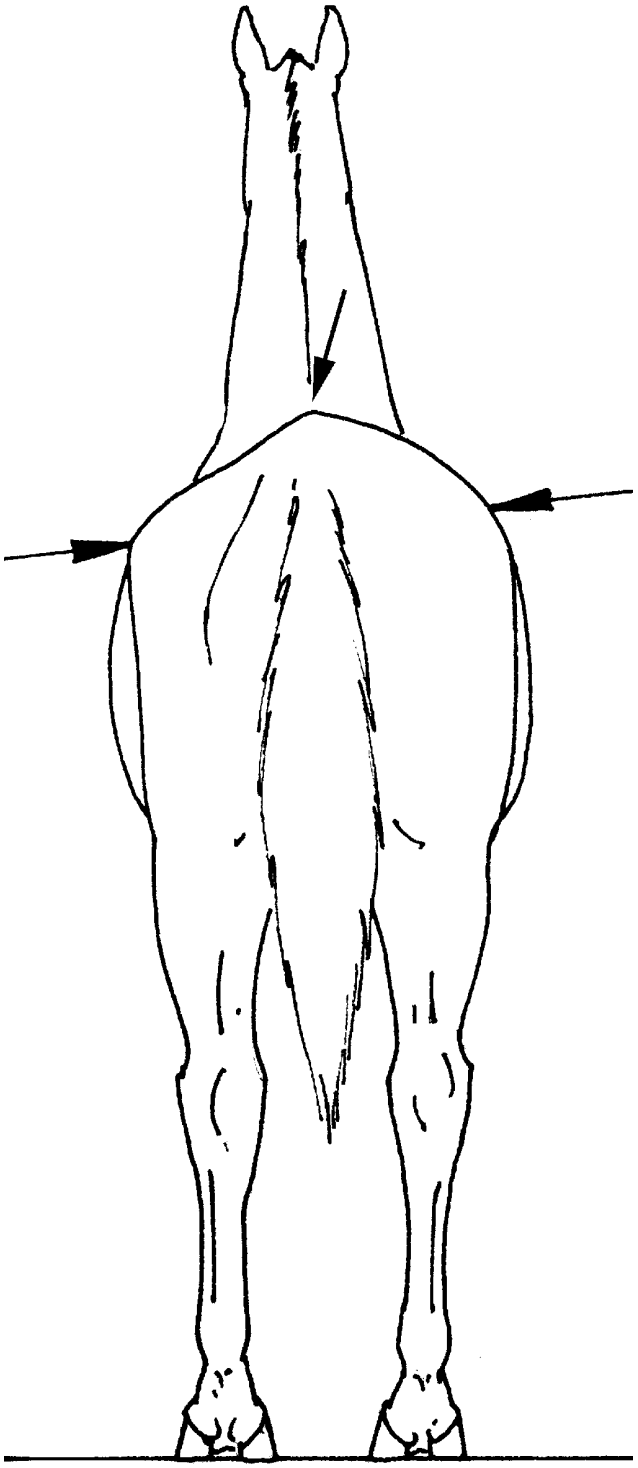


Figure 2.32. Asymmetry between the peaks of the croup, points of the hip, and points of the buttock (arrows).

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hindlimbs are not designed to point absolutely straight forward.[29] About 80% of all Warmblood riding horses and Standardbred trotters have outwardly rotated hindlimbs.[21] It is also normal for the stifles to point slightly outward, which in turn causes the points of the hocks to face slightly inward and the toes to point slightly outward. When the cannon bone faces outward, the horse is considered to be cow-hocked (Figure 2.33); when the cannon bones face inward, the horse is bowlegged (Figure 2.34).

Faults in Conformation of the Hindlimbs

Excessive Angulation of the Hock (Sickle Hocks, Small Hock Angles)

Mean hock angles in normal horses are thought to be approximately 155°–165°.[18,21] Hock angles less than 150°–153° are considered sickle (Figure 2.28).[21,29] When viewed from the side, the angle of the hock is decreased so that the horse is standing under from the hock down. This places the hock under greater stress and may predispose to synovial distension in the stifle and hock joints and bone spavin.[21,29] A small tarsal angle was significantly associated with radiographic signs of OA in the distal tarsus in a large number of Icelandic horses.[3] Small hock angles may also impair a horse's ability to attain the level of collection required for good performance in advanced classes. One study on elite Swedish Warmblood sport horses found that none of the show jumpers and only one of the dressage horses were "sickle hocked."[22]

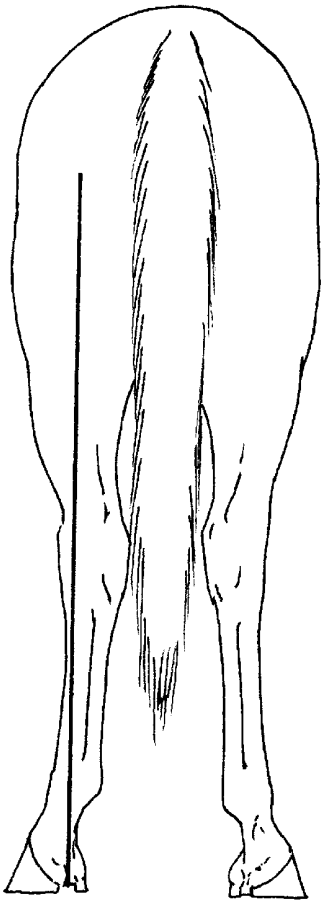


Figure 2.33. Cow hocks accompanied by base-wide conformation (compare with Figure 2.26). Such horses are often base-narrow at the hocks and base-wide from the hocks down.

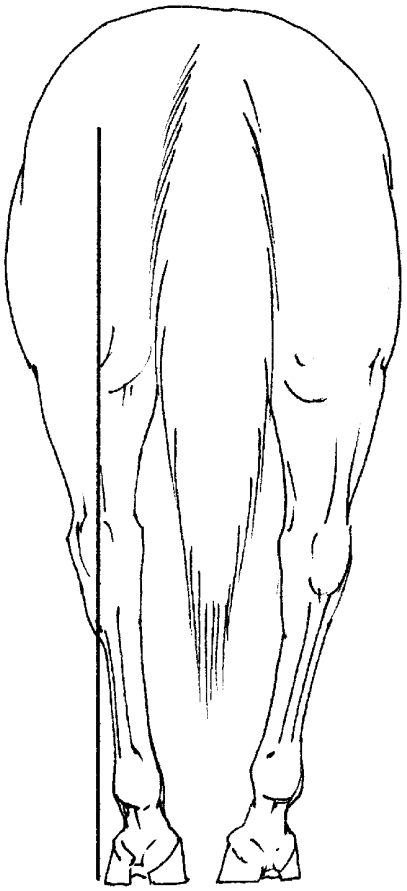


Figure 2.34. Base-narrow behind (compare with Figure 2.32). This is often accompanied by "bowlegs," as shown.

Excessively Straight Limbs, "Straight Behind"

When viewed from the side, there is very little angle between the tibia and femur, and the hock is excessively straight (large hock joint angle of more than 165°–170°) (Figure 2.29).[18,29] This is believed to predispose the horse to bog spavin, thoroughpin, upward fixation of the patella, and hindlimb proximal suspensory disease (PSD).[17,29,36] Older horses with straight angles are also prone to degenerative suspensory disease, and the prognosis of horses treated surgically for hindlimb PSD is negatively associated with straight hock conformation.[17] Horses with large tarsal angles showed less tarsal flexion and less energy absorption at the tarsus during the impact phase, potentially contributing to the development of OA.[18] Generally, the pastern conformation is usually too straight when the tarsus is too straight.

Medial Deviation of the Hock (Cow Hocks or Tarsal Valgus)

"Cow-hocked" means that the limbs are base-narrow down to the hock and then base-wide from the hock to the feet (Figure 2.33). The hocks are too close together and

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point toward one another, and the feet are widely separated. Cow-hocked conformation is a common defect, and some believe that it is a serious hindlimb conformation flaw.[36] However, others feels that a slight valgus deformity of the hindlimbs, provided that the metatarsal bones remain vertical, is not a cause for concern.[29] Sickle hocks and cow hocks can occur concurrently and may have a compounding effect on hindlimb lameness problems.

Base-Narrow

Base-narrow conformation of the hindlimbs means that the distance between the center of the feet is less than the distance between the center of the limbs in the thigh region, when viewed from behind (Figure 2.34). This is observed most commonly in heavily muscled horses. It causes excessive strain on the lateral aspect of the limb, and most of the horse's weight is placed on the outside of the hooves. Base-narrow conformation is often accompanied by "bowlegs" or a condition in which the hocks are too far apart. The limbs may appear fairly straight to the hock and then deviate inward, and the hocks may bow outward during movement.

Conformation of the Foot

Hoof anatomy and conformation are also discussed in Chapters 1 and 11.

The Forefoot

Ideally, the forefoot should be round and wide in the heels, and the size and shape of the heels should correspond to the size and shape of the toe. The bars should be well developed (Figure 2.35). The wall should be thickest at the toe and should thin gradually toward the heels; the inside wall should be slightly straighter than the outside wall. The sole should be slightly concave medial to lateral and front to back, but excessive concavity may suggest a chronic foot problem. There should be minimal contact between the ground and the sole, and toe lengths of greater than 1 1/2 inches cranial to the apex of the frog when viewed from the bottom of the foot may suggest excessive toe length.

The mean hoof angle of the forefoot is usually 50°–55°, and the angle of the heel should be approximately the same as the angle of the toe.[4,9] However, the angle of the hoof is less important than the hoof-pastern axis when evaluating foot conformation. Toe wall length relative to heel wall length should be approximately 2 : 1 in the forefoot and 2 : 1.5 in the hindfoot.[4] Break-over should occur squarely over the center of the toe and not over the medial or lateral portions of the toe. The frog should be large and well developed with a good cleft, have normal consistency and elasticity, and be dry and soft. It should divide the sole into two nearly equal halves, and the apex should point to the center of the toe. In most horses, the apex of the frog should end approximately 1–1 1/2 inches behind the toe.[7]

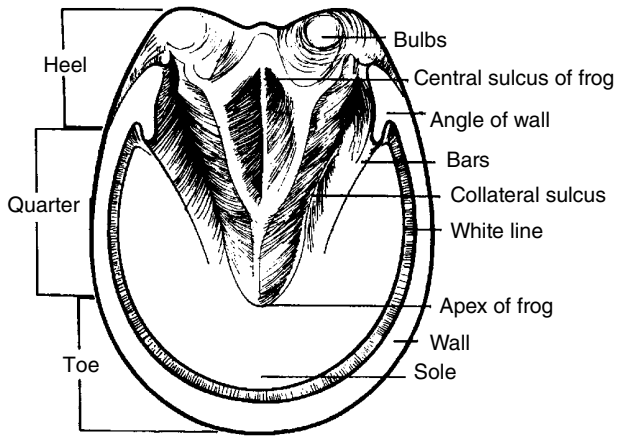


Figure 2.35. Normal forefoot with structures labeled.

The Hindfoot

The hindfoot is usually narrower, has a larger hoof angle, and has a more pointed appearance at the toe than the forefoot (Figure 2.35).[4,5] Break-over should be evident straight over the toe, and the frog should divide the sole into equal halves. The mean hoof angle in the hindfoot is usually 55° or greater, and there should be no defects in the wall.[4,36] The sole of the hindfoot is normally more concave than that of the forefoot.

Medial–Lateral Hoof Balance

ML hoof balance is often used to describe the relationship between the two halves of a hoof when viewed from the front and the levelness of the coronary band compared to the ground surface (Figure 2.37). It is used to assess the symmetry (or asymmetry in many cases) and can suggest the type of concussive forces on the hoof wall. In general, the coronary band should be level medial to lateral and parallel to the ground. In addition the lateral and medial aspects of the hoof wall should be approximately the same length and angle.

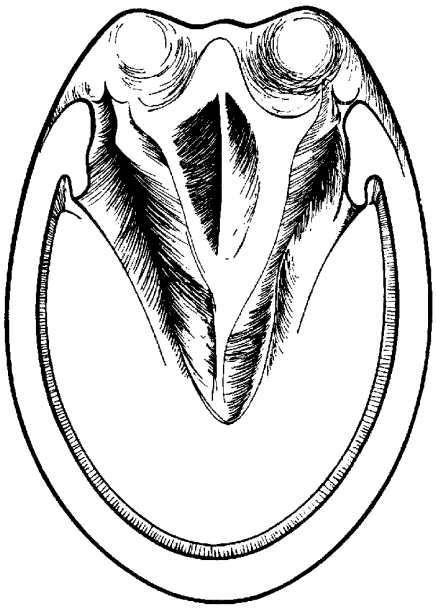


Figure 2.36. Normal hindfoot (compare with Figure 2.35). The toe of the hindfoot is more pointed than that of the forefoot.

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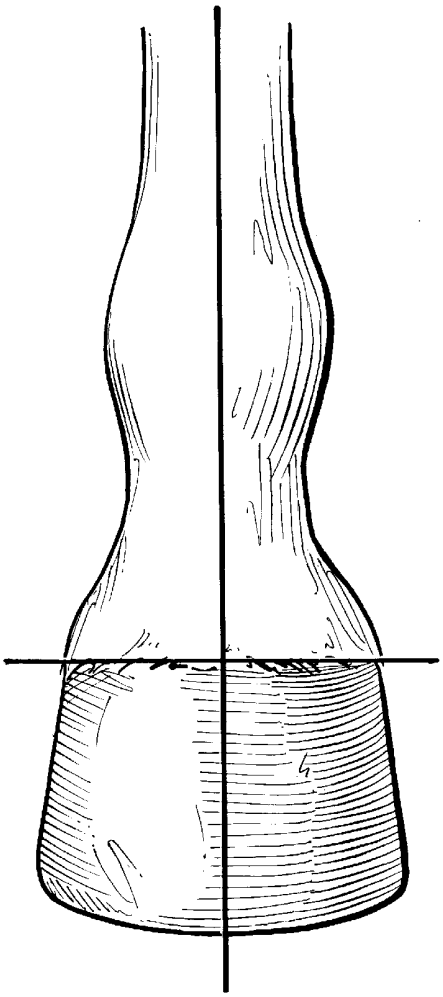


Figure 2.37. A hoof is considered to be in ML balance when an imaginary line through the coronet is parallel to the ground surface and perpendicular to a line that bisects the limb axis when viewed from the front.

Abnormal Conformation of the Foot

Flat Feet

A flat foot lacks the natural concavity in the sole; it is not a normal condition in light horses but is present in some draft breeds. Flat feet may be heritable and are observed more commonly in the forefeet than in the hind. The horse may land on the heels to avoid sole pressure with this condition. Sole bruising is a common sequela of flat feet. There is no known cure for flat foot, but corrective shoeing may help prevent resultant lameness.

Contracted Foot or Contracted Heels

A contracted foot is a condition in which the foot is narrower than normal, especially the back half of the foot (Figure 2.38). This condition is much more common in the front feet than in the hindfeet, and it may be unilateral or bilateral. Hoof contraction can occur rapidly in the heels with a long-toe, low-heel conformation, resulting in a low hoof angle.[7] Heel contraction also occurs with disuse of the foot as is seen with chronic lameness conditions. Heel contraction can be quantified using several techniques (Figure 2.39).

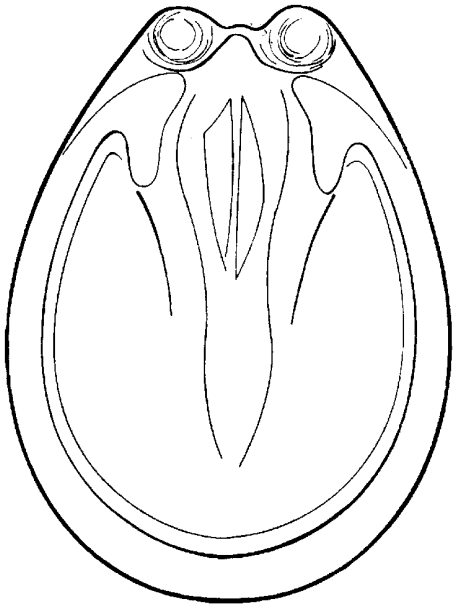


Figure 2.38. Contracted foot illustrating narrowing of the heels and quarters (compare with Figure 2.35).

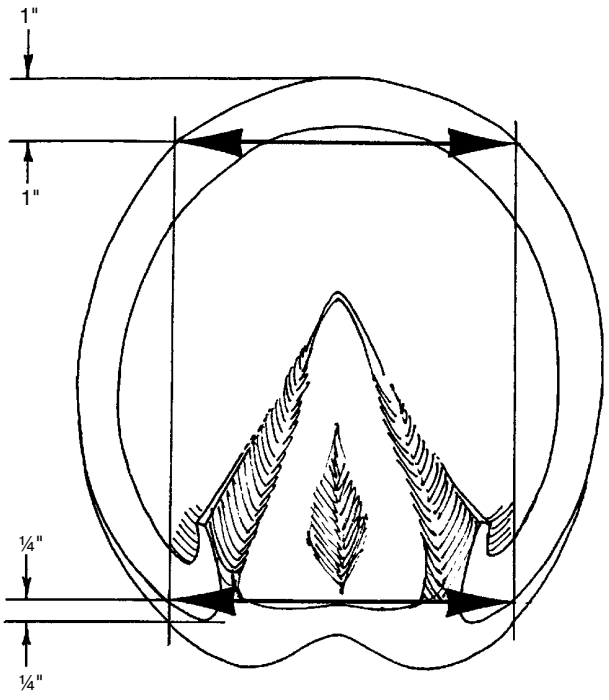


Figure 2.39. One method to quantify heel contraction. The width of the heels 1/4 inch from the heel buttresses should equal or exceed the width of the trimmed foot 1 inch behind the toe.

Certain breeds of horses normally have a foot that more closely approaches an oval than a circle in shape. A narrow foot is not necessarily a contracted foot; donkeys and mules normally have a foot shape that would be called contracted in a horse. Foot contraction may be present in the Tennessee Walking Horse and American

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Saddlebred when these horses are used for show because the hoof wall is allowed to grow excessively long.

A unilateral contraction of one forefoot may be present in some horses. This may be congenital or developmental, but it is not known whether it is a heritable abnormality. The contracted foot may or may not eventually show lameness, but it should be regarded as an undesirable feature. A small foot on one side with a normal opposite foot may be normal for certain horses, but it may also indicate a subclinical lameness in the foot with the steeper hoof angle.[42] This is often referred to as "mismatched" or "uneven" feet. In a recent study of Dutch Warmblood horses, the prevalence of "uneven" front feet was 53% with weak genetic correlations. However, the study concluded that reducing the occurrence of uneven feet by selection was possible without limiting progress in sport horse performance.[15]

Buttress Foot

"Buttress foot" is a swelling above the dorsal surface of the hoof wall at the coronary band (Figure 2.40). This swelling may be from low ringbone or a fracture of the extensor process of the distal phalanx (coffin bone). A conical deformity of the toe from the coronary band to the ground surface occurs due to the deformed hoof growth associated with chronic swelling at the coronary band. This often results in a triangular or pyramidal shape to the foot (Figure 2.41).

Club Foot

A "club foot" usually refers to a foot with a foot axis greater than or equal to 60° (Figure 2.42). A unilateral club foot may be due to an injury that has prevented proper use of the foot (disuse due to chronic lameness) or from a flexural deformity involving the deep digital flexor tendon, seen primarily in foals. It may be developmental or acquired and may or may not contribute to a lameness problem.[7,29]

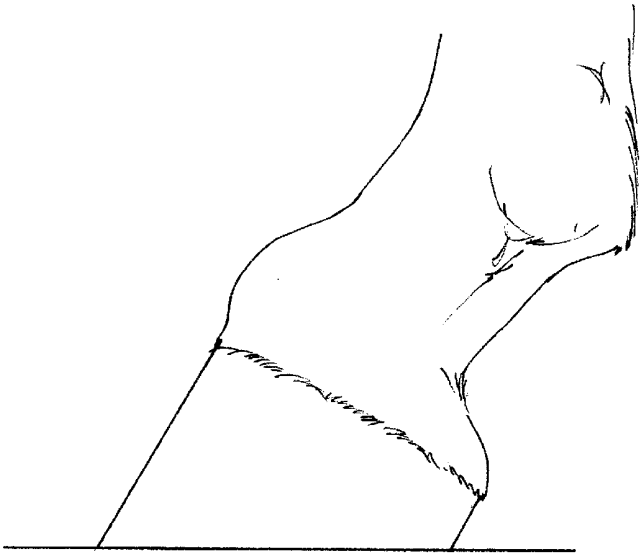


Figure 2.40. Buttress foot.



Figure 2.41. Front view of a horse with a buttress foot associated with a large extensor process fracture of the distal phalanx. The foot has assumed a triangular or pyramidal shape.



Figure 2.42. Young horse with a club foot due to a flexural deformity of the deep digital flexor tendon. There can be many causes of a club foot and not all contribute to lameness.

ACKNOWLEDGMENT

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PALPATION AND MANIPULATION

Gary M. Baxter and Ted S. Stashak

Palpation of the musculoskeletal system is a very important aspect of any lameness evaluation.[2] With practice and experience, subtle abnormalities can be detected that are often indicative of the site of the problem. Thorough palpation of the patient is often performed prior to observing it at exercise, but this is often patient and clinician dependent. Most manipulative tests are performed after exercise. A systematic approach of palpation is recommended to avoid missing abnormalities. One approach is to palpate the forelimb proximally to distally in a weight-bearing position and then distally to proximally with the limb picked up or unweighted (Video 2.1).[2] Palpation of the hindlimbs can be performed in the same manner, paying close attention to the medial aspects of the stifle and tarsus (Video 2.2). The back and axial skeleton are usually palpated last because some horses become agitated with manipulation of the back (Video 2.3). Hoof tester examination of the feet is usually performed after the entire musculoskeletal system has been palpated. The following discussion briefly describes techniques to visually examine, palpate, and manipulate the different anatomic regions of the equine musculoskeletal system to ensure a complete examination.

FOOT

The size and shape of the foot on the lame limb should be compared to its opposite member. The examiner is looking for asymmetry in foot size, abnormal hoof wear, ring formation and heel bulb contraction, shearing of the heels and quarters, hoof wall cracks, swellings that are primarily associated with the coronet, and foot imbalances.[15,17] Asymmetry in foot size may be a result of trauma, lack of weight-bearing leading to contraction, and congenital or developmental defects. In general, the limb with the smallest foot is usually the lame limb. Hoof wall ring formation can be unilateral (trauma) or bilateral and is not always associated with lameness (Figure 2.43). Heel contraction often results from decreased weight-bearing of the affected limb and is usually a symptom rather than the cause of the lameness (Figures 2.44 and 2.45). Visual examination of heel bulb contraction is best performed with the examiner standing or squatting near the flank and looking at both right and left heel bulbs at once (Figure 2.44). Asymmetry in heel bulb height (sheared heels) is most frequently associated with improper trimming and shoeing. Foot imbalances can either be dorsopalmar/plantar (DP), lateral/medial (LM), or a combination of the two (Figures 2.44 and 2.45). These imbalances often alter the shape of the hoof wall and can result in abnormal stresses applied to the foot and other support structures.

After superficial cleaning of the sole, abnormal wear on the shoe and/or sole, collapsed heels, heel bulb contraction, and frog atrophy should be noted (Figure 2.46). Secondary frog atrophy may accompany heel contraction



Figure 2.43. Bilaterally symmetrical hoof wall rings that were not associated with lameness or any other known problem within the hoof wall.



Figure 2.44. View of both front feet in a horse with forelimb lameness. The front feet of the same horse as viewed from the side are illustrated in Figure 2.45. The medial wall of the right foot is concave with the coronary band pushed proximally suggesting excessive concussion. In addition the heels are contracted and overgrown.

in chronic cases. The sole should be slightly concave. Some horses are flat-footed and therefore predisposed to sole bruising. Convexity of the sole dorsal to the apex of the frog ("dropped soles" in front of the frog) is considered abnormal and is often associated with rotation of the distal phalanx. In some cases, the offending cause of lameness may be identified immediately, such as a nail wedged in the frog (Figure 2.47). However, the clefts of the frog may need to be opened with a knife to properly evaluate the depths of the sulci for evidence of thrush or canker.

The hoof wall should be checked for cracks that may extend into the sensitive laminae (they are most common in the toe and quarter), uneven wear, and excessive

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Figure 2.45. Front feet of the horse in Figure 2.44 as viewed from the side. The front feet have markedly different hoof angles. The right foot has contracted heels resulting in a very upright conformation compared to a low underrun heel with a long toe on the left foot, resulting in a low hoof conformation. In addition, the coronary band on the right has an abnormal contour suggesting foot imbalance.



Figure 2.46. A front foot with severely overgrown heels that have resulted in atrophy of the frog. The frog should be prominent and approximately level with the widest part of the heels.

dryness (Figure 2.48). Dishing (concavity) of the dorsal aspect of the hoof wall is often indicative of chronic rotation of the distal phalanx, excessive toe length, or a flexural deformity involving the deep digital flexor tendon (DDFT) (Figure 2.49).

A hoof tester is an instrument that permits deep palpation of the sole, frog, and wall of the hoof (Figure 2.50). When applied properly, the examiner tries to identify and localize hoof sensitivity (Video 2.4). Most normal horses should be able to withstand a fair bit of hoof tester pressure without showing signs of discomfort. The arm of the hoof tester that is applied to the hoof wall is continually checked so pressure is not being applied to the coronary band.



Figure 2.47. This horse presented for an acute onset hindlimb lameness. A nail was found protruding from the apex of the frog. Based on the location, entry into the navicular bursa would be unlikely.



Figure 2.48. Partial-thickness dorsal hoof crack associated with a long toe and a concavity of the dorsal hoof wall. Both factors most likely contributed to the development of the crack in this horse.

The order of hoof tester application is less important than being systematic and complete (Video 2.4). One method is to begin at the lateral or medial angle of the sole and continue hoof tester pressure at 2- to 3-cm intervals until the entire surface of the sole is checked. This is followed by pressure applied to the frog (caudal, central, and cranial) from both the medial and lateral heel. Lastly,

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Figure 2.49. Concavity of the left front foot in a horse with chronic laminitis. This horse was most lame in the left forelimb.

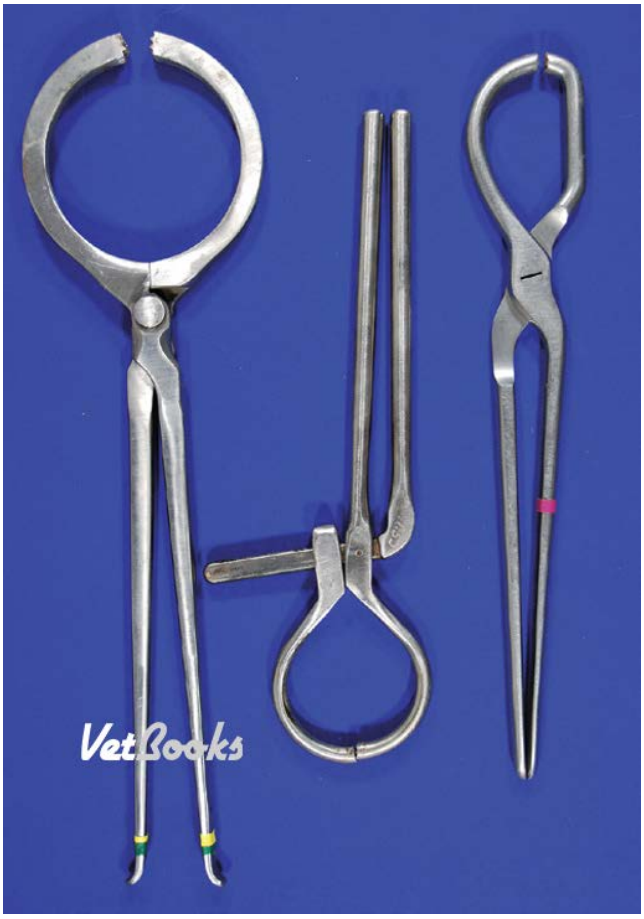


Figure 2.50. Examples of several types of hoof testers. Left: GE Forge and Tool Works, 959 Highland Way, Grover Beach, CA, 93433. Middle, Ryding Hoof Tester, Jorgenson Labs, 2198 W 15th St., Loveland, CO, 80537. Right: Kane Enterprises, AG-TEK Division, P.O. Box 1043, Sioux Falls, SD, 57101.

the hoof tester is applied to the hoof wall across the heels, and then it can be applied diagonally from the medial heel to the dorsolateral hoof and then from the lateral heel to the dorsolateral hoof. If sensitivity is encountered, it is necessary to confirm whether the response is from pain and not just a whimsical reaction by the horse. Repeatability is the key to being confident with your findings. True sensitivity is identified by repeated intermittent hoof tester pressure that results in persistent reflexive withdrawal (flexing the shoulder and trying to take the foot away) with hoof tester pressure. Obviously varying amounts of hoof tester pressure are applied to elicit a response, and this is dependent on sole thickness and the painfulness of the condition. Hoof tester responses should be compared with those obtained from the opposite foot.

In general, diffuse sole sensitivity may suggest a sagittal fracture of the distal phalanx, extensive sole bruising, and in some cases laminitis. More localized hoof tester sensitivity is usually obtained with sole bruising, puncture wounds, close or hot nail, and localized subsolar abscesses. Hoof tester sensitivity over the central third of the frog is usually consistent with pain in the region of the navicular bone (Figure 2.51). A hoof tester or a hammer can also be used to strike (percuss) the hoof wall. A painful response may be suggestive of laminitis or a defect along the hoof wall. A hollow sound over the dorsal hoof wall may suggest separation between the sensitive and insensitive laminae (e.g. white line disease, chronic laminitis, etc.).

The coronary band should be palpated for heat, swelling, and pain on deep pressure. Effusion of the distal interphalangeal (DIP) joint is usually palpable just dorsal and proximal to the coronary band (Figure 2.52).[2,5] Using both hands can facilitate determining if the swelling is fluid or thickened tissue. Firm, often nonpainful swelling in this region may also be evidence of low ringbone. Heat, pain, and swelling with or without drainage of one of the heel bulbs are often found in horses with subsolar abscesses (Figure 2.53) as many of these abscesses tend to break out in the heel bulb region.



Figure 2.51. A Ryding Hoof Tester being applied over the central third of the frog of the forefoot to produce direct pressure over the navicular region.

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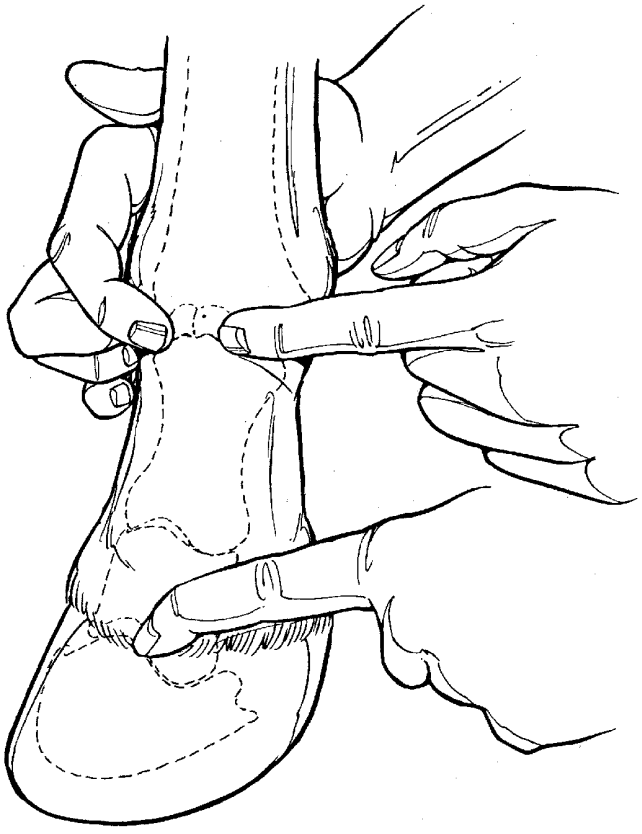


Figure 2.52. Lower finger marks the site of swelling and pain associated with fracture of the extensor process. Upper fingers are applied over the dorsal aspect of the fetlock to identify synovial distension and thickening of the joint capsule.

PASTERN

The dorsal, medial, and lateral surfaces of the proximal interphalangeal (PIP) joint should be palpated for enlargement and heat, which may be suggestive of high ringbone (Figure 2.54). Comparison to the opposite pastern is always recommended. However, it is not uncommon to have the lateral to medial dimensions of one pastern to be slightly larger than its opposite member. With the limb off the ground, the distal sesamoidean ligaments and flexor tendons (superficial and deep digital flexors) are palpated deeply for pain, heat, and swelling (Figure 2.55). Particular attention is paid to the lateral and medial branches of the superficial digital flexor tendon (SDFT) as they attach to the middle phalanx. Tendonitis of the DDFT and/or tenosynovitis of digital flexor tendon sheath (DFTS) are often identified by swelling, effusion, and sometimes pain in this region. Pain on deep palpation of the DDFT just above the heel bulbs using your thumb may be suggestive of injuries to the tendon further distally in the hoof capsule. Deep palpation of the lateral and medial eminences (wings) of the middle phalanx may elicit pain if a fracture is present. With the hands placed on the hoof wall, the phalangeal joints should be rotated medially and laterally. The collateral ligaments can be stressed, laterally and medially, by placing one hand lateral or medial over the pastern joint, while the other hand is used to pull the foot toward that side (Figure 2.56). This bending force creates increased tension on the collateral ligaments of the phalangeal joints.



Figure 2.53. Palpation of the heel bulbs to identify heat, pain, and swelling that may be associated with subsolar abscesses.

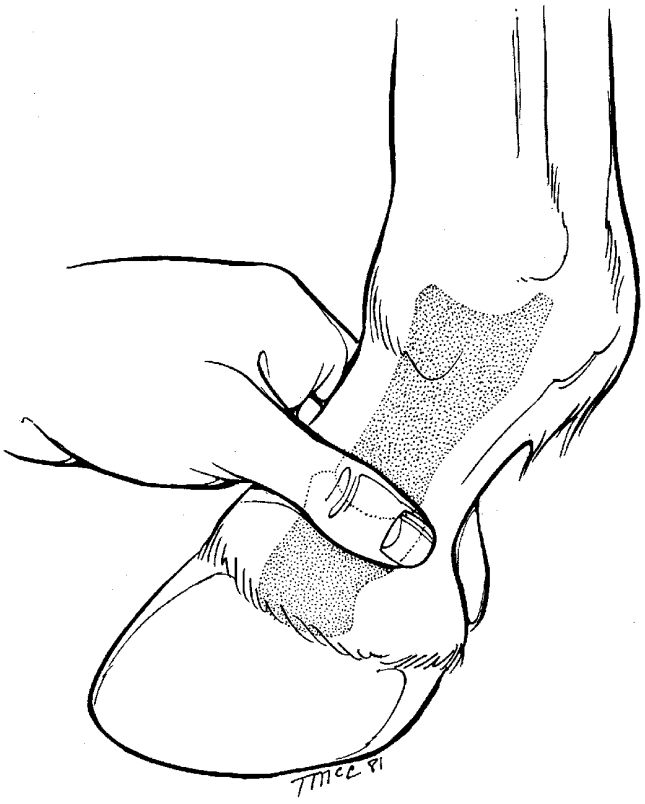


Figure 2.54. Palpation of the pastern. Thickening in this region may suggest the presence of ringbone.

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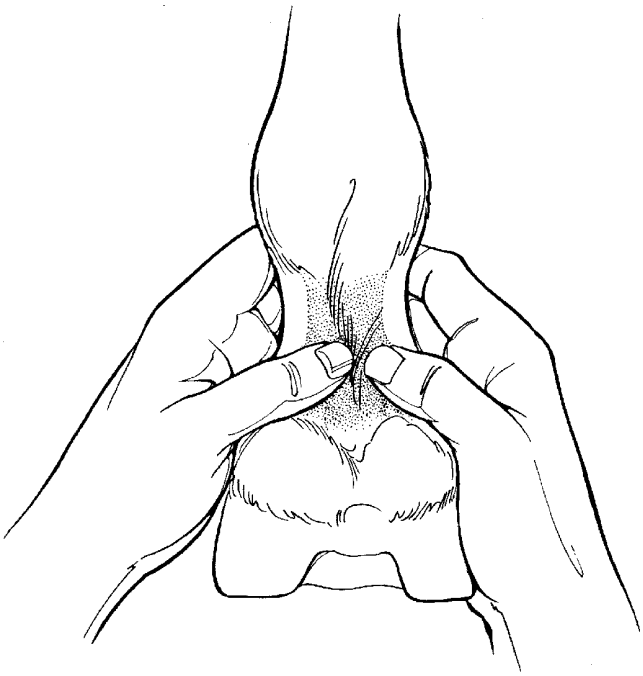


Figure 2.55. Palpation of the distal sesamoidean ligaments, branches of the superficial digital flexor tendon (SDFT), and the deep digital flexor tendon (DDFT) in the palmar/plantar aspect of the pastern. The DDFT should be palpated with digital pressure using your thumb as far distally as possible between the heel bulbs.

FETLOCK

The dorsal and palmar/plantar pouches of the metacarpophalangeal/metatarsophalangeal (MCP/MTP) joint should be palpated for swelling, effusion, or thickening of the joint capsule (Figures 2.52 and 2.57). Using both hands to help palpate the multiple joint pouches is very helpful.[2] Pressure should be applied to the lateral and medial branches of the suspensory ligament just above their attachments to the proximal sesamoid bones. Pain and/or swelling at this location may suggest desmitis of the suspensory branch, sesamoiditis, or apical/abaxial fractures of the sesamoid bone. The SDFT and DDFT and digital sheath should also be palpated for heat, pain, swelling, or effusion. Some distension of the DFTS of all four limbs is not uncommon in performance horses. This often is referred to as "wind puffs."

With the limb off the ground, thumb or finger pressure should be applied to the basilar, body, and apical portions of the proximal sesamoid bones (Figure 2.58). Sensitivity and pain may indicate a sesamoid fracture or desmitis of the suspensory ligament. The fetlock should be rotated and the collateral ligaments checked in a similar manner to that of the pastern joint (Figure 2.56). Additionally, the fetlock joint should be passively flexed to identify pain and assess the range of motion. This is accomplished by extending the carpus as much as possible and flexing the fetlock by placing one hand on the pastern (Figure 2.59). This technique flexes the fetlock joint separate from the phalangeal joints in contrast to all of the joints of the distal limb (Figure 2.60).

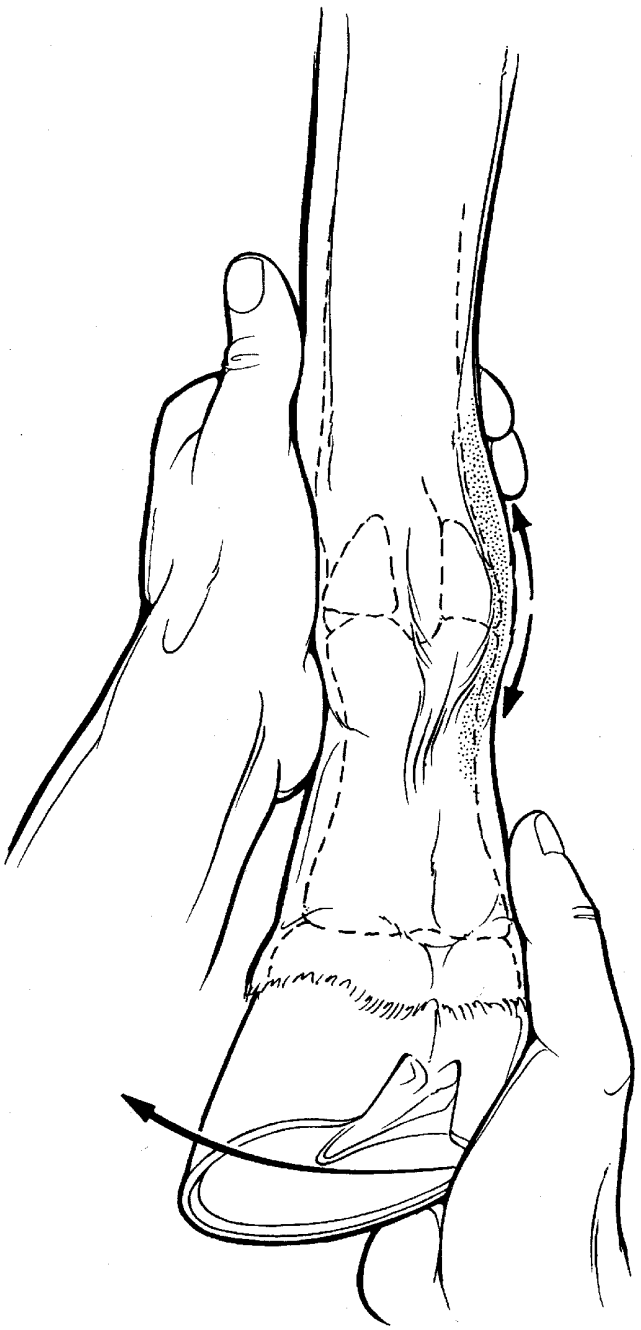


Figure 2.56. Tension is applied to the collateral ligaments supporting the fetlock and interphalangeal joints (pastern and coffin) to identify pain.

METACARPUS/METATARSUS (MC/MT)

Palpation of the dorsal aspect of the metacarpus should be performed, especially in young racehorses. Firm pressure applied with the fingertips often elicits a painful response in horses with dorsal metacarpal disease (buck shins). Heat and swelling over the dorsal middle third of the metacarpus may also be present (Figure 2.61). The extensor tendons on the dorsal surface of the MC/MT should be palpated for swelling, thickness, and pain, especially in horses with a history of trauma/laceration to this region.

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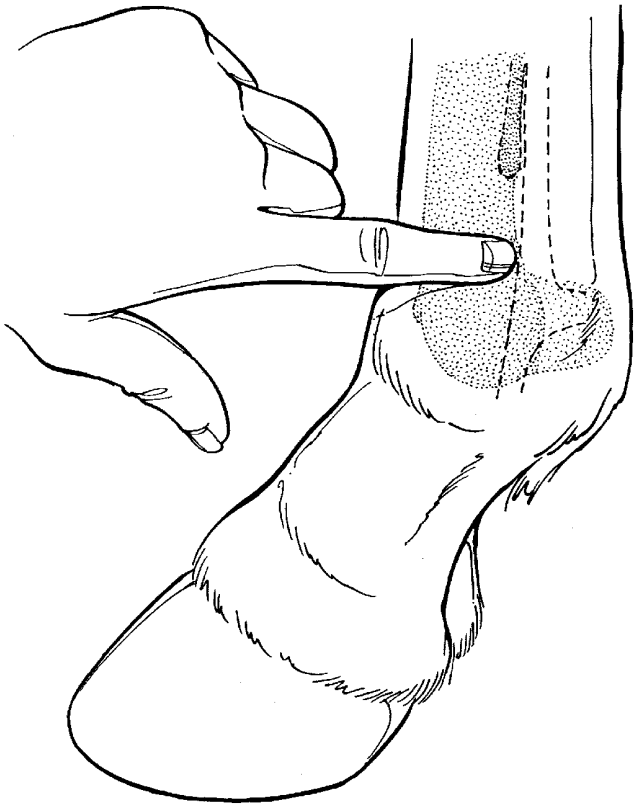


Figure 2.57. Finger marks the palmar recesses of the fetlock joint capsule. Distention at the site results from synovial effusion.

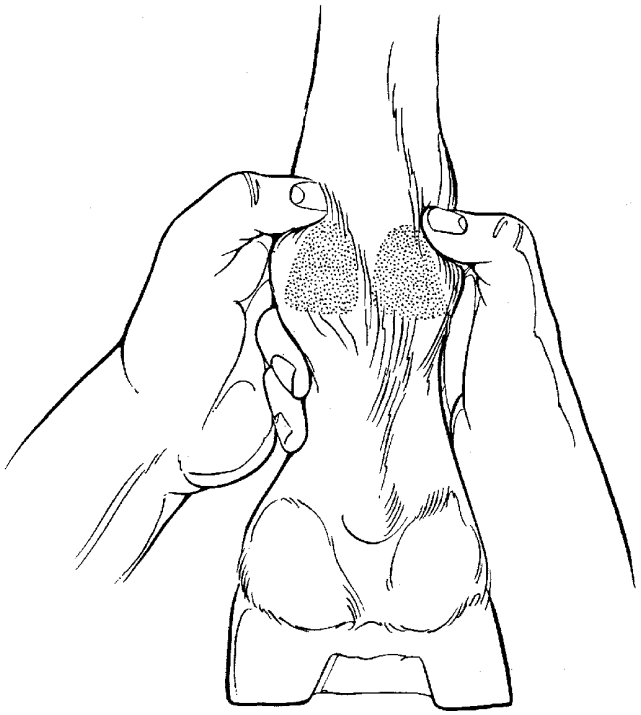


Figure 2.58. Digital pressure applied to the apical sesamoid region to detect pain, heat, and asymmetry. Palpation of the midbody and basilar aspects of the bones should also be performed.

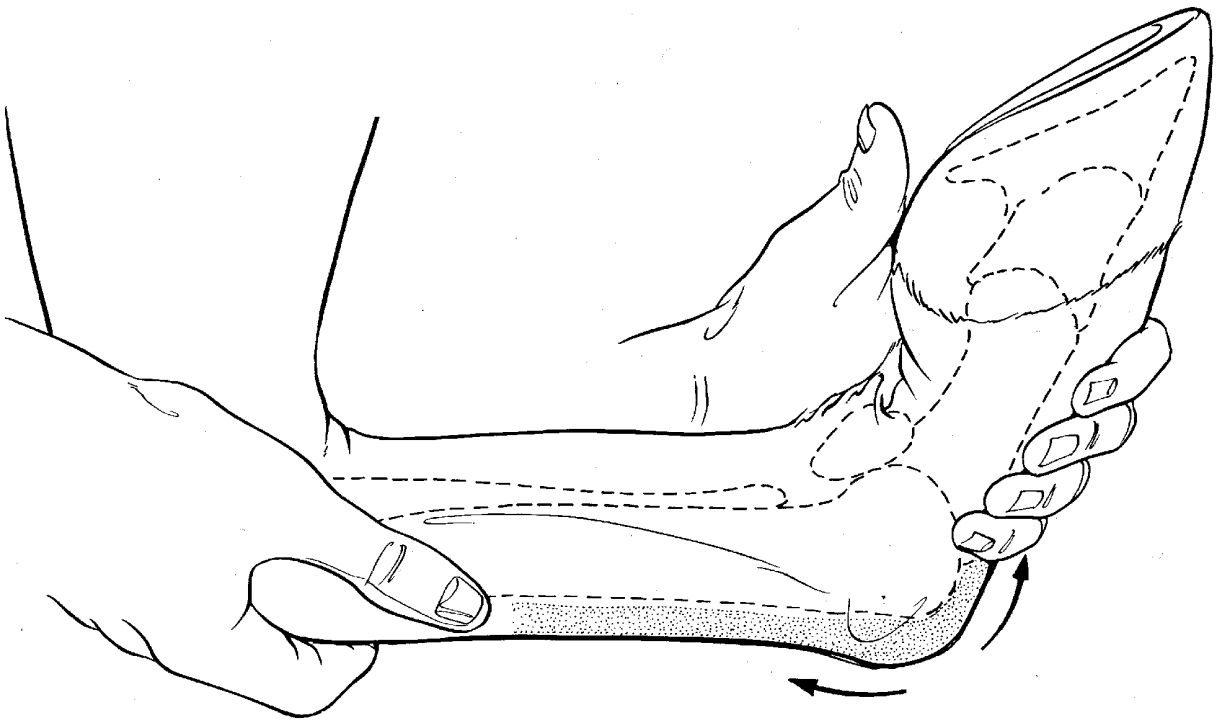


Figure 2.59. Fetlock flexion test is performed by extending the carpus and flexing the fetlock joint. One hand is placed on the dorsal aspect of the pastern to create fetlock flexion without flexion of the interphalangeal joints.

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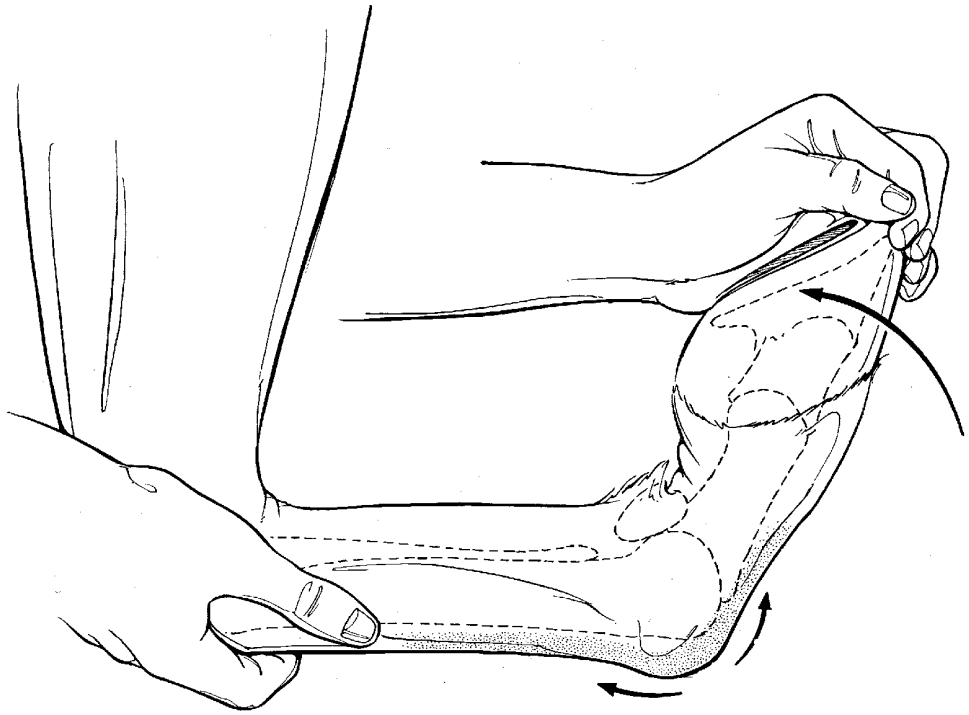


Figure 2.60. Distal limb flexion test in which the interphalangeal (pastern and coffin) and fetlock joints are flexed simultaneously.

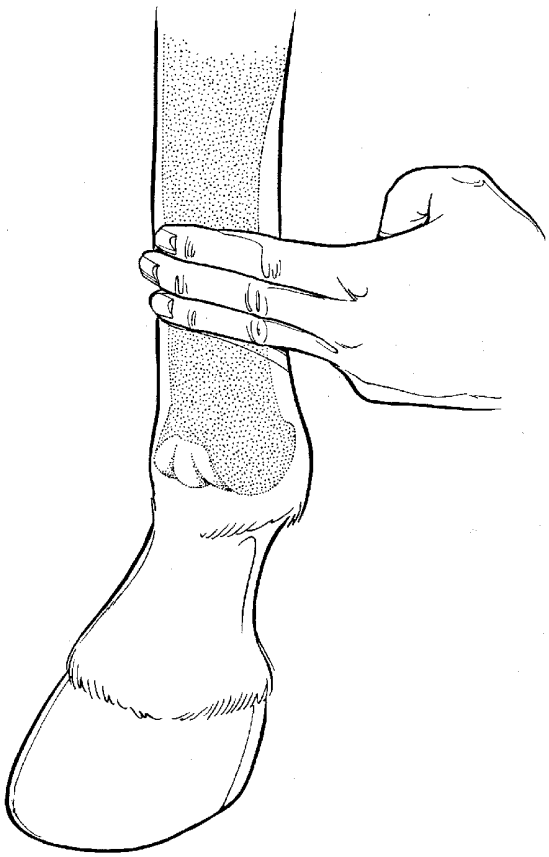


Figure 2.61. Palpation over the dorsal middle third of the metacarpus to identify heat, pain, and swelling associated with dorsal metacarpal disease.

The entire length of each small metacarpal/metatarsal bone (splint bone) should be palpated for heat, pain, and swelling with the limb weighted and unweighted. With the limb elevated, the palmar/plantar and axial surfaces of the splint bones can be palpated by pushing the suspensory ligament and tendons toward the opposite side (Figure 2.62). The splint bone can be palpated with the thumb applying pressure as needed. Heat, pain, and swelling may indicate a fracture or a condition referred to as "splints" if located in the proximal aspect of MCII. Splint fractures most commonly involve the medial splint bone in the forelimb and the lateral splint bone of the hindlimb. It is not uncommon to palpate nonpainful enlargements of the splint bone, which are often incidental findings. However, some middle and proximal splint bone fractures heal with excessive callus that may contribute to lameness.

SUSPENSORY LIGAMENT

The suspensory ligament (interosseous medius muscle) lies just palmar/plantar to the splint bones in the metacarpal/metatarsal groove. It should be palpated with the limb weight-bearing and with the limb flexed. Deep palpation is often needed to identify swelling and pain, and comparison between the lateral and medial branches may be helpful.[2,6] Damage to the suspensory tends to occur distally within the branches of the suspensory ligament or at its proximal attachment to the MC/MT. However, secondary suspensory desmitis may be associated with a healing splint fracture anywhere along its length. The proximal attachment of the suspensory ligament to the MC/MT can be palpated by pushing the flexor tendons to the side and applying pressure

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Figure 2.62. Palpation of the medial (axial) surfaces of the small metacarpal bones. The fetlock can be flexed to relax the suspensory ligament to permit easier palpation.

with the thumb with the limb held in a flexed position (Figure 2.63). Alternatively, pressure can be applied to this region by placing the palm of the hand on the dorsal MC and wrapping the fingers around the medial side of the MC. The fingertips are used to "squeeze" the limb and apply pressure to the proximal palmar MC region (Figure 2.64). Many horses may react initially by withdrawing (flexing) the limb. However, with constant pressure, this response often fatigues. A painful withdrawal that persists (does not fatigue) often suggests a problem in the proximal suspensory region.[10,15] Palpation of the proximal suspensory region is more difficult in the hindlimbs than the forelimbs because it is closely surrounded by the small metatarsal bones and the SDFT is less easily pushed to the side (Figure 2.65).

FLEXOR TENDONS

The SDFT and DDFT are located palmar to the suspensory ligament and are intimately associated with each other. The proximal one-third of the flexors (associated with the carpus) and distal one-third (associated with the fetlock) are encased in tendon sheaths, whereas the central one-third is covered by a paratendon only. Each region should be palpated carefully for heat, pain, and swelling with the tendons weighted and then again with the limb unweighted and the tendons relaxed. With the limb held in one hand, an attempt should be made to roll or separate the SDFT from the DDFT with the thumb and forefinger (Figure 2.66). If normal, they can be easily separated and differentiated. With pathology such as tendonitis, varying degrees of adhesions between the two as well as thickening will result in an inability to separate them. Most horses respond slightly to "pinching" the SDFT between the thumb and index finger. Pain will be elicited easily with palpation in most horses with tendonitis. The inferior check ligament (accessory ligament of the DDFT) lies directly palmar to the suspensory ligament and can be palpated by holding the ligament between the index finger and the thumb or by applying pressure from the palmar aspect with the thumb.

CARPUS

The carpus should be visually examined for swelling/effusion on the dorsal and palmar surfaces. Point swelling associated with the radiocarpal and middle carpal joints that occurs medial to the extensor carpi radialis tendon is often present in horses with osteochondral chip fractures and/or OA (Figure 2.67). More diffuse swelling of these joints may indicate more severe articular pathology. Distention of the tendon sheaths of the extensor tendons overlying the carpus (common digital and extensor carpi radialis) is uncommon but may indicate tenosynovitis and/or rupture, particularly of the common digital extensor tendon in foals (Figure 2.68). A diffuse fluctuant, subcutaneous swelling over the dorsal surface of the carpus is consistent with acute hematoma/seroma or chronic hygroma (Figure 2.69). Swelling/effusion of the palmar carpal canal may be found with accessory carpal bone fractures, tenosynovitis (carpal tunnel syndrome), or osteochondroma formation of the caudal distal aspect of the radius (Figure 2.70).[15]

Palpation of the carpal joints and bones including the accessory carpal bone is best done with the carpus flexed. The degree of carpal flexion or range of motion should also be evaluated. In the normal horse the flexor surface of

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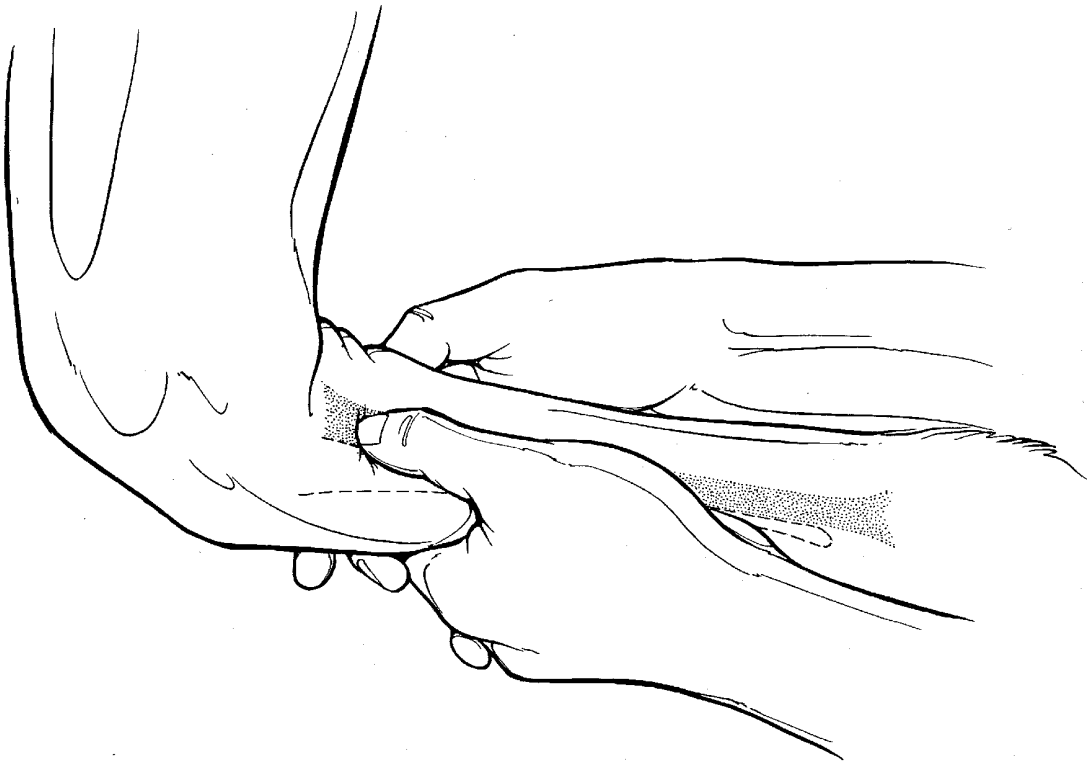


Figure 2.63. Palpation of the origin of the suspensory ligament in the proximal aspect of the metacarpal region. A repeatable painful response may suggest proximal suspensory desmitis.



Figure 2.64. Method used to apply digital pressure to the suspensory ligament in the proximal palmar metacarpal region.



Figure 2.65. Method used to apply digital pressure to the suspensory ligament in the proximal plantar metatarsal region.

the metacarpal region can approximate that of the forearm when the carpus is flexed (Figure 2.71). With the carpus held in flexion, the individual carpal bones are evaluated by deep digital pressure along the dorsal articular surfaces (Figure 2.72). With the carpus flexed and the tension of the ulnaris lateralis and flexor carpi ulnaris reduced, the accessory carpal bone can be manipulated, potentially identifying a fracture (Figure 2.73). In some cases, an osteochondroma on the caudodistal aspect of the radius may be palpable if effusion of the carpal canal is present.

FOREARM (ANTEBRACHIUM) AND ELBOW

The distal aspect of the radius, soft tissues of the forearm, and the elbow joint (cubital joint) should be palpated for swelling, heat, and pain. A firm, usually nonpainful but fluctuant swelling at the point of the elbow is consistent with an elbow hygroma, also known as olecranon bursitis (Figure 2.74). Severe swelling and an inability to extend the limb (dropped

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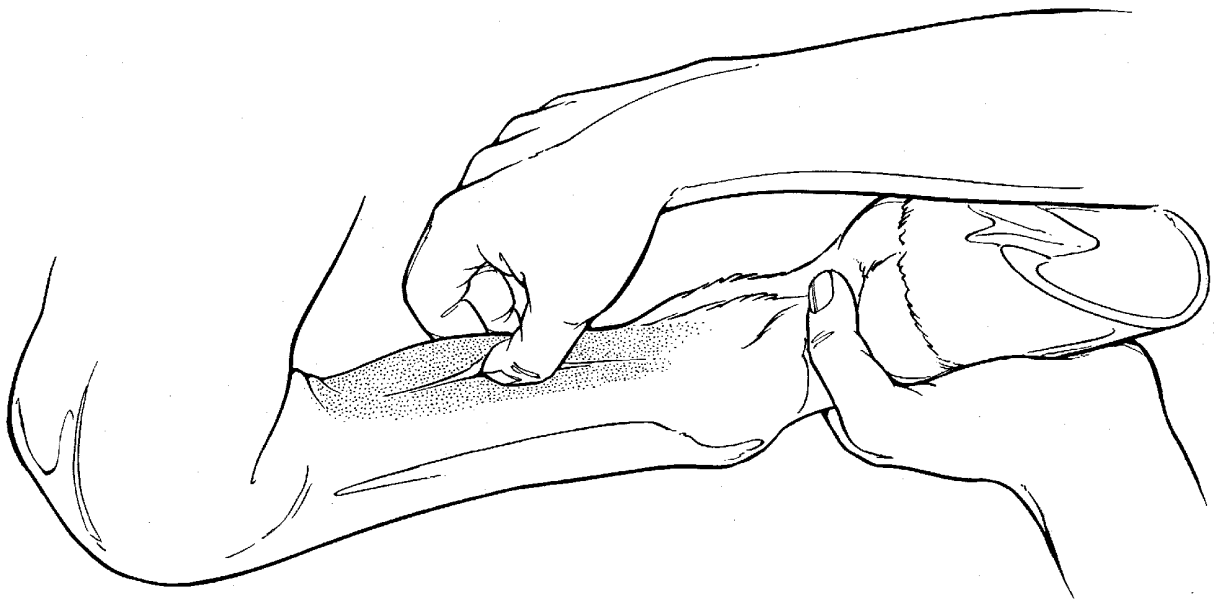


Figure 2.66. Palpation of the flexor tendons with the fetlock flexed to permit separation of the superficial and deep digital flexor tendons. Inability to separate the tendons usually suggests tendonitis.



Figure 2.67. Effusion of the radiocarpal joint was visible and easily palpable in both the dorsomedial and palmarolateral joint pouches in this horse.



Figure 2.68. Effusion of the extensor carpi radialis tendon sheath is usually characterized by swelling that courses up and down the cranial aspect of the carpus.

elbow) are consistent with a fracture of the olecranon. In cases of non-displaced or chronic olecranon fractures, palpation of the caudal aspect of the olecranon may reveal variable degrees of swelling and pain with digital pressure. Elevation of the limb into extension may elicit a painful response (Figure 2.75). Severe swelling, crepitation, and pain may be observed in horses with humeral fractures and can be confused with fractures of the olecranon.

SHOULDER AND SCAPULA

The soft tissues around the shoulder joint (scapulohumeral joint) should be observed for muscle atrophy and palpated for swelling, pain, and/or crepitus. Infraspinatus

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Figure 2.69. A hygroma of the carpus is a fluid-filled swelling located under the skin on the cranial aspect of the carpus. It is usually confined to the cranial aspect of the carpus and does not course up the limb.

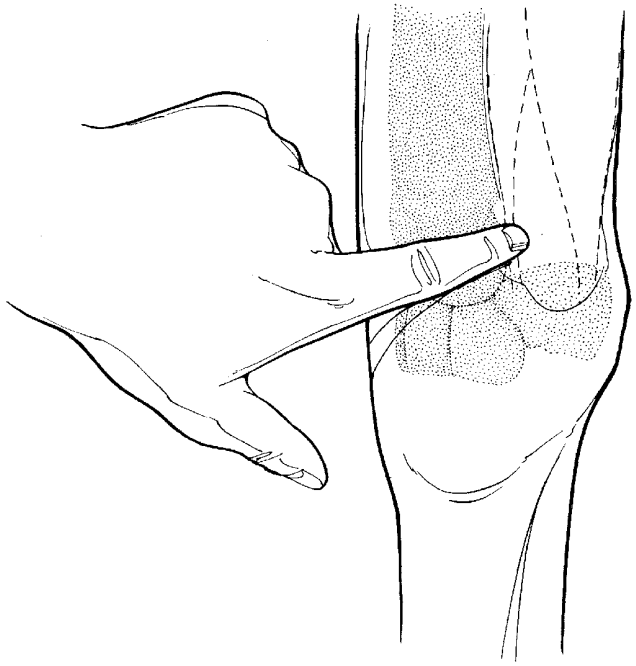


Figure 2.70. Finger marks the carpal canal. Effusion within the carpal sheath usually courses longitudinally up the medial aspect of the carpus but may also be present laterally.

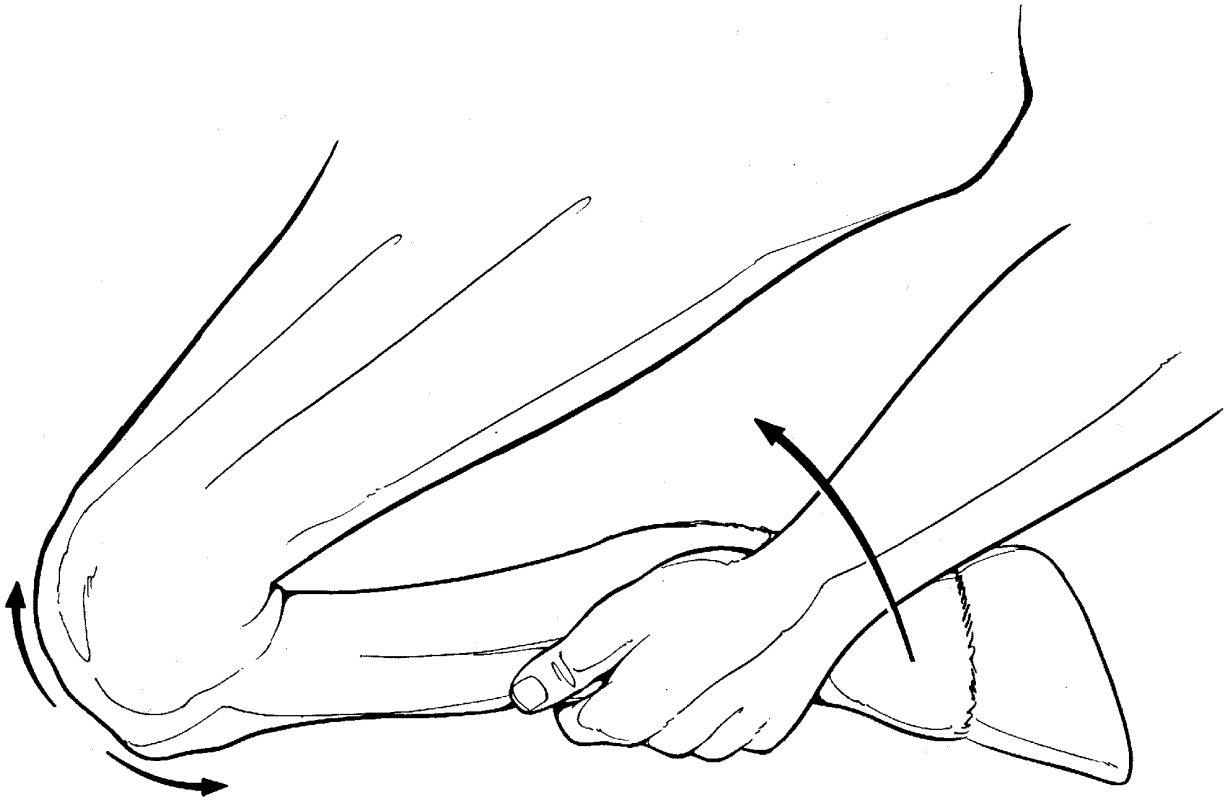


Figure 2.71. Flexion of the carpus to identify a painful response. In the normal horse, the flexor surface of the metacarpus approximates that of the forearm.

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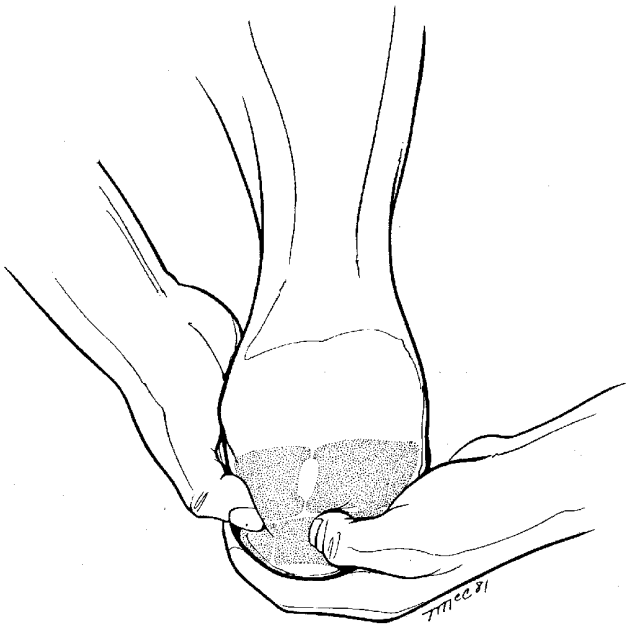


Figure 2.72. The dorsal articular margins of the carpal bones can be palpated after the carpus is flexed to identify pain within the individual carpal bones and/or capsulitis.



Figure 2.74. A small fluid-filled swelling was present on the point of the elbow in this horse, consistent with olecranon bursitis. The skin was also thickened in the region, but no lameness was present.

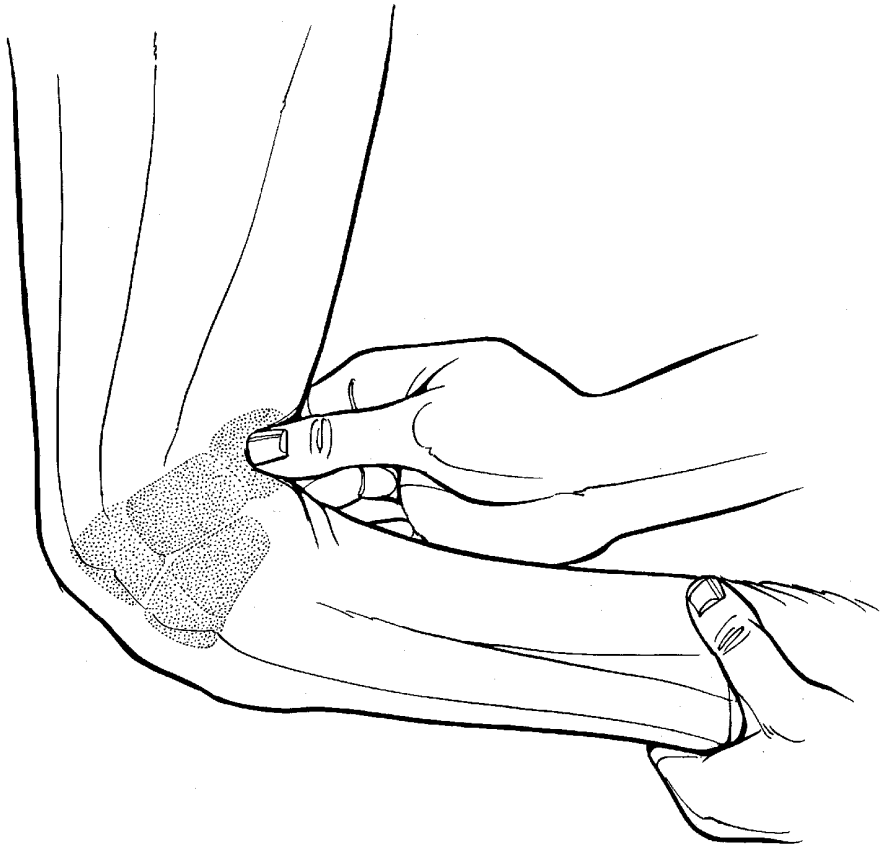


Figure 2.73. Palpation of the accessory carpal bone to identify a possible fracture. This is best done with the carpus flexed to decrease the tensional influence of the tendinous insertions of the ulnaris lateralis and flexor carpi ulnaris muscles.

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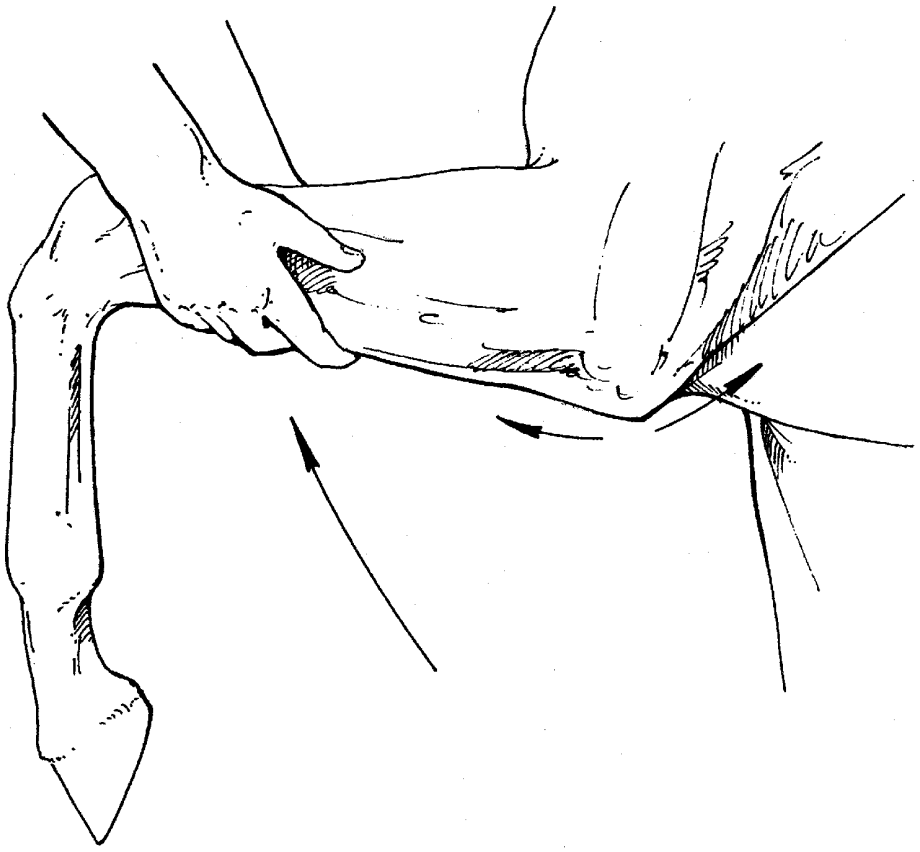


Figure 2.75. Elevating the limb into extension to flex the elbow joint extends the shoulder and increases the tension on the triceps brachii tendon at its insertion on the olecranon process.

and supraspinatus muscle atrophy is commonly seen in horses with suprascapular nerve paralysis or osteochondrosis (Figure 2.76). Particular attention should also be paid to the bicipital bursa region (cranial aspect of shoulder), and deep digital palpation is recommended in this region. The muscle and tendon should be grasped with the fingers and thumb and pulled laterally. The shoulder joint can be palpated deeply by applying thumb pressure cranial to the infraspinatus tendon at the location for intra-articular injection. A consistent painful response is suggestive of intra-articular pathology. Fractures of the scapula and proximal humerus may also elicit a painful response with this manipulation. Elevation of the limb as described for the elbow joint may also result in a painful response, particularly if a fracture or a lesion within the joint is present (Figure 2.75). Horses with midbody scapular fractures often have an external swelling located above the shoulder joint directly over the scapula. These, often young horses, will carry their distal limbs toward midline.

TARSUS (HOCK)

The tarsus is a common site contributing to hindlimb lameness and is complicated anatomically. Understanding the anatomy is critical to be able to accurately diagnose problems in this area. Synovial structures located in the tarsus include the tarsocrural joint, tarsal sheath, and calcaneal bursa. The tarsocrural joint has multiple outpouches that can result in tarsal swelling, and this soft, fluctuant swelling is often referred to as "bog spavin." The synovial effusion can be easily compressed from the dorsal pouches to distend the plantar pouches of the joint capsule and vice versa (Figure 2.77). Firm, diffuse swelling of the tarsus can occur due to trauma and inflammation of the fibrous joint capsule (capsulitis) and surrounding ligamentous support structures. This firm swelling may or may not be associated with synovial effusion of the joint (Figure 2.78). Effusion/swelling of the tarsal sheath is often referred to as "thoroughpin" and can usually be observed and palpated on the medial aspect of the tarsus. Unlike effusion of the tarsocrural joint, tarsal sheath effusion is asymmetrical on the limb (medially) and runs in a distal to proximal direction (Figure 2.79). Tarsocrural joint effusion is symmetrical and courses circumferentially around the tarsus. Tarsal sheath effusion may be a cosmetic blemish but can be indicative of problems of the DDFT or the sustentaculum tali of the talus (Figure 2.79). Effusion of the calcaneal bursa is often characterized by small pockets of fluid on each side of the SDFT above and below the point of the hock (Figure 2.80). Swelling associated with a capped hock is subcutaneous, whereas effusion within the calcaneal bursa is beneath the SDFT.

The medial aspect of the distal tarsal joint region (distal intertarsal and tarsometatarsal joints) should be closely examined visually and with palpation to detect bone

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Figure 2.76. Atrophy of the shoulder muscles in young horses is often seen with osteochondrosis of the shoulder joint.

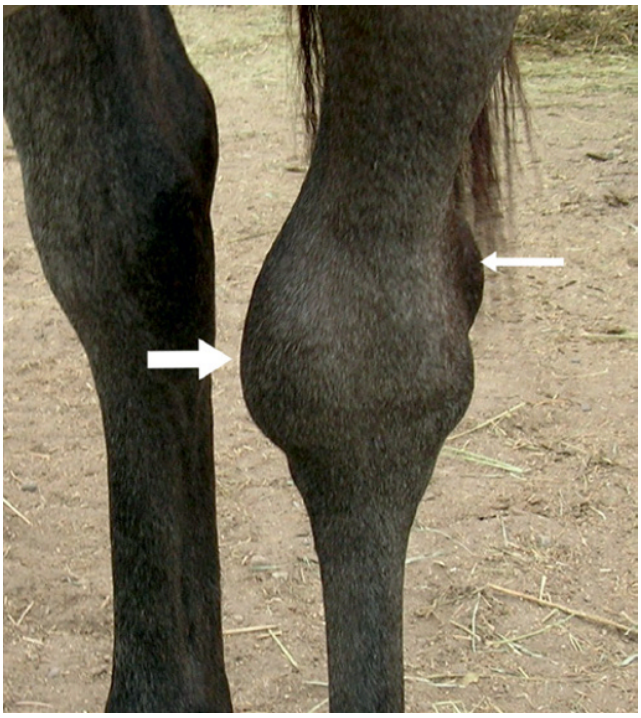


Figure 2.77. Young horse with effusion of the tarsocrural joint that is easily compressible and nonpainful (white arrows).



Figure 2.78. This young horse had a history of previous trauma to the tarsus. Part of the swelling was firm and painful to palpation, but there was also effusion within the tarsocrural joint.



Figure 2.79. Effusion of the tarsal sheath on the medial aspect of the tarsus that was associated with fragmentation of the sustentaculum tali.

proliferation associated with the distal tarsal joints (bone spavin) (Figure 2.81). In the normal horse there is a smooth contour that tapers to the distal tarsal bones as they join the proximal metatarsus. This is easily visualized from the rear and palpated from the side. If this region appears boxy with obvious enlargement, OA of the distal intertarsal and/or the tarsometatarsal joints should be suspected. These medial enlargements have also been referred to as "tarsal shelves" (Figure 2.82). Applying pressure over the medial aspect of the distal tarsus has been referred to as the Churchill pressure test. Using the index and middle fingers, firm pressure is applied to the plantar aspect of the proximal end (head) of the second

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Figure 2.80. Effusion within the calcaneal bursa can often be palpated as fluid outpouchings above and below the retinaculum of the superficial digital flexor tendon (arrows).

metatarsal (splint) bone (Figure 2.83). The test is considered positive if the horse flexes and abducts the limb away from the pressure especially if there is a marked difference in the response between the two tarsi.[2,4]

Enlargement of the long plantar ligament or SDFT (curb), luxation of the SDFT over the calcaneus, and subcutaneous swelling over the calcaneus (capped hock) are closely associated with the plantar aspect of the tuber calcis. This area should be palpated for enlargement of the plantar ligament (Figure 2.84), swelling and pain associated with the superficial flexor tendon (Figure 2.85), displacement of the SDFT (Figure 2.86), and a fluid swelling at the point of the hock referred to as "capped" hock (Figure 2.87). Swelling associated with a capped hock is always subcutaneous and does not involve the calcaneal bursa.

TIBIA

The tibial region can be a difficult region to detect abnormalities both visually and with palpation. Swelling and pain in the caudal tibial region may suggest myositis of the semimembranosus and semitendinosus muscles or gastrocnemius tendonitis. Focal swelling of the distal medial epicondyle of the tibia can be associated with a fracture or medial collateral ligament injury. Severe pain with deep digital palpation of the distal third of the tibia, together with a severe lameness and a positive flexion test, may suggest the possibility of an incomplete tibial fracture. A complete fracture of the tibia is associated with non-weight-bearing lameness, severe swelling, limb deviation, and crepitation on palpation and manipulation.

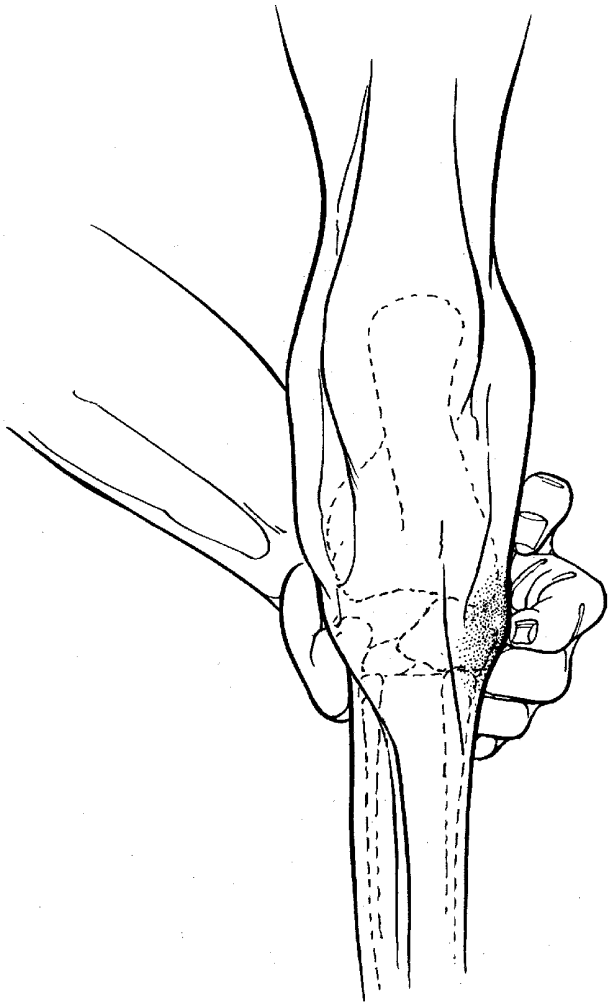


Figure 2.81. Palpation over the distal tarsal joints on the medial aspect of the tarsus, often referred to as the "Churchill test."

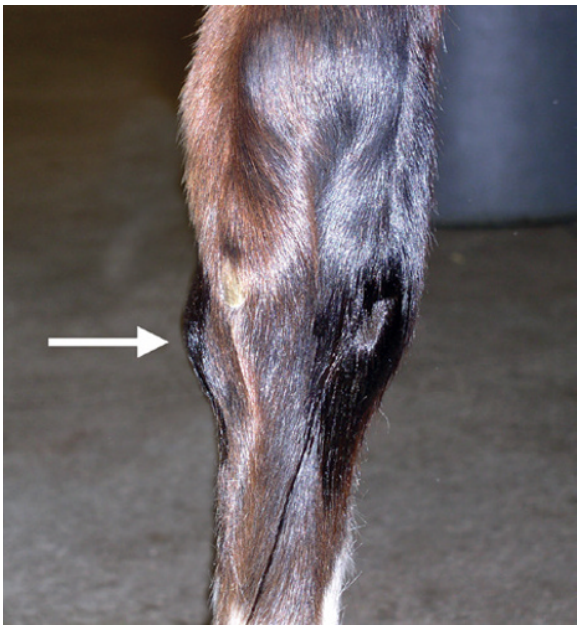


Figure 2.82. Enlargement of the medial aspect of the distal tarsus (arrow) consistent with distal tarsal OA (bone spavin).

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Figure 2.83. Limb and hand positioning to perform the "Churchill" test to detect pain on the medial aspect of the distal tarsal joints.

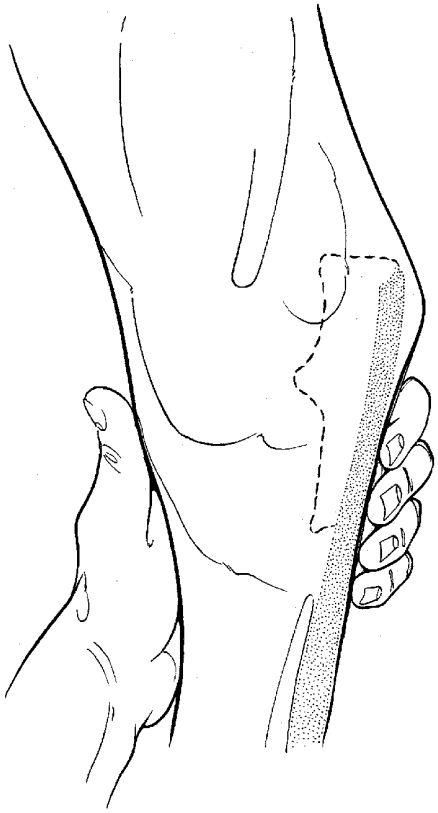


Figure 2.84. Palpation of the long plantar ligament over the plantar aspect of the calcaneus. Swelling in this region is often referred to as a curb but may represent tendonitis of the superficial digital flexor tendon instead. Foals with a "curby" appearance may also have incomplete ossification of the tarsal bones.



Figure 2.85. Tendonitis of the superficial digital flexor tendon in the proximal metatarsal reg that can be misdiagnosed as a curb in some horses.

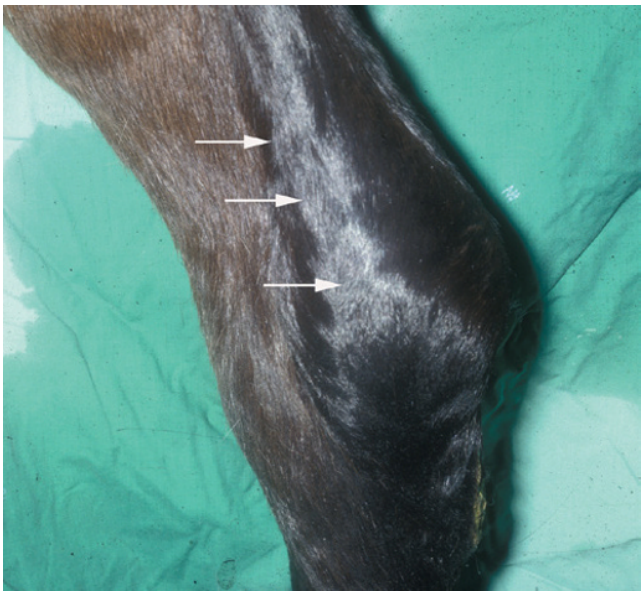


Figure 2.86. Lateral displacement of the superficial digital flexor tendon from the point of the calcaneus (arrows). Effusion of the calcaneal bursa is also usually present in these horses.

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The semimembranosus and semitendinosus muscles should be palpated for any evidence of pain and swelling suggestive of myositis (hamstring pull) and for firm scarring/fibrosis that is often present with fibrotic myopathy (Figure 2.88). Although an uncommon site for problems, the gastrocnemius tendon should be palpated for swelling and pain. An attempt should also be made to extend the hock joint if clinical signs consistent with rupture of the peroneus tertius muscle are present during exercise. With the stifle flexed, the hock can be extended, and a characteristic dimpling of the gastrocnemius tendon occurs (Figure 2.89).



Figure 2.87. Swelling of the subcutaneous bursa at the point of calcaneus, often referred to as a "capped hock."

STIFLE

The stifle should be observed and palpated for swelling and/or atrophy of the associated muscle groups and for fluid distension of the joints. Distension of the femoropatellar joint is best seen from the lateral view (Figure 2.90), and distension of the medial femorotibial (MFT) is best observed from the cranial aspect (Figure 2.91). However, palpation is the preferred method to detect effusion within the stifle joints. The femoropatellar joint pouch is located on the cranial aspect of the stifle beneath the patella ligaments. In general, effusion of the femoropatellar joint makes palpation of the three distal patellar ligaments



Figure 2.88. Horse with fibrotic myopathy of the left hindlimb. There is atrophy of the semitendinosus muscle and firm scar tissue palpable in the caudal tibial region (arrow).



Figure 2.89. Horse with rupture of the peroneus tertius as indicated by the ability to simultaneously extend the hock and flex the stifle.

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Figure 2.90. Effusion can be seen and palpated within the femoropatellar joint cranial to the patella ligaments (arrow).

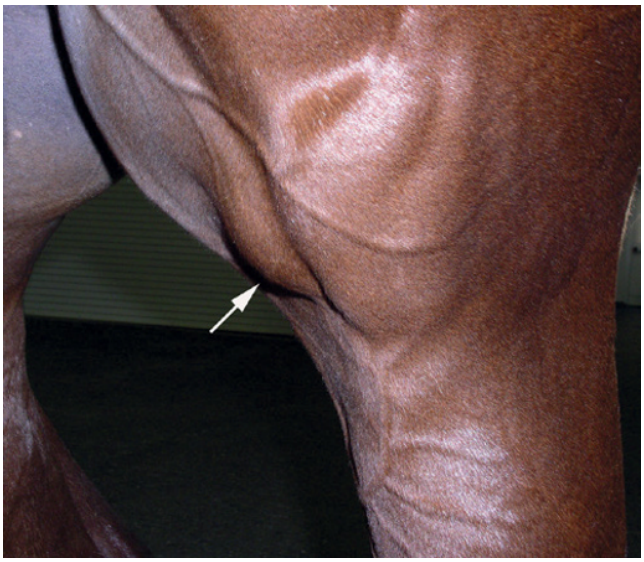


Figure 2.91. Visible and palpable effusion within the medial femorotibial joint is present just behind the medial patella ligament (arrow).

more difficult. These ligaments should be easily palpable across the dorsal aspect of the stifle and are the landmarks to locate the three synovial pouches of the stifle. The patella ligaments should be palpated for evidence of thickening and pain suggestive of desmitis. Fluid distension of the femoropatellar pouch may indicate pathology within the femoropatellar joint or within the MFT joint because they communicate with each other. The MFT joint pouch is located medial to the medial patella ligament directly above the tibial plateau. The lateral femorotibial joint is located lateral to the lateral patella ligament, and effusion within this joint is rarely detected. Palpation of the stifle joints for effusion should always be compared to the opposite limb. In general, abnormalities within the stifle joint are usually accompanied with synovial effusion. Effusion of the femoropatellar and MFT joints can be associated with a variety of clinical problems (see the stifle section in Chapter 5 for details).

The patella and patellar ligaments should be palpated for parapatellar inflammation and pain, crepitation, and displacement. The patellar displacement test involves pushing the patella proximally (upward) and laterally (outward) in an attempt to engage the medial patellar ligament over the medial trochlea. Most horses object to this manipulation, but if the patella is easily displaced upward with apparent locking, it may suggest intermittent upward fixation of the patella. With complete upward fixation, the horse will be unable to flex its stifle or hock and may drag its limb behind in extension (Figure 2.92).

Manipulative tests may also be performed on the stifle to assess the cruciate ligaments and medial collateral ligament.[15] These tests are very subjective and only used in those horses when other clinical signs suggest injury to these structures. The cruciate test can be performed from either the caudal or cranial aspect of the limb. With the caudal approach, the examiner stands behind the horse with his/her arms brought around the limb and the hands clasped together at the proximal end of the tibia (Figure 2.93). The examiner's knees or knee should be in close contact with the plantar aspect of the calcaneus, and the examiner's toe is placed between the bulbs of the heels. This positioning helps to stabilize the limb. In this position the examiner pulls the tibia sharply caudally and releases it to go cranially, feeling for looseness and crepitation that may suggest cruciate ligament damage. With a cranial cruciate ligament rupture, the looseness is felt as a sliding movement in a cranial direction (cranial drawer sign). However, a generalized looseness within the stifle is often the only definitive finding because it is difficult to identify the phase (caudal or cranial) in which the movement occurs.

With the cranial approach, the examiner stands in front of the affected limb with one hand placed on the proximal tibial tuberosity. The other hand is used to pull the tail to that side to force the horse into weight-bearing. The tibia is pushed caudally as quickly and forcibly as possible, which is thought to stress the cranial and caudal cruciate ligaments (Figure 2.94). This may be repeated multiple times, after which the horse may be trotted off and the degree of lameness observed. An increase in lameness may indicate a sprained or ruptured cruciate ligament, but is not specific for this injury.[15]

The medial collateral ligament test is performed by placing the shoulder or outside hand over the lateral aspect of the stifle and abducting the distal limb with the other hand (Figure 2.95). Increased lateral movement of the distal limb indicates complete rupture of the medial collateral ligament. The opposite approach may be performed to test the integrity of the lateral collateral ligament, but injuries to this ligament are uncommon. If a sprain to the ligament is suspected, the limb may be repeatedly abducted several times, after which the horse is trotted off and the degree of lameness assessed.

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Figure 2.92. This young horse had a completely locked patella that caused it to drag its left hindlimb.



Figure 2.93. The caudal cruciate test is performed by grasping the tibia from behind and quickly pulling the tibia caudally. Increased movement of the tibia or a painful response may suggest damage to one of the cruciate ligaments.

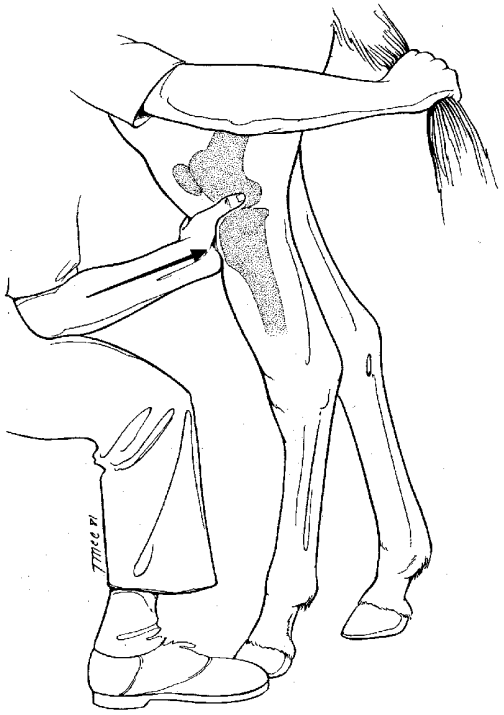


Figure 2.94. Positioning to evaluate for potential problems associated with the cranial cruciate ligament. The examiner places one hand on the proximal tibia and forces it caudally (arrow) to check for increased movement (cranial drawer) of the tibia in relation to the femur. The examiner may also repeatedly push the tibia caudally several times and then trot the horse off to see if this manipulation worsens the lameness.

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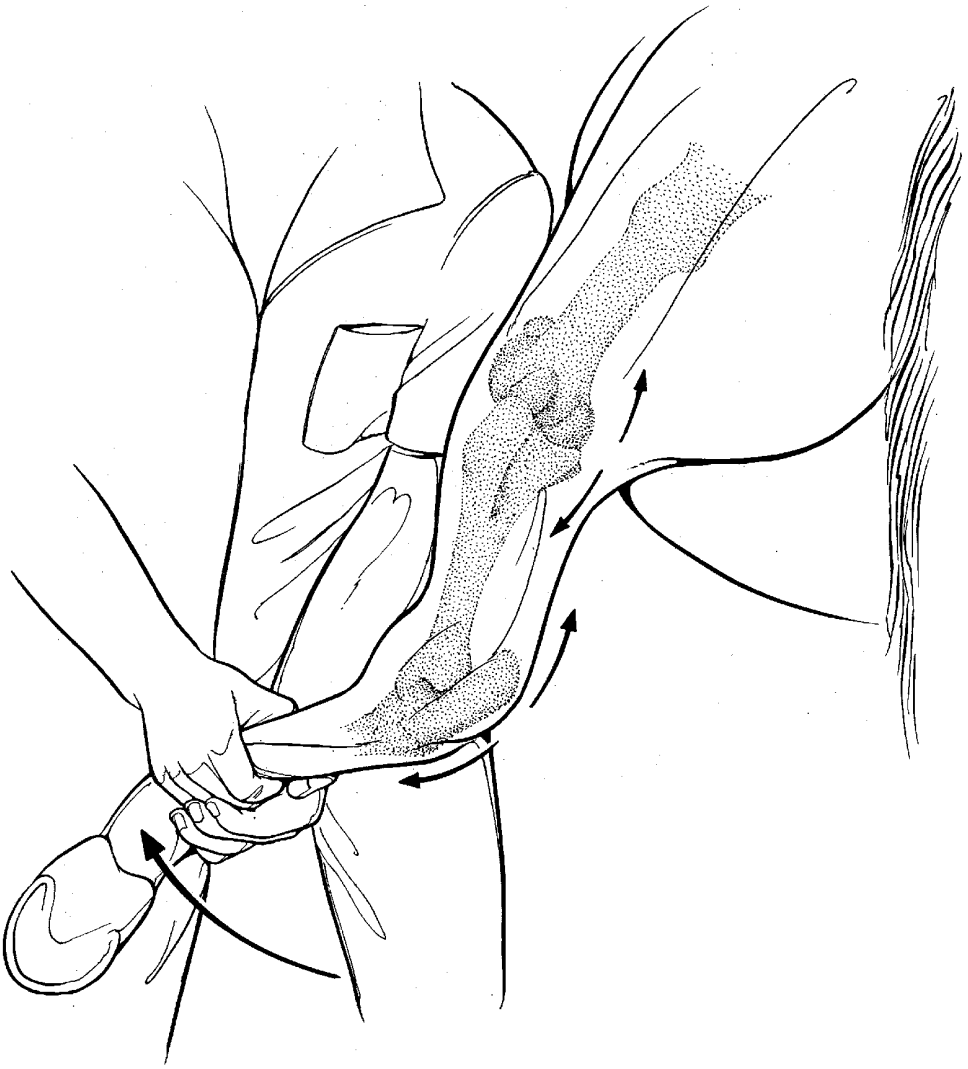


Figure 2.95. Test to stress the medial collateral ligaments of the hock and stifle. Alternatively, one hand can be placed on the medial aspect of the distal tibia to selectively stress the medial collateral ligament of the femorotibial joint. The examiner's shoulder can be placed over the middle of the tibia and both hands on the distal metatarsus to selectively stress the medial aspect of the hock.

FEMUR

The muscles surrounding the femur are primarily examined for swelling and/or atrophy. The femoral artery should be palpated for the quality of pulsations on the medial side of the thigh in the groove between the sartorius muscle cranially and the pectineus muscle caudally. If the pulse is weak or nonexistent, thrombosis of the iliac artery may be contributing to the lameness. Pressure can be applied to the greater trochanter, and if painful, middle gluteal muscle strain or trochanteric bursitis (whirlbone disease) should be suspected[4]. Complete fractures of the femur usually result in non-weight-bearing lameness with severe swelling and limb shortening due to overriding of the fracture (Figure 2.96). Femoral neck fractures are more difficult to diagnose because they typically cause less swelling and lameness than diaphyseal femoral fractures. With time the swelling may migrate distally on the medial side of the thigh, giving the impression that the distal femoral region is involved.

HIP

The hip should be examined for asymmetry, swelling, and atrophy of associated muscle groups. With hip problems, swelling over the coxofemoral joint may be visually apparent, and pain can often be elicited with deep palpation directly over the joint using the palm of the hand. At a walk, a stifle-out, hock-in, toe-out gait (external rotation) is frequently observed, with an apparent shortening of the limb length (Figure 2.97). From the side, the affected limb may appear to be straighter than the contralateral limb. With the metatarsus held in hand, the coxofemoral joint can be manipulated into extension, flexion, and abduction to check for evidence of pain and crepitation. Additionally, the hip can be intermittently flexed and auscultated with a stethoscope at the same time to identify crepitation. Limb abduction is often painful to horses with hip conditions, and repeated limb abduction will often exacerbate the lameness.

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Figure 2.96. Swelling of the femoral region associated with a fracture of the distal femur in a yearling Quarter horse.



Figure 2.97. Typical toe-out, hock-in stance that often accompanies problems within the hip and pelvic region.

PELVIS

Visual identification of asymmetry of the bones and musculature of the pelvis is an important aspect of examination of the pelvis. This includes the tuber coxae, the tuber ischium, the tuber sacrale, and the gluteal muscles on each side. Asymmetry of the bony pelvis often suggests a pelvic fracture, subluxation of the sacroiliac region, or fracture of the specific bony prominence. Gluteal muscle atrophy often accompanies chronic pelvic fractures but can be seen with any chronic hindlimb lameness (Figure 2.98). Crepitus associated with pelvic fractures can usually be elicited by swaying the horse from side to side or can sometimes be detected on rectal examination.

BACK

Visual assessment of the horse's back includes observing the muscle contour from the side and axial alignment from the rear (Figure 2.99). The dorsal spinous processes should be palpated for axial alignment, protrusion or depression, swelling, and interspinous distance (Figure 2.100). Any muscle swelling, atrophy, or asymmetry in the epaxial musculature should also be noted.

Palpation is usually best performed with firm fingertip pressure using both hands simultaneously (Figure 2.101). Alternatively, the palm of the hand can be used to apply downward pressure to the epaxial muscles (Video 2.3).[2] Palpation of the epaxial muscles lateral to the dorsal spinous processes along the entire length of the back should be performed. Many horses may respond to downward pressure in the lumbar region by ventroflexing their backs, but this response often fatigues and withdrawal is less prominent. In horses that have clinically significant back pain, ventroflexion of the back is often severe, and any increase in finger- or palm-applied pressure greatly increases this response. The horse attempts to "drop down" to get away from hand pressure. Palpation may also cause the horse to vocalize, swish its tail, or actually kick out behind. Back palpation is somewhat subjective, and, therefore, the assessment requires clinical experience. In some cases, tightening of the longissimus dorsi muscle may be felt with palpation rather than a painful withdrawal response. This usually signifies that the horse is attempting to fix the vertebral column because ventroflexion is painful.[15]

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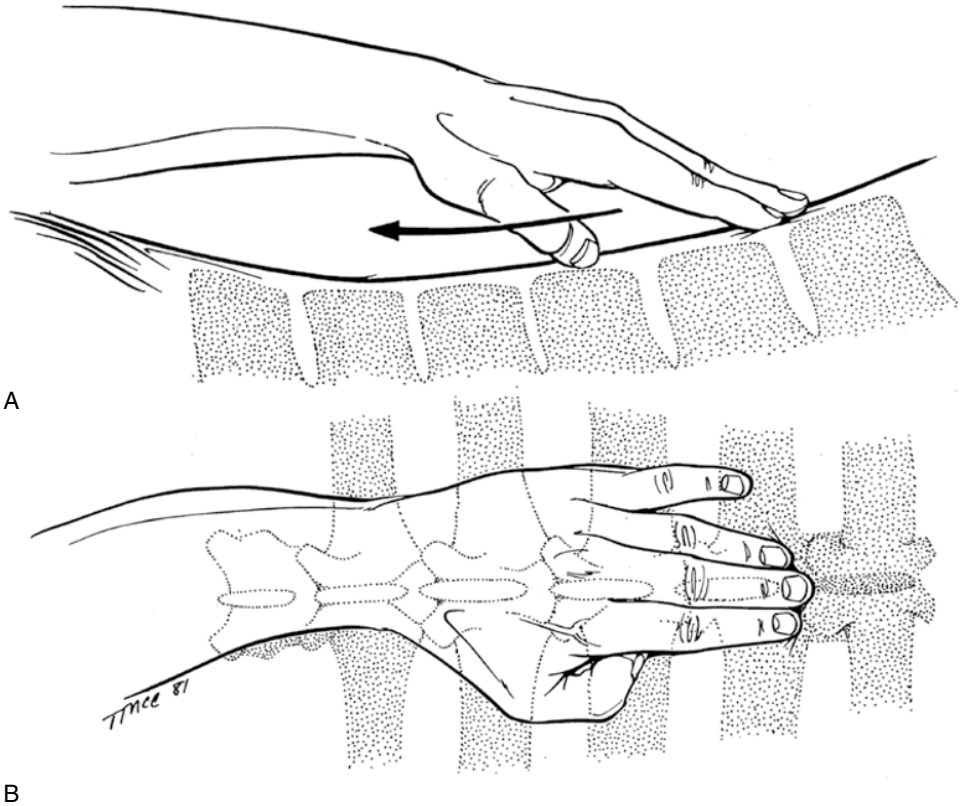
The examiner may also wish to assess the horse's willingness to ventroflex, dorsiflex, and lateroflex its thoracic and lumbar vertebrae.[15] Assessment of the horse's ability to ventroflex the back is obtained by exerting downward pressure to the muscles in the thoracolumbar region. A blunted instrument (needle cap) run over the croup usually causes dorsiflexion or arching of the back. Lateral flexion can usually be assessed by firmly stroking either side of the thoracolumbar region with a blunted instrument. Reluctance to flex the back in any of these directions may suggest muscle tightening and back rigidity within the thoracolumbar region (see Chapter 6 for further information on examination of the back and axial skeleton).



Figure 2.98. Severe atrophy of the left gluteal muscles secondary to a pelvic fracture. Disuse gluteal atrophy tends to occur more quickly and more profoundly with pelvic fractures than other lameness problems more distal in the limb.



Figure 2.99. Kyphosis of the thoracolumbar region was present in this foal and was due to a developmental malformation of the vertebral column in this location.



A

B

Figure 2.100. (A) Palpation of the summits of the dorsal spinous processes to identify depressions, protrusions, swelling, and pain. (B) Palpation of the axial alignment of the dorsal spinous processes.

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NECK

The neck should be examined for contour from the side and axial alignment from the front and rear. Excessive ventral arching of the neck in the mid-cervical region may be seen in some cases of cervical vertebral malformation. A straight (extended) poll can be seen with atlanto-occipital and atlanto-axial malformations. Splinting and spastic contraction of the neck muscles with or without signs of spinal ataxia is often consistent with vertebral fracture. Generally these horses are very painful.

The transverse processes should be palpated for alignment and symmetry and to detect evidence of atrophy. Muscle atrophy is most often observed in the caudal neck region dorsal to the cervical vertebrae and may be symmetric or asymmetric. Swelling of the neck either lateral or ventral is generally a sign of trauma or infection.

The neck should be flexed laterally and ventrally and extended to assess flexibility, range of motion, and pain. Lateral flexing can be done by pulling the horse's head by the halter to one side and then to the other. Alternatively, lateral neck flexion can be encouraged by holding a "treat" at the horse's shoulder. Most horses should be able to flex their neck laterally enough that the muzzle almost contacts the craniolateral shoulder region. Ventroflexion is assessed by feeding the horse from the ground level, and extension is evaluated by elevating the head and neck. Resistance to neck movement in any direction is usually due to pain and can be from many potential causes.

FLEXION TESTS/MANIPULATION

Most flexion tests, regardless of the location, are usually performed for 30–60 seconds and are a subjective method to further isolate the site of the lameness (Videos 2.5 and 2.6). In one recent study, flexion duration for 5 seconds had similar responses to tests performed for 60-second questioning whether the duration of flexion is that important.[1] In addition, the response to flexion tests must be interpreted in light of clinical findings because many otherwise normal horses may demonstrate positive responses.[11,18] These false-positive responses are thought to be directly related to the force applied to the limb. In one study only 20 of 50 horses responded to a "normal" distal limb flexion, while 49 of 50 horses responded to a "firm" distal limb flexion.[11] Another study revealed that more than 60% of 100 sound horses had a positive response to distal limb flexion and that the positive outcome increased significantly with age.[3] Both of these studies question the validity of distal limb flexion tests to predict future joint-related problems.[3,11] In addition, false-positive responses to flexion seem to occur more commonly in horses in active work than in horses that have been rested or turned out to pasture.[12] In general, there are more false-positive results to flexion at any location than false-negative results, but both can occur. False-positive responses are most common in front fetlock flexion tests.

Because both the amount of force and the duration for which it is applied may affect the response to flexion, the procedure should be standardized as much as possible to minimize variability.[7,8,18] For instance, the same person should flex the right and left limbs at any location for the same period of time so they can be accurately compared. Different people may have slight differences in the way they hold the limb or apply pressure to the limb, which can alter the responses. Despite this potential for variability, the flexion techniques performed by experienced veterinarians are usually sufficient to objectively assess responses to flexion.[7] Two relatively recent studies using inertial sensor-based systems have documented that a positive response to flexion results in significant changes to the objective measurements of pelvic movement.[9,14] However, the variation in measured symmetry following flexion within and between horses showed that the individual response to flexion may be highly variable.[14]

Passive flexion usually refers to manipulation of a joint during routine palpation of the horse, and pain detected with passive flexion often predicts a significant response to a subsequent flexion test. However, flexion tests can also be used to subjectively assess the severity of damage within an affected joint(s). In general, the severity of damage is often proportional to the severity of the response to the flexion test. For instance, horses with severe responses to carpal, fetlock, or stifle flexion typically have significant intra-articular or extra-articular pathology. However, flexion tests are not specific for the joint because it is nearly impossible to flex a single joint without affecting other nearby joints and soft tissues. Flexion of a joint not only increases the intra-articular and subchondral bone intraosseous pressures within the joint but also compresses and stretches the joint capsule and surrounding soft tissues.[12,16] The numerous other "structures" that are being manipulated with any flexion test should always be considered when interpreting the clinical significance of flexion tests.

The responses to flexion should be graded in some manner and included in the records. This is most important when reevaluating the lameness to more accurately determine whether improvement is being made. The author uses a grading scale of negative, mild response, moderate response, and severe response to assess the flexion tests. Alternatively, a plus-minus system may be used with "–" being no response and 1+ equating to mild, 2+ to moderate, and 3+ to severe. Regardless of the system used, the responses to flexion are a very important aspect of the lameness examination and should be recorded (Video 2.5). Additionally, changes in lameness in the limb not being flexed (weight-bearing limb) should also be recorded because this is often an important clinical finding. This contralateral response to flexion tends to occur most commonly in horses with bilateral hock and carpal problems.[12]

DISTAL LIMB/PHALANGEAL/FETLOCK FLEXION

Attempts can be made to isolate the fetlock joint from the pastern and coffin joints during flexion of the distal limb (Video 2.5). However, it is nearly impossible to only flex the fetlock or only flex the pastern or coffin joints. Flexion of the fetlock joint can be performed by placing one hand on the dorsal MC/MT and pulling up on the pastern with the opposite hand (Figure 2.59). Flexion of just the phalangeal joints is performed by maintaining fetlock extension by placing one hand on

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the fetlock while flexing the phalanges by pulling up on the toe with the opposite hand (Figure 2.102). All three joints can be flexed together (distal limb flexion) by pulling up on the toe and phalanges with both hands while facing toward the back of the horse (Figure 2.103). In general, it is nearly impossible to isolate the phalanges from the fetlock region in the hindlimb; therefore, most distal limb flexion tests performed in the hindlimb include all three joints (distal limb flexion test; Figure 2.103).

Regardless of how the flexion test is performed, the fetlock/phalanges are held in position for 30 seconds, after which the horse is trotted off and lameness is observed. Differences in the severity of the responses may be used to suggest whether there may be a problem in the fetlock vs. the pastern or coffin joints. A painful response to phalangeal flexion and a negative response to fetlock flexion may suggest a problem in the coffin or pastern joint or any soft tissue structure in the area. A negative response to phalangeal flexion together with positive responses to both the fetlock and distal limb flexion suggests a fetlock problem. Any positive signs should be checked with the opposite limb because a marked asymmetry in responses to distal limb flexion tests is an important clinical finding and further indicates



Figure 2.102. Hand and limb positioning to perform flexion of the phalangeal joints without flexing the fetlock.

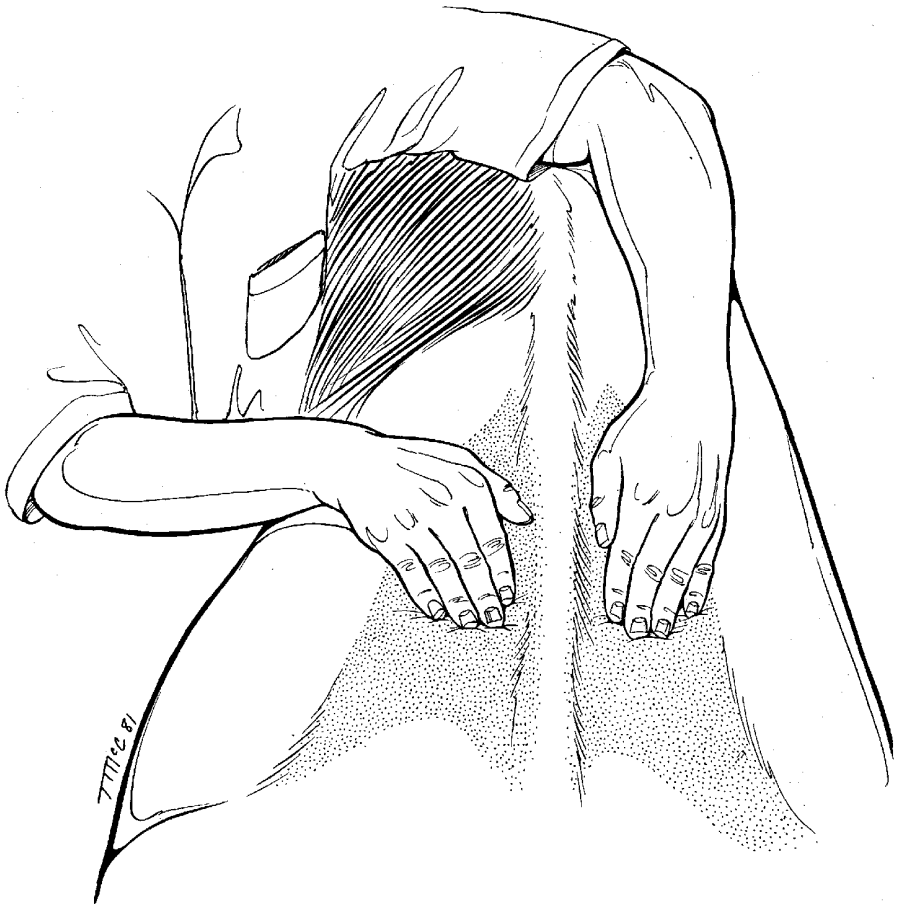


Figure 2.101. Firm pressure applied to the back musculature from the withers to the tuber sacrale to identify a painful response. The fingers should be held flat to prevent "digging in" with the fingertips. Firm pressure can also be applied using the palms of your hands.

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Figure 2.103. Hand and limb positioning to perform distal limb flexion (phalangeal and fetlock joints) of the hindlimb.

a potential problem in the area. However, false-positive fetlock flexion tests do occur, especially in horses in work, and many horses may show a positive response if a large amount of force is applied to the fetlock/distal limb. One study in normal horses evaluating the force applied for the fetlock and phalangeal flexion test by different examiners found that the force varied considerably and was frequently too high.[18]

CARPAL FLEXION

The carpal flexion test is very useful to help isolate a problem to the carpus. A negative response does not rule out a problem in the carpus (many horses with osteochondral fragmentation are not positive to carpal flexion), but a positive response is highly suggestive of a carpal problem (few false-positive responses). This is in contrast to the fetlock, where false-positive responses are much more common. Carpal flexion is performed by grasping the metacarpus with the outside hand while facing the horse, pulling up on the distal limb (Figure 2.71). The foot should be able to contact the caudal aspect of the olecranon in normal horses. The carpus is held in this position for 30–60 seconds, after which the horse is jogged away and observed for increased lameness.

ELBOW FLEXION

It is difficult to completely separate the elbow from the shoulder when performing upper limb flexion tests in the forelimb. This is analogous to the tarsus and stifle in the hindlimb because flexion of one area often affects the other. However, flexion of the elbow can be performed by lifting the antebrachium (forearm) so that it is parallel to the ground but not pulled forward (Figure 2.75). This flexes the elbow and causes the carpus and distal limb to hang freely. The limb is held in this position for 30–60 seconds and the horse is jogged off. Elbow flexion is usually not part of a routine lameness evaluation and is performed when an abnormality in the elbow region is found on physical examination.

SHOULDER/UPPER FORELIMB FLEXION

Manipulation of the upper forelimb can be performed either by pulling the limb cranially and upward or by pulling the limb caudally. The cranial approach is similar to flexing the elbow; only the limb is pulled forward and will flex the elbow and extend the shoulder. This is usually performed by standing in front of the limb, grasping the antebrachium, and lifting the limb up and forward (Figure 2.104). This exacerbates lameness problems in the caudal aspect of the elbow and the cranial aspect of the shoulder. The more the limb is elevated, the more pressure is applied to the cranial aspect of the shoulder. The position is maintained for 30–60 seconds (or as long as the horse tolerates it), and the horse is jogged off. Horses with supraglenoid tubercle fractures of the scapula, and horses with bicipital bursitis often respond to this type of shoulder manipulation.

The caudal approach to flex the shoulder joint can be performed by placing one hand on the olecranon process and pulling the limb caudally. Alternatively, the cranial antebrachium may be grasped and pulled caudally together with the distal limb instead of applying pressure to the olecranon (Figure 2.105). The position is maintained for 30–60 seconds (or as long as the horse tolerates it), after which the horse is jogged off.

TARSAL/HOCK FLEXION

The tarsal flexion test or spavin test is somewhat of a misnomer because it flexes the fetlock, stifle, and hip in addition to the hock (Video 2.6). A positive response to hock flexion is not synonymous with a tarsal problem but can be used together with other physical examination findings to suggest a problem in the tarsus. Hock flexion is performed by placing the outside hand when facing the rear of the horse on the plantar surface of the distal third of the metatarsus and elevating the limb to flex the hock (Figure 2.106). The opposite hand is then placed around the metatarsus, and the limb is held with both hands while facing the back of the horse. The grip



Figure 2.104. Upper limb flexion test in which the limb is pulled cranially and upward to "stress" the shoulder region.

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Figure 2.105. Flexion of the upper forelimb can be performed by grasping the antebrachium and foot and pulling the limb caudally.

should avoid the sesamoid bones and be loose enough to avoid excessive pressure to the flexor tendons and suspensory ligament. The fetlock and phalanges should not be forcibly flexed.

Alternatively, the tip of the toe can be held so the pastern and fetlock joints are extended and the hock is flexed (Figure 2.107). It may also be beneficial to gradually flex the tarsus to its fullest extent over a 15-second period to avoid resentment by the horse. In some cases in which the horse tends to lean away from the examiner, it may be helpful to place the horse adjacent to a solid support (i.e. wall or fence) or have an assistant provide counterbalance to the tuber coxae of the opposite hip. Once the tarsus is in full flexion, it is held in this position for 30–60 seconds, the limb is released gradually, and the horse is trotted off.

A positive hock flexion test is indicated by an increase in lameness of the flexed limb. However, increased lameness of the opposite limb (standing limb) is thought to occur with some upper limb lameness problems (sacroiliac problems). The first few steps the horse takes after this test are often the most important. If there is any question regarding the validity of the flexion test, it should be repeated. Two of the most common errors in performing this test are not obtaining full tarsal flexion and spooking the horse so that it balks at the onset of

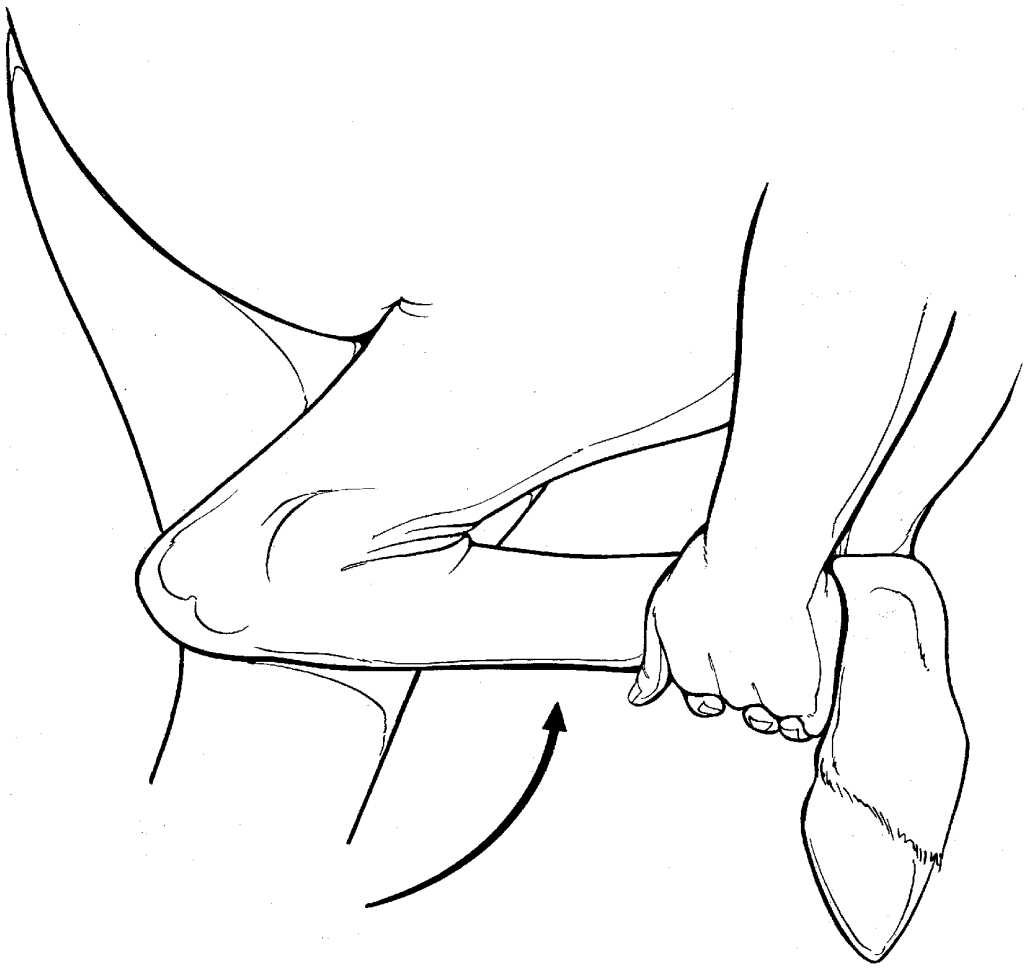


Figure 2.106. Hock or tarsal flexion (spavin) test. The hindlimb is flexed so that the metatarsus is approximately parallel to the ground. This test is not specific for the tarsus because it flexes both the stifle and the fetlock to some degree.

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Figure 2.107. Positioning to perform hock or tarsal flexion of the hindlimb where the limb is held by the hoof.



Figure 2.108. Flexion of the stifle is performed by pulling the hindlimb caudally and lifting up on the distal tibia.

the test rather than jogging off at a smooth pace. The response to tarsal flexion should nearly always be compared to the opposite limb because an asymmetrical response is often an important clinical finding.

STIFLE FLEXION

The stifle flexion test is often used in an attempt to separate stifle pain from tarsal pain in horses that respond to a tarsal flexion test. In most cases the stifle flexion test will flex the hock less than the tarsal flexion test will flex the stifle. In many cases it can be more specific for stifle problems, but this is debatable. It is performed by grasping the distal tibia and pulling the limb backward and upward until maximal stifle flexion is achieved (Figure 2.108). It is best to face toward the back of the horse with the limb in front of you when performing this test (Video 2.6). The limb is held in this position for 60 seconds and the horse is trotted off. Some clinicians prefer to perform the stifle flexion test before the hock flexion test, while others do the opposite. A positive tarsal flexion together with a more positive stifle flexion may suggest that the lameness is due to a stifle problem and vice versa.

FULL-LIMB FORELIMB AND HINDLIMB FLEXION

Full-limb forelimb and hindlimb flexion tests can be used as quick screening tests to determine whether more isolated flexion tests may be necessary. If there is no response to flexing all of the joints at one time, the potential for getting positive responses to individual flexion tests is thought to be unlikely. Full-limb forelimb flexion is performed by grasping the foot and lifting the leg to flex the fetlock, carpus, and elbow. The opposite hand is placed on the metacarpus, and the limb elevated and pulled forward to extend the shoulder (Figure 2.109). Full-limb hindlimb flexion is performed by grasping the foot and flexing the fetlock, hock, and stifle simultaneously. The hindlimb is pulled out behind the horse to help flex the stifle (Figure 2.110). The limb is usually held in position for 30–60 seconds. A negative response is thought to suggest that individual flexion responses will also be negative, but this has not been determined definitively for either the forelimb or hindlimb.

NAVICULAR WEDGE TEST

The navicular wedge test can be performed in two different ways.[2] A wedge (usually a block of wood) may be placed beneath the frog of the affected foot while the opposite limb is held up. It is thought to apply direct pressure to the frog region, similar to hoof testers, and is usually performed for 30–60 seconds, after which the horse is trotted off.[2] Alternatively, the toe is forced into an

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Figure 2.109. Positioning to perform a full-limb flexion of the forelimb. All of the joints in the limb are flexed simultaneously.



Figure 2.110. Positioning to perform a full-limb flexion of the hindlimb.

elevated position in relation to the heel by placing a wooden wedge under the toe (Figure 2.111). This serves to increase the tension on the DDFT and increase pressure over the navicular bone. The wedge may also be applied to the medial or lateral aspects of the foot to manipulate the soft tissues of the digit.[12] The opposite limb is elevated for 30–60 seconds, and the horse is trotted off. Although these tests are often used for horses with suspected navicular syndrome, one clinical study found that they were not helpful in differentiating navicular region pain from other causes of palmar heel pain.[13] Any cause of heel/foot pain may be exacerbated with these tests.

DIRECT OR LOCAL PRESSURE PLUS MOVEMENT

The premise for applying direct pressure to a specific site and watching the horse trot is to confirm the significance of palpation findings. A positive response to static palpation does not necessarily indicate the site of the problem. However, increasing the baseline lameness by deep palpation of a suspicious area or anatomic structure will often confirm the potential of a problem in the area. Direct pressure is usually applied manually, but hoof testers can be used to apply pressure to the sole of the foot. The limb is usually elevated, the site is compressed for 15–30 seconds, and the horse is trotted off. Exacerbation of the lameness by one or more grades is considered a positive response.[2] The direct pressure test is most commonly performed over swellings of the splint bones, dorsal metacarpus, flexor tendons, suspensory body and branches, and medial aspect of the tarsus. It also may be used to assess pain in the proximal suspensory region of both the forelimb and hindlimb and several areas of the axial skeleton.

Rectal Examination

A rectal examination may be indicated in some horses with upper hindlimb lameness. It is most commonly performed in horses with suspected pelvic fractures or problems in the sacral region. Information also may be obtained rectally in horses with iliopsoas myositis, fractured vertebrae, or thrombosis of the iliac arteries.[15] Rectal examination is usually performed with the horse standing still, but it may be beneficial to walk the horse while performing the rectal exam (so-called walking rectal). The examination is often performed in a cranial-to-caudal direction. Pressure should be applied to the iliopsoas muscle located cranial to the pelvic brim. The aorta and iliac arteries should be checked for normal pulsation. The symmetry of the pelvis is palpated by comparing one side to the other (Figure 2.112). With displaced ilial fractures, an obvious asymmetry may be present, and rocking the horse from side to side may reveal crepitus or movement of the bones. The ventral aspect of the sacral vertebral bodies should be checked for alignment and any depression or protrusion into the pelvic canal, which may indicate fracture or subluxation. Rectal ultrasound of the pelvis may be performed at the same time as palpation to further document abnormalities.

Neurological Examination

Any horse with a suspected neurological problem should have a complete neurological examination performed (see Chapter 12 for this description). However, determining the presence of hindlimb weakness and correct limb placement can be performed quickly and may

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A B C D

Figure 2.111. A (10″ × 10″) 15° wooden wedge block with a nonslip surface can be used for distal limb manipulation during lameness evaluation. The foot is typically placed in four different orientations; toe wedge (A), heel wedge (B), lateral wedge (C), and medial wedge (D). Depending on the orientation of the block, different tissues within the hoof are placed under compression or tension. Source: Courtesy of Dr. Randy Eggleston.

help suggest neurological problems. Hindlimb weakness can be assessed by either pushing the horse from one side to the other or more commonly be pulling its tail to each side. This has been referred to as the sway test or tail pull test.[15] Normal horses resist pulling the tail, while many ataxic horses appear relatively weak and do very little to resist being pulled from one side to the other. However, this test is very subjective, and many young Warmblood horses with underdeveloped hindlimb musculature often appear to be very weak but are not neurological.

Limb placement can be assessed by walking the horse over a curb, circling, positioning the limb across midline while standing, and trotting in a serpentine pattern. Repeated stumbling, hitting the curb, circumduction of the hindlimbs, and a general sense of not knowing where the hindfeet are suggest a neurologic problem. Some horses that have bilateral hindlimb ataxia may also appear very "bouncy" when trotted and when stopped suddenly from a trot may stumble in the hindlimbs. A variety of neurologic conditions may be confused with lameness

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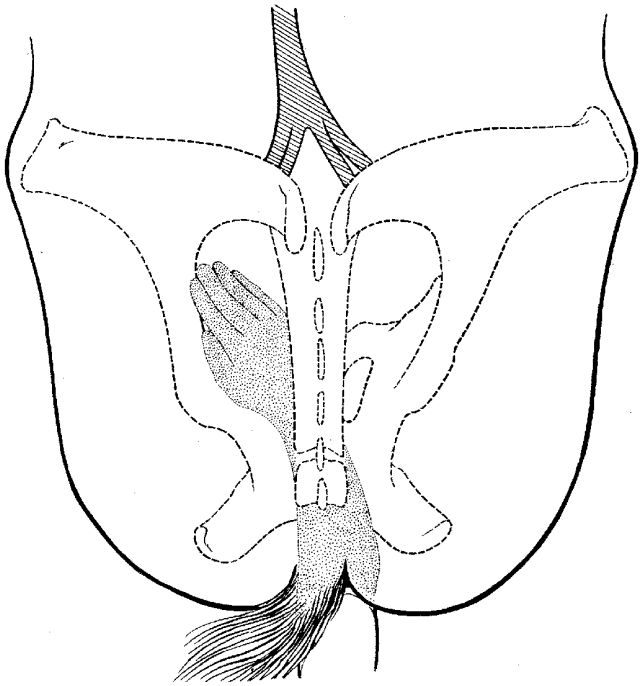


Figure 2.112. Dorsal view of the rectal examination showing evaluation of the symmetry of the pelvis. One side of the pelvis should be compared with the other. The pelvis can also be palpated for crepitation while the horse is swayed from the side to side while standing or during movement.

problems and at times differentiating between lameness and neurologic disease can be very difficult (see Chapter 12 for more information on neurologic examination).

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SUBJECTIVE ASSESSMENT OF LAMENESS

Kevin G. Keegan

The word "lame" stems from the Proto-Indo-European language root *lem*, meaning literally "broken," with comparative usage in Old English (*lame*), Old Norse (*lami*), and Proto-Germanic (*lahm*).[31] The word *lame* is an adjective, as in a *lame* limb. In modern veterinary usage, we use a derivative of the word as the noun *lameness*, the state of being *lame*. *Lameness* is a clinical sign not a disease.[32,48] A synonym is *limp*, also used as a verb, as in "the horse *limps*." A horse with lameness is a limping horse.

Most lameness is caused by pain during weight-bearing, with shifting of load away from the source limb onto the other three, non- or less afflicted, limbs. In human medicine lameness due to pain on weight-bearing is called *antalgic* ("against pain") gait and is considered the medical term for "limping." In the bipedal human the main feature of antalgic gait is decreased duration of stance time of the affected versus unaffected limb.[36] But this is frequently not true in the quadruped equine unless the lameness is severe, and, instead, it is a decrease in peak vertical ground reaction force on the affected limb that is the primary effect of weight-bearing pain.[10,54,55] But also decreased vertical ground reaction force can be a sign of a comparative unilateral weakness, neurologic dysfunction, anatomical asymmetry, and, to a limited degree and uncommonly, even primary functional asymmetry (handedness, laterality), a dominance of one side of the brain in controlling particular activities. Horses without pain, neurologic abnormality, or weakness in the limbs or torso usually move symmetrically and are not lame.

Detection of the clinical sign of lameness by observing the horse move in most situations is a simple concept. It is the recognition of asymmetric gait. However, it is more difficult in practice because of inherent limitations in spatial and temporal resolution of unaided human eyesight. In contrast, determination of the cause and importance of lameness is frequently both difficult and complex, requiring detailed knowledge of anatomy; understanding of how movement of the head, torso, and limbs are affected by limb loading and unloading; a complete and systematic palpation and manipulation of the entire limb and torso; knowledge of breed- and use-specific diseases that cause lameness; localization of the focus of pain with local anesthesia; and quality imaging. Obtaining relevant history, visual examination of the horse at rest, and palpation and manipulation of the limbs and torso are covered in earlier chapters. Subjective evaluation of the horse during movement to detect and assess lameness is the purpose of this chapter.

A horse without lameness moves symmetrically, with equal amplitude of movement in the head, torso, and limbs during the right and left halves of the stride. A horse with lameness due to pain in a limb moves asymmetrically. This does not mean that all horses with musculoskeletal or neuromuscular abnormality move asymmetrically or show lameness. It is possible for horses with bilateral pain or disease to move symmetrically, at least in some strides, especially when moving in a straight line. But one limb is usually slightly worse, and an average asymmetry of movement, or lameness, over all strides is retained. Axial skeletal disease in particular may cause relatively equal amounts of pain during both steps in a stride so that lameness may not be displayed. However, most horses with musculoskeletal disease will display lameness, and many horses that display lameness have musculoskeletal disease.

The asymmetric movement in horses with musculoskeletal or neurologic abnormality causes a decrease in resultant ground reaction forces, because most pain or dysfunction occurs during weight-bearing. Some abnormalities may also cause pain in the swing (non-weight-bearing) phase of the stride, but there is objective evidence that the abnormal movement due to pain or dysfunction in the swing phase of the stride affects kinematics (and likely ground reaction forces) also in the stance phase of the stride.[27] In other words, pure swinging limb lameness conditions without weight-bearing signs have not been shown to exist and if they do exist are not common.[9] In addition, asymmetric limb movement without asymmetry in weight-bearing, though technically defined as lameness, is more likely simply a sign of conformational difference or handedness and not clinically significant as a sign of disease.

Movement of the midline of the horse's body, more precisely movement of the head and pelvis, in the same sagittal plane of the body's center of mass, is directly related to vertical ground reaction forces on the limbs during weight-bearing.[13] Movements of the abaxial appendages are more naturally variable (right vs. left and stride by stride) and more affected by idiosyncratic factors, like conformation and handedness (predominance or preference of one side of the body over the other). Higher variability and more complex association to multiple causative factors decrease the usefulness of specific limb movements to reliably signal painful weight-bearing lameness.[1,44,45] For these reasons, observation of how the midline (head and torso) moves is more sensitive and specific for weight-bearing lameness detection than evaluation of limb movement. Exceptions to this generalization include dorsiflexion of the fetlock during weight-bearing and amplitude of hindlimb protraction, both of which, when decreased, have been shown to be reliable indicators of abnormality.[12,19,30] These are discussed later in this section.

However, there are a few musculoskeletal/neurological abnormalities that are most easily recognized as specific "odd" limb movements and, if seen unilaterally, satisfy the definition of lameness. Fibrotic myopathy of the stifle flexor muscles, stringhalt, momentary or intermittent upward fixation of the patella, gastrocnemius muscle injury, peroneus tertius rupture, and "shivers" can be recognized merely by observing limb movement

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during the swing phase of the stride or when not weight bearing. Although these are thought to be pure "swinging limb" lameness conditions, with pain or dysfunction isolated to the swing phase of the stride, and therefore exceptional to biomechanical descriptions of weight-bearing lameness, the author has measured horses with these conditions and found that decreases in vertical movement of the torso are likewise seen. Thus, in these few exceptional conditions, the descriptions of asymmetric vertical movement of the head and pelvis may not adequately explain the quantity of dysfunction, but they remain valid for describing side of lameness. Detection of these lameness conditions by observing characteristic abnormal limb movement is discussed later.

EVALUATION OF THE HORSE FOR LAMENESS AT THE WALK

Because limb and torso movements are slower when walking, it is the easiest gait in which to observe lameness. However, for disease of mild severity, vertical ground reaction force may not reach a minimum threshold for pain, but the horse walks without visible asymmetric movement and is not subjectively lame. It is thought that sometimes, and for some conditions, lameness is more visible at the walk than at other gaits, but for weight-bearing lameness, this is most likely because movement speed is within the temporal resolution of unaided human sight. Weight-bearing lameness at the trot measures greater than lameness at the walk.[4]

The trajectory of vertical head movement during the walk of a horse with unilateral forelimb lameness is asymmetric (Figure 2.113). At the walk, as in other gaits at slow speeds, the front limbs acts almost exclusively like a strut or crutch, so head movement trajectory in a horse with forelimb lameness is usually shaped like depicted below, being highest from the ground right before or during the first part of stance of the lame or lamer forelimb and lowest during stance of the not lame or less lame forelimb. This corresponds only vaguely to the concept of "head nod" understood by many veterinarians and taught to veterinary students of the horse's head moving "down on the sound" limb and "up on the lame" limb. This explanation of "head nod," however, is actually not precisely true, and a more accurate description of "head nod" during the trot will be developed later in this section.

Vertical movement of the pelvis at the walk does not follow this downward and then upward trajectory. Instead it is upward and then downward with the low positions at the beginning of, and the high positions, during stance. Also with two and three limbs alternately bearing weight at the same time, vertical pelvic movement (VPM) at the walk may not be a simple biphasic pattern. Total vertical pelvis movement is also less than that of the head, and the amount of asymmetry between right and left hindlimb strides is more difficult to see. These characteristics make detecting hindlimb lameness using VPM at the walk more difficult than using head movement to detect forelimb lameness at the walk. Alterations in hindlimb movement like decreased hindlimb protraction, prominent circumduction, or excessive rotation are more obvious than asymmetry of VPM.

Other forelimb and hindlimb movement parameters, besides vertical head and pelvic movement, which indicate lameness, may also be seen at the walk, but these will also be seen at the trot.

Evaluation for lameness at the walk can be enhanced by turning the horse in a tight circle, especially on hard surfaces or on surfaces with high frictional forces between the bottom of the foot and the ground. If weight-bearing or torque of the inside limb elicits pain, then the outside limb will display accelerated protraction and earlier-than-expected initiation of weight-bearing. This abnormal movement is more prominent

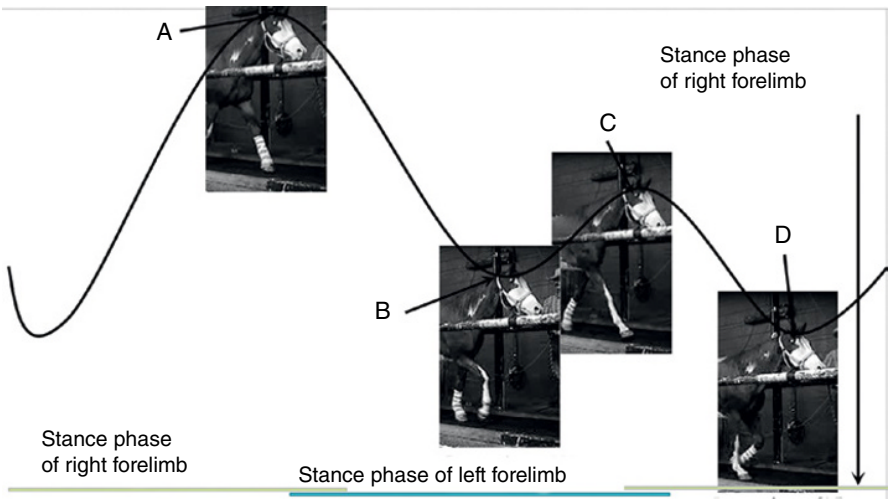


Figure 2.113. Typical vertical head trajectory of a briskly walking horse with a forelimb lameness (in this specific case a left forelimb lameness). (A) Highest head height immediately before impact of the lame or more lame (left) forelimb (note bandage: inferior check ligament previously injected with collagenase to induce lameness). (B) Local minimum head position during stance phase of lame or more lame forelimb that is higher than that during stance phase of the non-lame or less lame forelimb (D). (C) Local maximum head position right before impact of the non-lame or less lame forelimb that is lower than before impact of the lame or more lame forelimb (A). (D) Lowest head position during stance phase of the non-lame or less lame forelimb.

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for forelimb compared with hindlimb lameness, most likely because of higher percent of total body weight borne by the forelimbs compared with the hindlimbs at the walk. Limb position and foot placement under the body, as well as head height from the ground (in forelimb lameness), can also be assessed during turning. The outside limb swings forward and lands quickly, crossing over the lame inside limb. Head movement trajectory during turning with forelimb lameness is typical of an impact-type forelimb lameness seen at the walk and the trot, with the head moving upward greatly during impact of the lame, inside, forelimb.

Lameness conditions that can usually be recognized and diagnosed almost solely by evaluation of gait at the walk include:

1. Fibrotic myopathy: Injury to the stifle flexor muscles (biceps femoris, semimembranosus, semitendinosus) most likely occurs with stifle hyperextension when the hindlimbs are protracted forward and the horse either falls or the hind torso lowers as in a sliding stop. Injury to the stifle flexors can result in pain soon after injury, but fibrosis or ossification at the musculotendinous junction, which can be palpated, is a common, more chronic result. This injury leads to a characteristic hindlimb swing trajectory beginning with increased retraction, followed by the limb swinging forward with low flight arc and slightly shortened length of protraction, and ending with an elevation of the hoof, which precedes an unusual, goose-stepping-like retraction of the hoof backward immediately before impact (Figure 2.114). In contrast the normal hoof flight trajectory has shorter retraction, higher swing trajectory, and greater protraction, with the hoof ending its flight moving forward and downward. It was previously believed that this condition was an isolated swinging limb lameness seen especially and primarily at the walk, with a decrease or elimination of signs at higher speed gaits, and rarely causing clinically significant restricted use or decrease in function. However, recent kinematic studies prove that the condition also causes decreased hindlimb propulsion with characteristic decreased rise of the pelvis after affected hindlimb stance, i.e. a lack of hindlimb vertical propulsion. Moreover, when the restriction in movement is eliminated by transection of the fibrosis, the abnormal swing movement and decreased hindlimb propulsion are both eliminated.
2. Stringhalt: Stringhalt causes lameness of presumed neurologic origin characterized as a sudden hyperflexion of the hindlimb. Known causes are chronic toxicity from ingestion of certain species of weeds (dandelion family) in times of drought and prior trauma to the dorsal surface of the hindlimb metatarsus and tarsus. The hindlimb hyperflexion can be slight or dramatic and may vary in amplitude from stride to stride. When severe, it appears to disturb the horse, but in many cases the horse seems indifferent when it occurs. It is generally thought only to cause dysfunction during the hindlimb hyperflexion, but this author has also measured decreased weight-bearing in the affected limb, and treatment by surgical removal of a length of long digital extensor tendon/muscle can eliminate the hyperflexion and decrease both lameness and apparent discomfort in severe cases.
3. Upward fixation of the patella: Momentary or intermittent upward fixation of the patella is dysfunction in the normally smooth locking and then unlocking of the patella over the top of the medial condyle of the femur during an otherwise normal hindlimb movement. At the end of limb extension, before lift-off of the hindlimb foot, to prepare for initiation of swinging the limb forward, quadriceps muscle activity elevates the patella, unlocking it from the femur and allowing stifle flexion. Delayed or incomplete

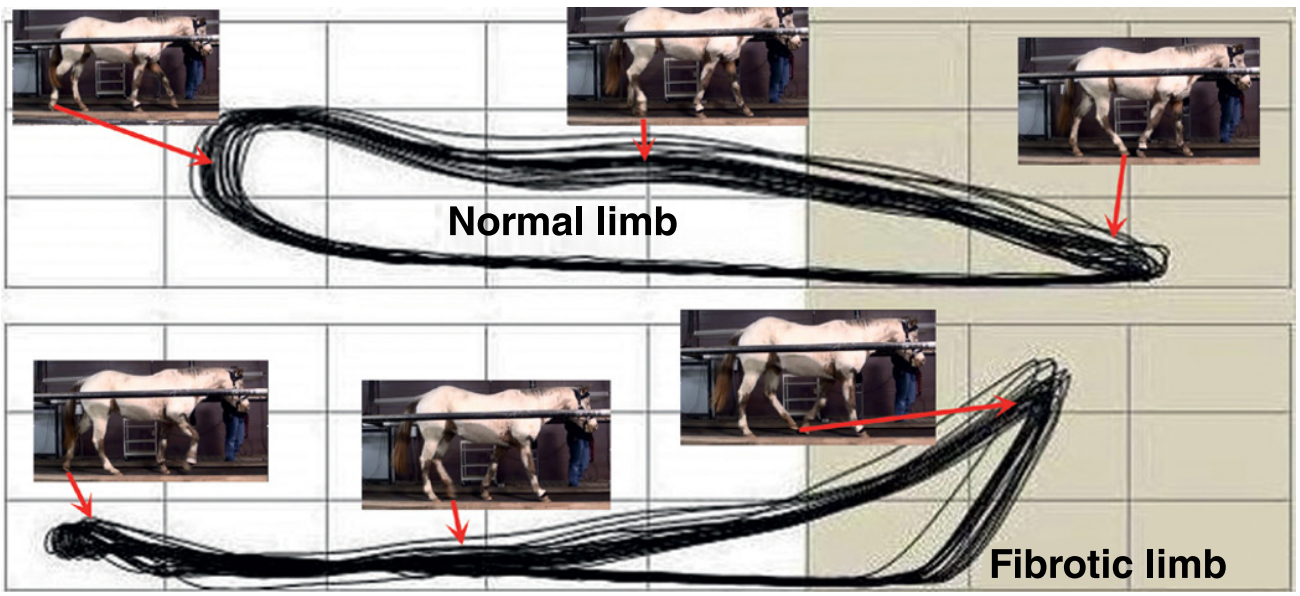


Figure 2.114. Hoof flight trajectories during fibrotic myopathy of the stifle flexor muscles (biceps femoris, semitendinosus, semimembranosus) with associated still video frames indicating limb positions. Upper trajectory is that of the contralateral normal hindlimb foot. Lower trajectory is that of the affected hindlimb foot.

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unlocking results in increased tension in the patellar ligaments at the end of limb extension, followed by a forced unlocking, and then an abnormal release of stored energy, as limb hyperflexion, when finally released. The resulting hyperflexion of the hindlimb is not unlike that of stringhalt (see previous paragraph), but is different in that it is preceded by delayed stifle extension with prolonged weight-bearing and increased hindlimb retraction. Also, momentary or incomplete upward fixation of the patella, compared with stringhalt, is more intermittent (thus the alternative name of the condition, *intermittent* upward fixation of the patella), from stride to stride. In addition to these swinging limb abnormalities, momentary/incomplete/intermittent upward fixation of the patella causes hindlimb weight-bearing lameness, primarily decreased propulsion of the pelvis upward after hindlimb pushoff.

4. Gastrocnemius muscle injury: Injury to the gastrocnemius muscle insertion causes characteristic internal rotation (calcaneus moves abaxially) of the hindlimb at the end of weight-bearing. Only a few cases with definitive diagnosis have been described.[52]
5. Peroneus tertius rupture: Rupture of the peroneus tertius is usually caused by the horse falling with the hindlimbs retracted, falling in a full hindlimb cast, or struggling to free an entrapped or restrained hindlimb. Rupture of this dense fibrous ligament that spans two highly mobile joints (stifle and hock) disrupts the normal function of the hindlimb of coupled stifle and hock flexion and extension. With this injury, at the termination of hindlimb stance, stifle flexion initiates swing of the hindlimb forward, but the hock does not actively flex. Hock flexion is thus delayed, weight-bearing and retraction of the affected hindlimb is prolonged, and the hock flexes only with distal limb momentum as it swings forward. Although at rest the horse can seemingly bear full weight when standing, decreased weight-bearing during movement occurs in the affected limb.
6. Shivers: Shivers is a presumed degenerative neurologic abnormality that causes a hyperflexion or lifting of the hindlimbs, which is more like a spasm than a gait abnormality.

EVALUATION OF THE HORSE AT THE TROT

In many disciplines and breeds of horses, the trot is the most common gait used for observing lameness. In principle the other symmetrical gaits are similar to the trot with the head and pelvis falling and rising during the first and second halves of stance, but with greater (less commonly) or lesser (more commonly) amplitude. So, the basic biomechanical principles describing alteration of head and pelvic height with lameness are the same, but the amplitudes of asymmetries are different. Additional information on lameness in "gaited" horses is explained in Chapter 9.

Detection of lameness in quadrupeds is easiest during the symmetrical gaits, like the trot. A normal expected symmetry provides a standard against which amplitudes of asymmetry between right and left halves of a stride can be compared within the individual horse. However the limbs experience greater vertical force and faster rates of force application at the trot compared to the asymmetric gait of the canter at equivalent speeds, despite the presence of a single-limb weight-bearing portion of the stride.[5,49,56] Decreased loading and weight shifting off a painful limb is more easily accomplished in the symmetrically moving horse. All of these factors contribute to understanding and appreciating why lameness is not only easiest but also more accurately and more precisely evaluated at the symmetric gaits. It has been shown that lameness even at a slow trot will be detected earlier and more consistently than lameness at the asymmetric gait of the gallop.[34]

Speed of movement at the trot has been shown to affect amplitude of lameness, especially when using limb movement as the indicator of lameness. It is reasonable to think that the amplitude of lameness displayed by the horse will increase with speed of movement, but this is not the case. When relying on subjective evaluation, it frequently appears that lameness is greater at slower trotting speeds,[36] but this is most likely because it is easier to see. When measuring lameness, it seems that, instead, there is an optimum speed of movement when lameness amplitude is greatest and stride-by-stride variation is least.[37] Finding this optimal speed is most likely to be successful by allowing the horse to move naturally. This can be confusing when blocking horses, since horses, feeling better, may want to move faster after a successful block. To prevent changes in amplitude of lameness due to speed of movement, one should attempt to keep speed of movement the same before and after block.

The most sensitive body-movement indicators of lameness in quadrupeds is the pattern of vertical movement of the midline of the horse or vertical movement of the head for forelimb lameness and vertical movement of the entire pelvis for hindlimb lameness. Limb movement parameters are useful in some cases, but in general they are too variable and inconsistent to be considered more sensitive and specific indicators of lameness than vertical movement of the head and pelvis.

EVALUATION OF VERTICAL MOVEMENT OF THE HEAD FOR FORELIMB LAMENESS

In order to understand the biomechanics of vertical head movement as it relates to forelimb lameness, some simple principles must be understood. When a horse is trotting, the body moves down and then up in one half of the stride and then down and then up again in the other half of the stride, and the head normally follows this movement pattern. A stride begins when a limb strikes the ground and ends when that same limb strikes the ground again at the start of the next stride. In the non-lame horse, the downward movement in the first half of the stance of one forelimb is equivalent to that in the first half of stance in the other forelimb. Likewise, in the non-lame horse, the upward movement in the second half of stance in one forelimb is equivalent to that in the second half of stance in the other forelimb. Therefore, horses without lameness will have no difference in the lowest positions of the head during stance or in the highest positions of the head before and after stance.

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Therefore, vertical head movement in the perfectly "not lame" horse can be modeled as if there is only one predictable periodic signal at twice the stride rate when the horse is trotting, which is that following the vertical movement of the center of mass of the body (Figure 2.115).[13]

Alternatively, when a horse is lame or more lame in one forelimb, then another periodic factor, at the stride rate, or ½ the frequency of the normal expected vertical movement, additionally influences vertical head height from the ground. The horse will always try to move the head upward during pain with maximum effect at the moment of peak pain. Overall head trajectory is the simple summation of these two components in the horse with lameness. The phase or timing relationship between these two components determines the overall shape of vertical head trajectory. This phase relationship between vertical movement components has been described previously.[2,29,38]

When pain (or other dysfunction) is greatest during the first half of stance, component summation results in an augmentation (a raising) both of the maximum head position before lame (or more lame) stance and of minimum head position during stance of the lame (or more lame) forelimb. Overall vertical head trajectory (a combination of the two components) features differences in maximum and minimum head positions between right and left sides of the stride that are of the same sign, meaning that the highest maximum head height occurs before and the highest minimum head height occurs during lame or more lame forelimb stance (Figure 2.116).

Conversely, when the pain is greatest during the second half of stance, there is augmentation of the minimum head position during stance of the lame or more lame forelimb, as in an impact lameness, but, instead, there is also augmentation in the maximum position of the head after pushoff of the lame or more lame forelimb, which is unlike an impact lameness. Overall vertical head trajectory features differences in maximum and minimum head positions that are of opposite sign, with highest minimum position during and highest maximum position following lame or more lame forelimb stance (Figure 2.117).

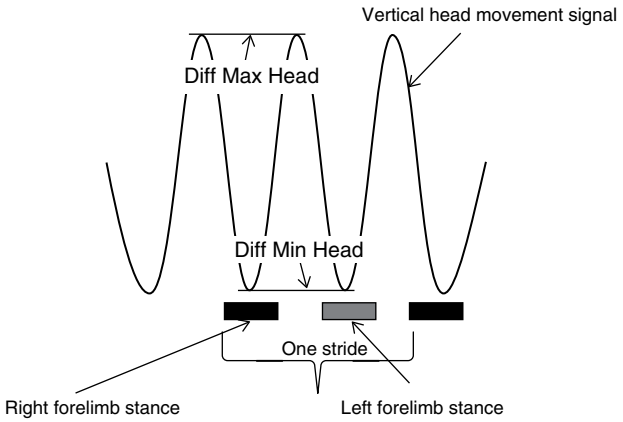


Figure 2.115. Head movement trajectory in non-lame trotting horse following trajectory of center of mass of the body. Trajectory is similar to a sine wave at frequency of twice stride rate because movement is down and then up twice per single stride. In a horse without lameness, downward movement in first half of stance of one forelimb is equivalent to that in the opposite forelimb, and upward movement in second half of stance of one forelimb is equivalent to that in the opposite forelimb. Therefore, the differences in positions of the head from the ground at local minimums (during stance) or, Diff Min Head, is equal to 0. Likewise, difference in positions of head from the ground at local maximums (between the stance phases of each forelimb), or Diff Max Head, is equal to 0.

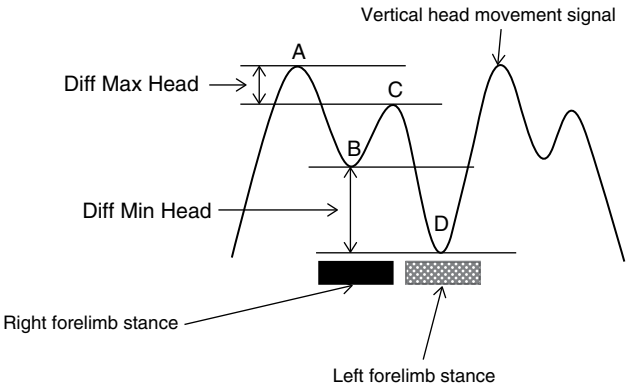


Figure 2.116. Overall vertical head trajectory pattern for horse with impact-type right forelimb lameness or lameness with pain greatest in the first half of, in this case, right forelimb stance. Peak (highest maximum) head vertical position occurs near beginning of stance of the lame or more lame forelimb. Diff Max Head or first maximum head height (A: near beginning of stance of the right forelimb) minus second maximum head height (C: near beginning of stance of the left forelimb) is a positive value. Lowest head position occurs during stance of the not or less lame forelimb. Diff Min Head or first minimum head height (B: during stance of the right forelimb) minus second minimum head height (D: during stance of the left forelimb) is a positive value. Diff Max Head and Diff Min Head are the same sign. For right forelimb lameness, both Diff Max Head and Diff Min Head are positive.

In both of the above types of lameness, the entire amplitude of forelimb lameness is reflected in a combination of the difference in low and high heights of the head. The classic description of "head nod" as only "down on sound" or only as "up on bad" neglects consideration of differences in both the minimum and maximum head positions and will therefore underestimate amplitude of forelimb lameness.

When pain is greatest near the middle of stance, the effect of the head moving upward during peak pain will augment only the minimum head position during stance of the lame or more lame forelimb so that there is a difference in the minimum head positions between right and left stance but no difference in maximum positions of the head (Figure 2.118; Diff Max Head = 0). In this case the amplitude of "head nod," usually described as "down on sound" or "low on sound," captures the entire amplitude and will not underestimate forelimb lameness. This is probably why midstance forelimb lameness is easiest to detect subjectively and veterinarians infrequently disagree on limb involved or amplitude with this type of forelimb lameness.

The classic "head nod" as "down on sound" implies that in a forelimb lame horse, the head moves down when the non-lame or less lame forelimb is weight bearing, and then up when the lame or more lame forelimb is weight bearing. This is a deficient simplification that

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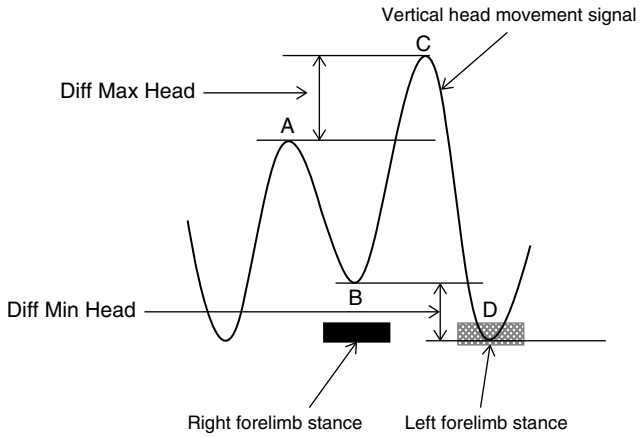


Figure 2.117. Overall vertical head trajectory pattern for horse with pushoff type, in this case right forelimb lameness, or lameness with pain greatest in the second half of right forelimb stance. Peak (highest maximum) head vertical position occurs near beginning of stance of the not or less lame forelimb. Diff Max Head or first maximum head height (A: near beginning of stance of the right forelimb) minus second maximum head height (C: near beginning of stance of the left forelimb) is a negative value. Lowest head position occurs during stance of the not or less lame forelimb. Diff Min Head or first minimum head height (B: during stance of the right forelimb) minus second minimum head height (D: during stance of the left forelimb) is a positive value. Diff Max Head and Diff Min Head are of opposite signs. For a right forelimb pushoff lameness Diff Min Head is positive (+) and Diff Max Head is negative (–).

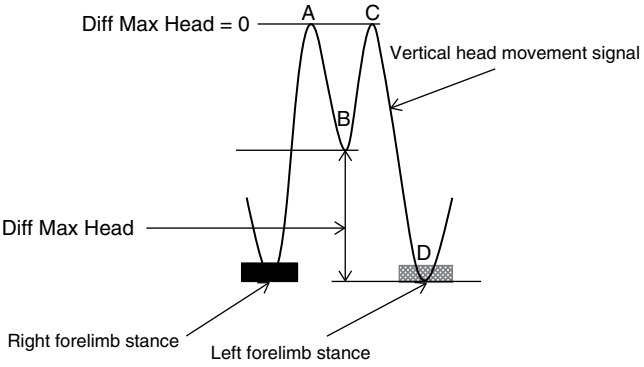


Figure 2.118. Overall vertical head trajectory pattern for horse with midstance right forelimb lameness or lameness with pain greatest in the middle of right forelimb stance. Peak (maximum) head vertical position before right and left forelimb stance is equivalent. Diff Max Head or first maximum head height (A: near beginning of stance of the right forelimb) minus second maximum head height (C: near beginning of stance of the left forelimb) = 0. Lowest head position occurs during stance of the not or less lame forelimb. Diff Min Head or first minimum head height (B: during stance of the right forelimb) minus second minimum head height (D: during stance of the left forelimb) is a positive value.

is really only true in horses with severe lameness. But, in horses that are trotting, the head follows the center of mass of the body, moving down when the horse is landing on a forelimb and then up when the horse pushes off that forelimb, even with moderate to severe lameness. The head does not just move up during the stance phase of the lame limb and down during the stance phase of the sound limb. So why is this incorrect maxim "down on sound" propagated in our veterinary lexicon and still taught to veterinary students?

Examining the different shapes of the trajectories of vertical head movement as one (the most simple) type of forelimb lameness is gradually increased suggests the origin and context of this false maxim (Figure 2.119). Considering a very mild lameness, the asymmetry of the down and then up movement, twice per stride, is small, so there is just a little less downward motion of the head (upper left quadrant, Figure 2.119) during lame limb stance. The head halts its fall at a slightly higher vertical position during lame or more lame forelimb stance compared with not lame or less lame forelimb stance. As the lameness increases, this asymmetry is increased, but the overall shape of the vertical head movement trajectory (upper right and lower left quadrants, Figure 2.119) is retained. Ultimately, as the amplitude of lameness is further increased, an inflection point in the trajectory is reached, and the downward and then upward movement of the head is now upward in the first and then downward in the second half of stance. This can appear as primarily upward movement with subjective evaluation. This deficient version of "down on sound" seems correct only when the lameness is severe, so, in the practical sense, it is not a useful explanation.

With pain in the forelimbs that is maximum during the first half of weight-bearing, the horse will move its head upward so that it is decelerating upward at the beginning of stance and then accelerating downward during the remaining first half of stance. Thus the head will fall from a relatively higher position but then halt its descent prematurely so that it ends up at a local minimum position higher compared with its position during the contralateral (less lame side) stance. The common description of this "head nod" phenomenon as "down on sound," meaning moving down more on the sound limb, is therefore also not always true, and the actual length of downward descent may be greater than during the same period of contralateral (not lame or less lame) forelimb stance. The more accurate descriptive maxim in this type of forelimb lameness would therefore be "lowest on sound."

Alternatively, if the pain is present during the second half of stance when the head is normally moving upward with the center of mass of the body, the head will move up more, decelerating at the end of stance. The head will then reach its highest vertical position preceding the beginning of stance in the contralateral, non-lame or less lame, forelimb. There will be a difference in the maximum heights of the head with the head highest before the stance phase of the not lame or less lame forelimb. This is opposite that of lameness that occurs during the first half of stance, where the head will move up more after pushoff of the non-lame or less lame forelimb. However, side of lameness remains determined correctly by the maxim "lowest on sound." "Lowest on sound" is a correct description of side of lameness, regardless of timing of lameness within stance.

Thus the side of forelimb lameness is identified by the difference in the low or minimum positions of the head during stance, and timing of lameness is determined by the difference in the high or maximum positions of the head occurring after pushoff of each forelimb.

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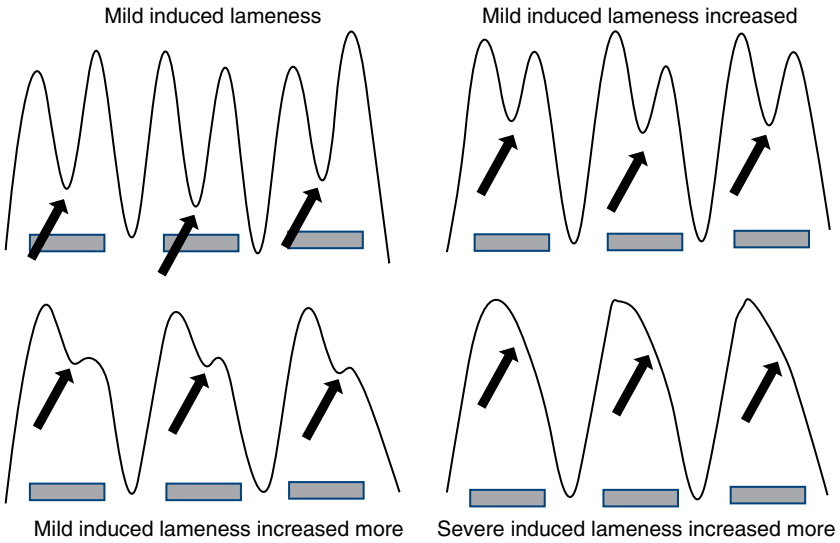


Figure 2.119. Overall vertical head trajectory patterns for a horse with gradually increasing forelimb lameness from sole pressure. Least amount of induced lameness shown in upper left plot and most amount of induced lameness shown in lower right plot. Gray bars indicate stance phase of one forelimb. As amplitude of lameness increases, characteristic downward and then upward head movement changes (reverses) to upward and then downward head movement until lameness amplitude increases to the point that greatest observed movement direction is upward during lame or more lame forelimb stance and downward during not or less lame forelimb stance.

The amplitude of lameness, which affects both the difference in the low and high positions of the limb, is proportional to some combination of these differences. The better maxim, or definition of "head nod," to determine side of lameness, is "lowest on sound." However, the amplitude of this explanation of "head nod" will not always be proportional to the true amplitude of forelimb lameness, because a difference in low positions of the head, depending on the timing of lameness, may represent only a portion of the head movement asymmetry. The side of lameness can be determined by the difference in the low positions of the limb, but the amplitude of lameness must take into consideration both the differences in the high and low positions of the head.

When horses are trotted in a straight line while restrained by a handler or during lunging, both which are common activities used by practitioners to evaluate horses for lameness, the speed of movement is such that most forelimb lameness is measured as greatest during the first half of stance (impact to midstance). This is most likely because at slow speeds the front limb acts mostly as a strut or crutch with little contribution to active propulsion of the body forward, most of which is provided by pushoff of the hindlimbs. At higher speeds pushoff-type forelimb lameness is measured with greater frequency. Although timing of forelimb lameness is most likely dependent upon speed of movement and ground surface qualities,[3] knowledge of whether the lameness is manifested primarily during the first or second half of stance may be valuable to the practitioner in helping to localize the cause of lameness within the affected forelimb.

Other random causes of vertical head movement, which will interfere with subjective evaluation of head nod, must somehow be extracted and ignored by the examiner when detecting forelimb lameness. This can be difficult with misbehaving horses when they are randomly shaking or jerking the head in the vertical plane.

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Vertical movement of the withers may also be used to detect and measure amplitude of forelimb lameness, and there is evidence that the character of withers vertical movement may help to differentiate compensatory from primary lameness in the multiple limb lameness situation.[42] When the asymmetry of withers movement is opposite in sign to that of the asymmetry of head movement, for example, if lowest head position is during the stance phase of the left forelimb (a sign of right forelimb lameness) but lowest withers position is during the stance phase of right forelimb, a primary hindlimb lameness (right) should be suspected. Whereas when the asymmetry of withers movement is the same sign as that of asymmetry of head movement, a primary forelimb lameness should be suspected.

However, in general asymmetric withers movement is less sensitive than asymmetric head movement at detecting amplitude of forelimb lameness. Because the forelimb is attached to and supports the axial skeleton by a soft tissue sling mechanism, ground reaction force and body center of mass effects on each other are damped, and the amplitude of asymmetric movement is suppressed.

EVALUATION OF VERTICAL MOVEMENT OF THE PELVIS FOR HINDLIMB LAMENESS (THE *V*ERTICAL *P*ELVIC *M*OVEMENT [VPM] METHOD)

Evaluation of hindlimb lameness is easier conceptually, but more difficult practically, than evaluation of forelimb lameness. It is easier conceptually because movement of the posterior half of the horse's torso can be modeled as a simple single free body moving down and up in response to resultant and applied ground reaction forces.[7] It is more difficult practically because the amplitude of vertical movement is small and more difficult to see.

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The most direct observational sign of hindlimb lameness is asymmetric vertical movement of the entire posterior end (pelvis) of the horse. The horse has no large mass on a long lever arm projecting out of the hindquarters, so the simple mechanism achieved by altering head movement to adjust forelimb weight-bearing is not available to the horse with hindlimb lameness. Other adjustments must be performed. To decrease force on the hindlimbs, the pelvis either falls less (like dropping a ball from a lower height), if the pain is greatest in the first half of stance, or rises less, from decreased hindlimb vertical propulsion, if pain is greatest during the second half of stance. As the severity of hindlimb abnormality increases, both decreased fall and decreased rise (impact and pushoff lameness) are seen. The adjustments made by the horse to decrease fall and to decrease rise of the pelvis are different.

Decreased fall of the pelvis results in a difference in low positions of the pelvis near midstance, lower (i.e. closer to the ground) on the not lame or less lame hindlimb, if the pain is primarily in the first half of hindlimb stance (Figure 2.120). For purposes of classification, this can be called an "impact"-type hindlimb lameness.

When pain is greatest in the second half of stance, the horse pushes off less, causing the pelvis to rise less, resulting in a difference in the high positions of the pelvis (Figure 2.121). It is lower after lame limb pushoff. For purposes of classification, this can be referred to as a "pushoff"-type hindlimb lameness.

Hindlimb lameness can be both impact and pushoff, which becomes more common as hindlimb lameness increases in severity. Hindlimb lameness can also be impact on one side and pushoff on the other, a bilateral lameness, but this is not common. Unlike forelimb lameness, where at moderate speeds most lameness is of the impact type, for hindlimb lameness a predominant impact- or pushoff-type lameness occur with approximate equal frequency. This is probably because the hindlimb acts as both a propulsive force during pushoff and a braking force during impact even at moderate speed of movement. There is objective evidence that impact and pushoff hindlimb lameness have different vertical and horizontal ground reaction force profiles.[6]

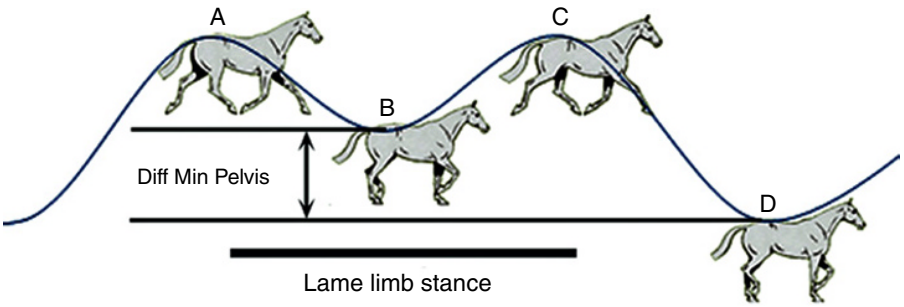


Figure 2.120. Overall vertical pelvis trajectory pattern for horse with impact-type hindlimb lameness or lameness with pain greatest in the first half of hindlimb stance. Peak (maximum) vertical pelvic position before right and left hindlimb stance is the same. Diff Max Pelvis or first maximum pelvis height (A: near beginning of stance of the right hindlimb) minus second maximum pelvic height (C: near beginning of stance of the left hindlimb) is zero. Lowest pelvic position occurs during stance of the not or less lame hindlimb. Diff Min Pelvis or first minimum pelvic height (B: during stance of the right hindlimb) minus second minimum pelvis height (D: during stance of the left hindlimb) is a *positive value for right hindlimb lameness (and a negative value for left hindlimb lameness)*.

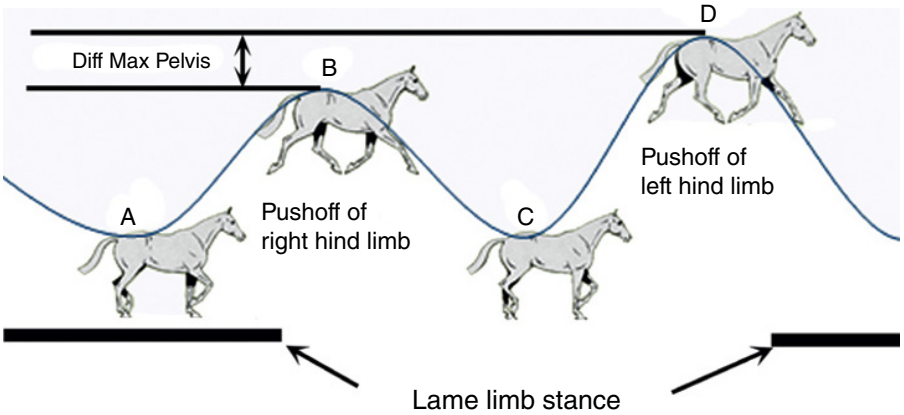


Figure 2.121. Overall vertical pelvis trajectory pattern for horse with pushoff-type right hindlimb lameness or lameness with pain greatest in the second half of right hindlimb stance. Peak (maximum) vertical pelvic position after pushoff of left hindlimb (D: before impact of right hindlimb) is greater than vertical pelvic position after pushoff of right hindlimb (B: before impact of left hindlimb). Diff Max Pelvis or first maximum pelvis (near beginning of stance of the right hindlimb, D) minus second maximum pelvic height (B: near beginning of stance of the left hindlimb) is positive. Lowest pelvic position occurs during stance of right and left hindlimbs. Diff Min Pelvis or first minimum pelvic height (A: during stance of the right hindlimb) minus second minimum pelvis height (C: during stance of the left hindlimb) is zero.

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Knowledge of and ability to detect the different types of pelvic asymmetry may be helpful to veterinary practitioners isolating lameness within the affected limb.

Decreased pelvic fall during hindlimb lameness is accomplished by complex muscular activity in the proximal hindlimb, by shifting weight to the contralateral forelimb, and by "hip hiking" as the horse attempts to use the lame-side hemipelvis in a similar (but not so successful) manner as it uses its head in forelimb lameness, elevating the lame-side hemipelvis immediately before weight-bearing. Alternatively, decrease pelvic rise during pushoff-type hindlimb lameness is primarily caused by reduced extensor muscle activity, resulting in decreased propulsion of the body mass upward and forward. Because the pelvis rises to a lower height relative to the ground after deficient pushoff, swinging the limb forward subsequently requires more limb flexion to prevent toe dragging. This increase limb flexion during swing creates the "hip dip" that occurs after stance of the lame-side hindlimb. Insufficient limb flexion and "hip dip" result in toe dragging, an additional sign of a hindlimb lameness characterized by decreased pushoff. Because the causes of decreased hindlimb impact and decreased hindlimb pushoff are different, impact- and pushoff-type hindlimb lameness should be considered separate entities, and total amount of hindlimb lameness is the sum of both.

EVALUATION OF PELVIC ROTATION FOR HINDLIMB LAMENESS (THE *P*ELVIC *R*OTATION *M*ETHOD [PRM])

Some veterinarians observe for hindlimb lameness at the trot by assessing amount of "hip hike" and "hip dip," which are manifestations of asymmetric pelvic rotation in the frontal plane, instead of observing whole pelvis rise and fall as described above.[35,39,40] However, reference to the "hip" (coxofemoral joint) in this observation method is actually a misnomer as the hip joint is not visible under the gluteal muscle masses. The evaluator is actually observing vertical movement of the tubera coxae.

When a horse is trotting, the vertical movement trajectory of each tuber coxae is normally asymmetric in time (i.e. it is not similar to a perfect sine wave). It moves downward during the first half of stance to reach a local minimum at midstance and then upward as the horse straightens and pushes off the hindlimb. The tuber coxae then moves down a greater amount as the limb flexes and swings forward, reaching its lowest position from the ground after that hindlimb pushoff. Finally, during hindlimb protraction, the tuber coxae moves upward, back to its original height, in preparation for hindlimb hoof contact and the beginning of the next stride's stance (Figure 2.122).

The pelvis is minimally rotated, and the right and left tuber coxae are at equivalent heights when the normal (i.e. not lame) horse is not weight bearing with one hindlimb in retraction just after pushoff and one in protraction, just before impact. The pelvis is maximally rotated when one hindlimb is weight bearing and the other is swinging forward, with the tuber coxae on the weight-bearing side higher than the tuber coxae on the non-weight-bearing side. The pelvis is rotated toward the hindlimb swinging forward.

With most hindlimb lameness, total vertical movement of the tuber coxae is greatest on the lame or more lame side. This may be because of both a "hip dip" (a low position of the tuber coxae) right after pushoff of the lame hindlimb and a "hip hike" (a high position of the tuber coxae) right before impact of the lame limb. A "hip dip" occurs because the lame hindlimb, after weak pushoff and small amplitude of pelvic rise, must have increased flexion (and dipping of the tuber coxae

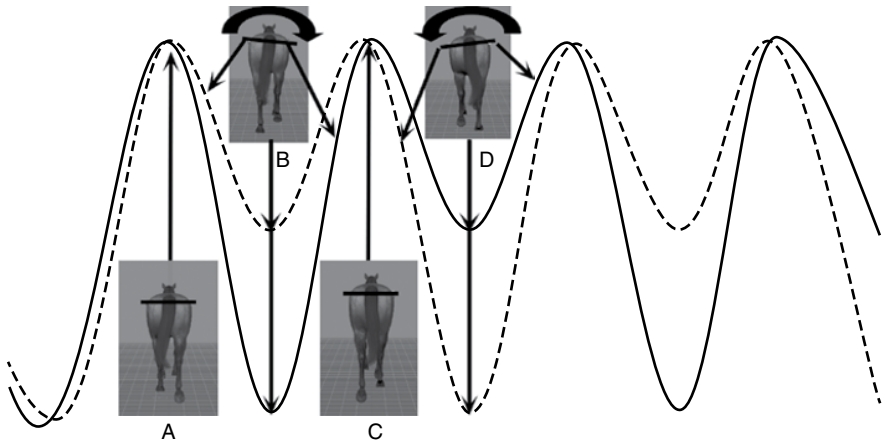


Figure 2.122. Vertical trajectories of right (solid line) and left (dashed line) tuber coxae in horse without hindlimb lameness. (A) Just before impact of left hindlimb and after pushoff of right hindlimb, both tuber coxae at same vertical heights from the ground and the pelvis are not rotated relative to the ground. (B) During left hindlimb stance, left tuber coxae at local minimum that is higher than right tuber coxae local minimum when right hindlimb at midswing. The pelvis is rotated to the right with the right hindlimb flexed. (C) Just before impact of right hindlimb and after pushoff of left hindlimb, both tuber coxae at same vertical heights from the ground and the pelvis, once again, are not rotated relative to the ground. (D) During right hindlimb stance, right tuber coxae at local minimum, which is higher than left tuber coxae local minimum when left hindlimb at midswing. The pelvis is rotated to the left with the left hindlimb flexed. *Source*: Screenshots of horse animations taken (with permission) from LamenessTrainer.com (authors/developers S.D. Starke, G.C. Miles, and S.A. May, funded by the Eranda Foundation, developed at the Royal Veterinary College, Hatfield AL9 7TA, UK).

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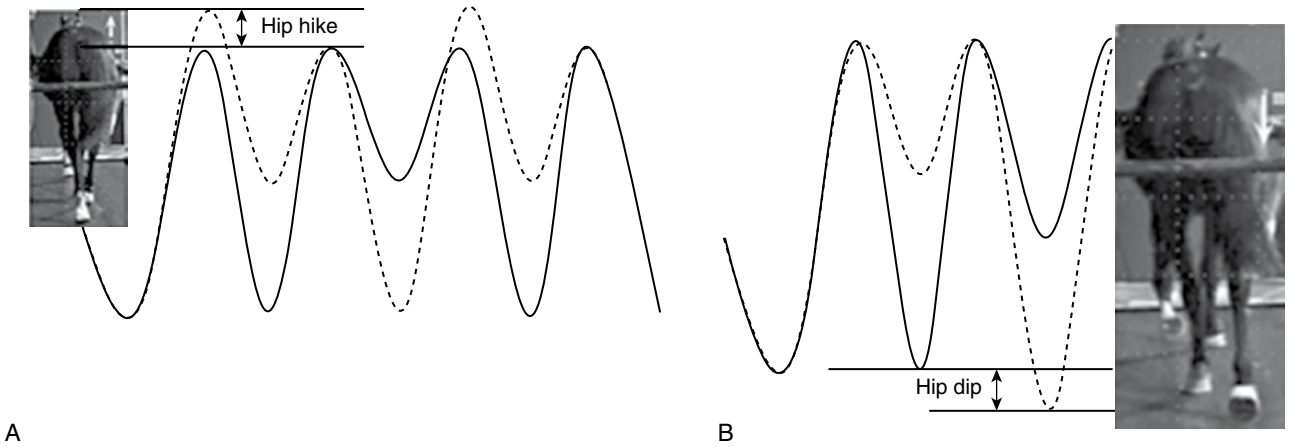


Figure 2.123. (A) Description of "hip hike" as a modification of lame-side tuber coxae trajectory. Upward movement of tuber coxae reaching highest vertical position before impact of lame hindlimb, such that vertical position of tuber coxae before lame (or more lame) side hindlimb impact is higher than that of the opposite side vertical tuber coxae position before the normal (or less lame) side hindlimb impact. (B) Description of "hip dip" as a modification of lame-side tuber coxae trajectory. Downward movement of tuber coxae reaching lowest vertical position after pushoff of lame hindlimb, such that vertical position of tuber coxae after pushoff of lame (or more lame) side hindlimb is lower than that of opposite side vertical tuber coxae position after pushoff of normal (or less lame) hindlimb.

to that side) to prevent toe dragging (Figure 2.123). Sometimes this flexion is insufficient to prevent toe dragging, and toe dragging is also considered a sign of hindlimb lameness. A "hip hike" occurs in an attempt to prevent or dampen pelvic fall before lame hindlimb impact. As such, the "hip hike" signifies an impact-type hindlimb lameness (which, for the VPM, would create a difference in low pelvic positions), and "hip dip" signifies a pushoff hindlimb lameness (which, for the VPM, would create a difference in high pelvic positions). An alternative description of "hip hike" that does not account for type of hindlimb lameness (impact or pushoff) defines it as the total movement upward of the tuber coxae on the lame side instead of the relative differences in tuber coxae height from the ground between right and left stances.[40] This definition suggests that "hip dip" and "hip hike" are not separate phenomena describing different timing of hindlimb lameness.

Both VPM and pelvic rotation, compared to most hindlimb movement parameters, are more sensitive and specific indicators of hindlimb lameness. When veterinary trainees were tested for determining hindlimb lameness, those that concentrated observation on evaluating pelvic movement scored better than those that spent time observing limb movement.[50] The pelvis rotation method, which is easiest to see because the motion is greater, relies on pelvic anatomy symmetry and pelvic rotation around the lumbosacral joint. However, preexisting asymmetry of pelvic anatomy may cause asymmetry of pelvic rotation, or the appearance of asymmetric pelvic rotation, when VPM is symmetric between right and left hindlimbs. The VPM, which is more difficult to see in some horses because the amplitude of vertical displacement can be small, depends directly on the force of impact and pushoff of the hindlimbs. An easily visible marker fixed to the most dorsal aspect of the pelvis between the tuber sacrale may help to detect this asymmetric movement.

There is some evidence that pelvic rotation, or the range of motion of the vertical movement of the tuber coxae, is not proportional to severity of lameness above mild hindlimb lameness.[51] It is clear that pattern of vertical movement of the tubera coxae (pelvic rotation method [PRM] of hindlimb lameness detection) does not accurately quantify the true state of vertical tubera sacral movement (VPM method) and vice versa; in other words, they do not exactly evaluate the same thing.[51] In one study "hip hike" was the most sensitive indicator of mild hindlimb lameness.[40] In one study vertical movement of the tuber sacrale had the highest discriminating power for determination of hindlimb lameness.[15]

The best position for the evaluator using the PRM is behind the horse with the horse moving away from the evaluator with spatial resolution of the observer degrading with each stride. The VPM method is more versatile because it can be observed equally well from all perspectives. Nevertheless, most practitioners today use the PRM in some form for detection and evaluation of hindlimb lameness. Table 2.2 and Figure 2.124 compare and contrast the observations for hindlimb lameness using the VPM and PRM.

An excellent online training site, www.lamenesstrainer.com, lets one practice both methods of hindlimb lameness detection (VPM and PRM).

BILATERAL LAMENESS

In theory asymmetric head and pelvic vertical movement will not be observed with a bilateral lameness of equal intensity in both limbs.[11] However, horses with bilateral lameness most often have one side slightly worse than the other, and an asymmetry representative of this difference will be observed. Occasionally the lameness will be shifting from one side to the other between strides or between trials of trotting away and then toward the observer. When a bilateral lameness is suspected, attempts to "lateralize" the lameness assist in the evaluation. Turning the horse in a circle, lunging in both directions, flexing or stressing one limb, and blocking and then observing the horse in motion are methods that "lateralize" a bilateral lameness.

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Table 2.2. Methods of lameness evaluation using pelvic movement.

	Method of Lameness Evaluation Using Pelvic Movement	
Type of Hind Limb Lameness	Vertical Pelvic Movement(VPM)	Pelvic Rotation (PRM)
Impact Hind Limb Lameness	Diff Min Pelvis Positive for right, negative for left	Hip Hike Greater on lame side
Pushoff Hind Limb Lameness	Diff Max Pelvis Positive for right, negative for left	Hip Dip Greater on lame side

Compares and contrasts the pelvic movement parameters for both the vertical pelvic movement method (VPM) of hind limb lameness detection as well as that of the pelvic rotation method (PRM). Diff Min Pelvis is the difference in whole pelvis minimum position between left and right hind limb stance. Diff Max Pelvis is the difference in whole pelvis maximum position before right hind impact minus whole pelvic position before left hind limb impact. "Hip Hike" is the greater elevation of the tuber coxae before lame (or more lame) hind limb impact compared to before not lame (or less lame) hind limb impact. "Hip Dip" is the more lower position of the tuber coxae when the lame (or more lame) limb is swinging forward compared to the low position of the tuber coxae when the not lame (or less lame) hind limb is swinging forward.

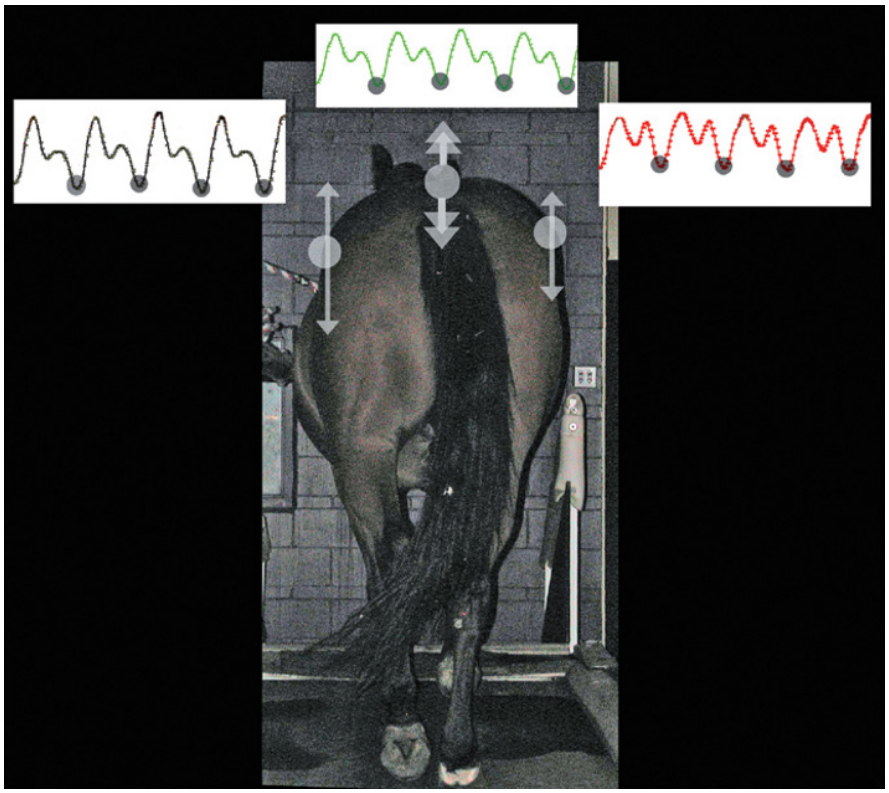


Figure 2.124. Two methods of hindlimb lameness detection and evaluation. Vertical pelvic movement method evaluates imaginary "ball" on midline of pelvis between the tuber sacrale. Pelvic rotation method evaluates imaginary "balls" located at left and right tuber coxae. Vertical pelvic movement method relies on temporal asymmetry of movement of entire pelvis, with less downward movement of the pelvis (pelvis stops downward movement at higher height) during stance phase of lame limb and/or less upward movement of the pelvis (pelvis stops upward movement at lower height) after pushoff of the lame limb, depending on whether the lameness is impact or pushoff or both. Top curve (green) indicates dorsal pelvic movement through four strides of horse with left hindlimb lameness with black circle at time of sound (right) hindlimb stance (synchronous with picture). Pelvic rotation method relies on greater total vertical movement of the tuber coxae on the lame hindlimb side. Curve on left (black) indicates left tuber coxae vertical movement through four strides in horse with left hindlimb lameness with black circles at time of sound (right) hindlimb stance (synchronous with picture). Curve on right (red) indicates right tuber coxae vertical movement through four strides in horse with left hindlimb lameness with black circles at time of sound (right) hindlimb stance (synchronous with picture).

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If attempts to lateralize the lameness do not successfully produce observable asymmetric movement, the evaluator must rely on absolute movement parameters, differentiating these from his/her memory bank of how a normal horse moves. For bilateral forelimb lameness, a diagonal advanced placement of the forelimb, with the forelimb hoof hitting the ground before the contralateral hindlimb (but this is normal in some breeds), and an overall suppression of vertical head movement (compared with vertical movement of the torso) are sometimes appreciated. For both bilateral forelimb and hindlimb lameness, a stiff, shuffling, and short appearing stride may be seen.[43] However, unless the norm of the horse before the onset of lameness is known, this can be difficult to differentiate from individual, idiosyncratic locomotion patterns.

OBSERVING MOVEMENT OF THE LIMBS

Many motion parameters have been measured and studied as lameness indicators in horses, and there are many limb movement parameters that have been associated with lameness. It is not unusual to find in the literature reports of kinematic studies in which many different limb motion parameters were measured but few were found to be significantly associated with lameness. Although there is considerable overlap, there is also considerable contradiction between studies as to which motion parameters are sensitive indicators of lameness in horses. Some difference can be explained by differences in the models of lameness being studied. Despite these qualifications, there are some limb movement parameters that can be used as indicators of lameness in horses. However, high variability and low consistency, with some exceptions, make observing limb movement changes less critical for detecting and evaluating for lameness.

JOINT ANGLE MEASUREMENTS ASSOCIATED WITH LAMENESS

Decreased weight-bearing due to lameness in the forelimbs or hindlimbs will decrease maximum fetlock extension and maximum coffin joint flexion during the stance phase of the lame limb compared with the stance phase of the contralateral sound limb (Figure 2.125).[12,19] Maximum fetlock extension and coffin joint flexion during stance are sensitive indicators of both forelimb and hindlimb lameness in the horse. Fetlock extension during lame limb stance was 8° less than sound limb stance in a sole pressure-induced lameness model of a grade 2 (out of 5) lameness.[12] If fetlock joint angle could be detected sensitively and accurately, it would be an excellent visual clue for the detection and evaluation of forelimb and hindlimb lameness.[12] Carpal extension during stance is reduced but only with moderate to severe lameness.[28]

STRIDE TIMING AND LENGTH VARIABLES ASSOCIATED WITH LAMENESS

Stride timing variables (stance duration, swing duration, etc.) are relatively insensitive and inconsistent indicators of lameness. Unless the lameness is severe, when stance time is shortened, stance time can be relatively

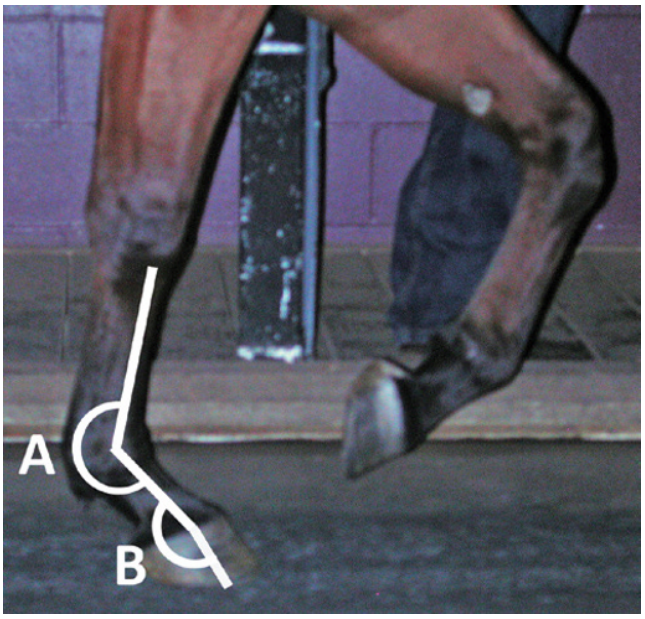


Figure 2.125. (A) Fetlock extension and (B) coffin flexion angles during full weight-bearing. With lameness (A) decreases and (B) decreases.

increased in either the lame or more lame hindlimb or in the non-lame or less lame contralateral hindlimb,[53,55] whereas relative stance duration as a percentage of total stride time and total stride time consistently decreases in the lame or more lame limb. Diagonal advanced placement, or initiating stance sooner in the limb diagonal to the lame limb (or ipsilateral limb for the rack and pace), may also be shortened. Step duration, or the time between pushoff of one limb and impact on the contralateral limb, on the other hand, is shorter between pushoff of the lame and impact of the sound limb than between pushoff of the sound limb and impact of the lame limb.[10] However, stride timing variables are strongly dependent on speed of forward movement and training, which makes them less useful for detecting lameness before and after blocking or treatment.[20]

Stride length is shortened significantly only in moderate to severe lameness.[4] Step length, or the distance between placements of opposite limbs, is less between placements of the lame and then sound limbs than between placement of the sound and then lame limb.[10] Height of foot flight arc may be increased or decreased in the lame forelimb compared to the sound forelimb, and the shape may be different, depending on the cause of lameness.[12] Dragging the hindlimb toe is commonly thought to be a sign of subtle hindlimb lameness, but this may not always be true. In the hindlimb, the height of hoof flight arc is determined by two competing factors with the strongest determining the overall effect. Decreased propulsion during pushoff of the lame hindlimb causes the rear torso to rise less. In order to bring the affected limb forward during the swing phase of the stride without dragging it on the ground, the proximal limb joints will flex more. The comparative extents of the decreased torso rise and increased limb flexion determine the height of the hoof flight arc. Amplitude of limb retraction is variable depending on type of lameness and conditions of evaluation.[12]

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Although amount of limb abduction or adduction during the swing phase of the limb has been thought to be indicative of certain lameness conditions, this has not been objectively studied. Placement of the hoof during weight-bearing either directly under (less lame limb) or abaxial (more lame limb) is, ostensibly, a sensitive indicator of lameness. However, this author has measured this in many horses with induced lameness and found this to be inconsistent and contradictory.

HOOF TRAJECTORY ASSOCIATED WITH LAMENESS

The length and shape of forelimb and hindlimb hoof trajectory during the swing phase of the stride are commonly perceived to be associated with lameness. However, objective measurements of these changes have not been confirmed, and in a few cases have contradicted, these perceptions. Forelimb protraction, the cranial phase of the hoof flight arc, will either decrease or increase with lameness, depending upon type of lameness.[28] For example, in the foot, heel lameness (e.g. navicular disease) generally decreases, but toe (e.g. laminitis) lameness generally increases forelimb retraction. However, hindlimb protraction is usually decreased in most cases of hindlimb lameness.[30] Unilateral decreased hindlimb protraction is easy to recognize in the lame limb at the trot by visualizing the distance between the affected hindlimb at full protraction and the ipsilateral forelimb at full retraction (Figure 2.126). This distance is longer on the lame side. However, a simultaneous increase in retraction of the forelimb, which occurs in some forelimb lameness conditions, will interfere with the proper interpretation of this sign.[12]

There are numerous other subjective observations thought to indicate lameness in the horse, including amount of head flexion when ridden, overall straightness of movement, sequence and timing of foot fall cadence, being "on the bit," amount of "engagement" of the hindlimbs, "ease" of transition from one gait to another, overall willingness to move, and many others. When lameness is difficult to detect because asymmetric vertical movement of the head and pelvis is less than what can be seen by the naked eye, the veterinarian is forced perhaps to then attempt to observe many different things. In general, limb movement, pelvic rotation, and other highly subjective observations may be used to help confirm the existence and extent of lameness and in some cases may even hint at the location of abnormality within a lame limb, but they should not contradict vertical head and pelvic parameters that are directly related to altered vertical ground reaction forces, and if they do, they should be considered idiosyncratic and not indicative of abnormality. Some clinicians have claimed even that lameness can be observed by detecting expressions on the horse's face during riding or lunging, an equine "pain face."[18] However "painful" facial expressions may signify discomfort in any body system or because of a variety of causes including those that cause lameness.

LAMENESS EVALUATION DURING IPSILATERAL GAITS

The vertical movement trajectories of the head and pelvis during the rack and pace are identical in concept but smaller in amplitude compared to that found at the trot. The body still moves down during the first half and up during the second half of stance. Therefore the same principles of altered movement with lameness can be



Figure 2.126. Hindlimb protraction is easily seen when viewing from the side of the horse. The hoof of the retracted ipsilateral forelimb acts as a point of reference. Hindlimb protraction is reduced in most hindlimb lameness; thus, the space between the retracted forelimb and protracted hindlimb is greater (arrows) on the lame hindlimb side.

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considered, with "lowest on sound" for both head and pelvic movement for fore- and hindlimb lameness, as an accurate indicator of side of lameness. However, the total vertical movement with these ipsilateral gaits tends to be less than the trot, with more rolling from side to side than vertical movement up and down. So, although, in principle, vertical movement of the head and pelvis should be good indicators of lameness, they may be more difficult to observe. Certainly, also, the compensatory lameness patterns for ipsilateral gaits will be different than that for the contralateral trot. More information of lameness in gaited breeds can be found in Chapter 9.

EVALUATION OF LAMENESS AT THE LUNGE

Evaluation for lameness during the lunge, on either soft or hard ground, is thought to exacerbate and bring out lameness that may not be seen when the horse is moving in a straight line. Moving in a circle, because the torso and limbs are tilted relative to ground surface, alters the distribution of ground reaction forces both within a single limb and between right and left limbs. However, because the torso is tilted relative to the ground,[24,25] there is normal asymmetrical peak vertical forces between the inside and outside limb[14,17] and normal vertical movement of both the head and pelvis.[41,46] These asymmetries must be appreciated and understood as normal. Large horses, small diameter circles, and high speed of movement will exacerbate these asymmetries. It has also been determined that the amplitude and characteristics of vertical head and pelvic asymmetries are dependent on type of ground surface on which the horse is lunged, and because of high normal horse-to-horse variability, an asymmetry observed in one direction should not be identified as abnormal until lunging in the other direction is also observed. However, deviations from these known asymmetric patterns can be used and analyzed to help locate or confirm lameness to a particular limb.

On soft surfaces the outside limbs are less efficient at turning and propulsion because the foot digs into the soft surface as the limb is pushing off the ground. When the outside forelimb is pushing off, the horse is essentially changing direction. On soft ground that "gives way," changing direction is less efficient. Therefore, on soft ground, the head is thrown upward during pushoff of the outside forelimb, assisting the horse in changing direction toward the inside of the circle. This might appear as an outside forelimb pushoff-type lameness to some observers. Similarly, pushoff of the outside hindlimb is less efficient, but this causes the pelvis to rise less, making the horse appear to have an outside hindlimb lack of vertical propulsion. Also, the pelvis, tilted toward the middle of the circle of movement, falls less during weight-bearing of the inside hindlimb, mimicking an impact-type hindlimb lameness. If using pelvic rotation instead of VPM to detect hindlimb lameness, these characteristics would appear as "hip hike" on the inside hindlimb and "hip dip" (if this can indeed be seen) on the outside hindlimb. The veterinarian must then determine if the inside hindlimb "hip hike" and the outside hindlimb "hip dip" are the same lunging in the other direction.

On hard surfaces, asymmetries of vertical head and pelvic movement are primarily related to rotation of the torso with decreased downward movement of the head and pelvis and decreased upward movement of the pelvis during and after inside forelimb and hindlimb stance (or "hip hike" and "hip dip" on the inside hindlimb). This might appear as inside forelimb and hindlimb lameness to an observer evaluating vertical movement of the head for forelimb lameness and vertical movement of the pelvis or rotation of the pelvis for hindlimb lameness.[41,46,47]

These characteristics of asymmetry should be considered when evaluating horses for lameness at the lunge. Also, it has been shown that even normal horses trained to lunge will frequently move in one direction differently than in the other direction.[8,46] This increased variability makes detecting lameness at the lunge more complicated compared to straight-line evaluation. Disagreement between veterinarians of the existence, side, and amplitude of lameness observed during the lunge is higher than that for evaluation of lameness in the straight line.[26] However, if horses show consistent lameness during a straight-line trot, they will most commonly show lameness in the same limb during the lunge on the same surface and rarely only in the opposite side limb.

EVALUATION OF LAMENESS UNDER SADDLE

Some veterinarians believe that a lameness evaluation that does not include evaluation under saddle is incomplete. Certainly, evaluation for lameness under circumstances and in surroundings that are far removed or different from the usual and expected activity of the horse may fail to expose the true abnormality, adversely affecting performance of the horse. In some environments, for example, in a large arena with an appropriate surface, evaluation for lameness under saddle is easier and in many cases preferred. This is especially true for some breeds that do not trot regularly unless under saddle. However, evaluation for lameness under saddle is not always practically available to the veterinarian, and it should not be expected that lameness under saddle would always be worse or more visible than lameness without a rider. Although the extra weight and back extension may induce more pain,[16] the influence of the rider and excitement of the horse may serve to mask lameness.[33] Rider position and activity can also create artifactual (i.e. "false") lameness that will interfere or confuse veterinary examination for clinical abnormality.

A rider properly sitting the trot, in synchrony with the horse's vertical torso movement, will create the least artifacts on the observed lameness. A rider posting (performing a rising trot) moves forward and upward when one forelimb (usually the inside limb if riding in circle) is weight bearing and then backward and downward when the same-side hindlimb is pushing off. The effect of the upward and forward movement is increased weight-bearing on one forelimb, which, if that limb is painful, could worsen the perceived lameness in that limb. However, the backward and downward movement during pushoff of the same-side hindlimb (the inside hindlimb when riding in a circle) will frequently blunt or reduce the expected pelvic rise. This effect will tend to create what appears to be a lack of opposite hindlimb propulsion, a false lameness in a normal horse.[47] Effects of posting and moving in a circle are frequently additive and, depending on the

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surface characteristics, may either exacerbate or mask existing lameness when the rider posts while riding in small circles. Thus a rider posting on the right forelimb riding to the left may have what appears to be a left hindlimb lack of pushoff lameness (or "hip dip"). If the horse has preexisting left hindlimb pushoff lameness, this lameness will be exacerbated. If the horse has a preexisting right hindlimb pushoff lameness, this lameness will be reduced or masked. All these findings should be considered when evaluating horses for lameness under saddle. As with lunging, if a horse displays lameness consistently without a rider, it will usually display lameness in the same limb with the rider, and it is uncommon for lameness to be displayed in only a different limb.

Hindlimb and concurrent hindlimb and forelimb lameness has also been shown to be associated with saddle slip, slightly more frequently toward the lame than the non-lame or less lame side. Saddle slip increased when lame horses were ridden in circles and when the rider was posting.[21–23]

SUBJECTIVE SCORING METHODS

The AAEP Lameness Scale

The American Association of Equine Practitioners (AAEP) lameness scale is a subjective scoring method used to describe lameness (Table 2.3). Consistency, and to some extent, severity, of lameness is assessed by the ability to observe lameness under different conditions. It is an accurate but imprecise (there are only five grades) method to assess severity of lameness. Lameness amplitude can significantly change but remain within a single grade. Practitioners use the AAEP scale primarily as a communication tool that estimates overall lameness severity and consistency.

In the United Kingdom a scale from 1 to 10, which is basically the same at the above AAEP scale, but splitting each grade into two separate categories, is used (Table 2.4).

Table 2.3. American Association of Equine Practitioners Lameness Scale.

Grade	Description
0	Lameness not perceptible under any circumstances.
1	Lameness is difficult to observe and is not consistently apparent, regardless of circumstances (e.g. under saddle, circling, inclines, hard surface, etc.).
2	Lameness is difficult to observe at a walk or when trotting in a straight line but consistently apparent under certain circumstances (e.g. weight-carrying, circling, inclines, hard surface, etc.).
3	Lameness is consistently observable at a trot under all circumstances.
4	Lameness is obvious at a walk.
5	Lameness produces minimal weight bearing in motion and/or at rest or a complete inability to move.

Table 2.4. United Kingdom Lameness Scale

Grade	Description
0	Not lame
1–2	Lameness hard to detect at walk or trot
3–4	Lameness barely detectable at walk, easy to see at the trot
5–6	Lameness easily detectable at the walk
7–8	Hobbling at the walk, unwilling to trot
9–10	Non-weight bearing

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OBJECTIVE ASSESSMENT OF LAMENESS

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The standard of practice to detect and assess severity of lameness in horses is observing the horse in motion with the naked eye and then scoring using a discrete scale, for example, the AAEP or UK lameness scales/grades. This is sufficient for some cases. However, clinical studies strongly suggest that detection and evaluation of lameness in horses using the naked eye is insufficient in many cases, especially when the severity of lameness is mild.[22,25,30,33,45] Multiple limb involvement and compensatory movement in the opposite half of the body, and observation of different movement parameters, contribute to variability in assessment. Agreement, even between experts, for the detection of mild lameness between AAEP grades 1 and 2 or for picking the most affected limb is only slightly above chance.[30] The most likely explanation for this is limited temporal resolution of the human eye to detect fast events and small amounts of asymmetry in the movement of the horse.[51] Furthermore, unblinded subjective assessment is predisposed to bias.[1] Objective assessments of lameness by precise and accurate measurement of ground reaction force or asymmetry of movement at high sampling rates can be used to mitigate these limitations.

MEASUREMENT OF GROUND REACTION FORCES (KINETICS)

Most diseases that cause lameness do so because of pain during weight-bearing. A horse will bear less weight on an affected limb to decrease pain, resulting in decreased ground reaction force, which can be objectively measured. The stationary force plate, force-measuring treadmill, force-measuring devices attached to the bottom of the hoof, and pressure-sensitive mats have been used to measure ground reaction forces in moving horses.[4,5,8,9,17,18,20,23,26–29,35,46,47,49,50,53,68,71,73–75] The stationary force plate is the most commonly used and cited method.

During an evaluation for lameness with the stationary force plate, the horse is moved over the force plate so that at least one, and preferably just one, hoof strikes the force plate completely within the confines of the surface of the force plate (Figure 2.127). Ground reaction forces can be measured in all three directions—vertical, horizontal, and transverse (Figure 2.128). Decreased vertical ground reaction force and, to some extent, altered horizontal ground reaction forces are most often associated with lameness in the horse. Lower peak forces and impulse (area under the force vs. time curve) are associated with increasing severity of lameness. It is also likely that the shape of the vertical and horizontal ground reaction force curves contains information relevant to determining timing of lameness and differentiating whether pain is maximum during limb impact, in the first half of stance or during pushoff, in the second half of stance.[5,20,75] It has been shown that horses with an impact hindlimb lameness, or less movement of the pelvis downward, have decreased peak vertical ground reaction force, decreased deceleratory horizontal ground reaction force, and decreased vertical ground reaction impulse in the first half of stance.[8] Horses with reduced hindlimb propulsion, or decreased pelvic rise after hindlimb pushoff, have decreased vertical ground reaction impulse in the second half of stance and a conversion of vertical force to acceleratory horizontal ground reaction force. In other



Figure 2.127. Stationary force plate set for evaluation of lameness in horses. White line outlines approximate area of top of force plate, which is embedded into the ground and covered with a rubber mat to prevent shying. *Source*: Courtesy of Dr. Michael Schoonover at Oklahoma State University.

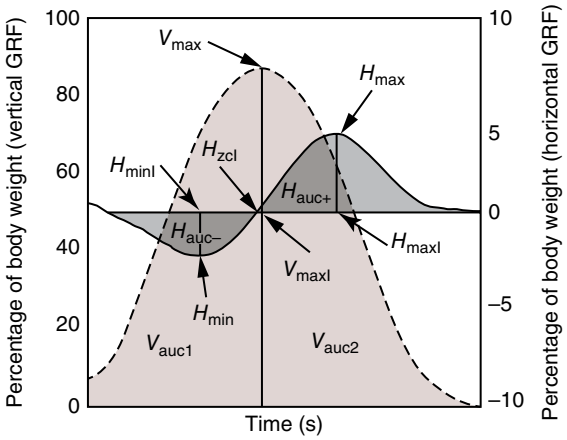


Figure 2.128. Stationary force plate data (from a single hoof strike) of relevance for determination of lameness. V_{max} = peak vertical ground reaction force. V_{auc1} = vertical impulse (force × time) in first half of stance. V_{auc2} = vertical impulse (force × time) in second half of stance. H_{min} = peak negative (deceleratory) horizontal ground reaction force in first half of stance. H_{max} = peak positive (acceleratory) horizontal ground reaction force in second half of stance. H_{auc-} = deceleratory horizontal impulse. H_{auc+} = acceleratory horizontal impulse. V_{maxl} = time index of V_{max}. H_{minl} = time index of H_{min}. H_{maxl} = time index of H_{max}. H_{zcl} = time index of horizontal ground reaction force cross over from deceleratory to acceleratory force. No transverse forces are shown.

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words, the horse pushes off with less vertical force but with more horizontal ground reaction force in the second half of stance.

The stationary force plate is a precise and accurate instrument. Variability between trials in controlled conditions is low (coefficients of variation below 10%), and sensitivity is high enough to detect subclinical lameness.[27] However, acquiring data requires controlled conditions. The hoof must strike completely within a relatively small area, often requiring multiple attempts. Speed of movement is controlled, both to increase the chance of successful hoof strike and to decrease variability between hoof strikes. The stationary force plate is considered by some to be a gold standard for objective lameness evaluation in horses, but the controlled conditions required for acquiring consistent results make it unlikely to be adopted for routine use in clinical practice. Although it varies with breed, the forelimb of horses that are not lame trotting across the force plate at moderate speed will strike the force plate with vertical force approximately equal in magnitude to 100% of its body weight.[4] The hindlimb of normal horses trotting across the force plate at slow to moderate speed will strike with vertical force equivalent to about 90% of body weight.[8] Induction of "subtle," mild, and moderate lameness decreased vertical ground reaction force in horses trotting on a treadmill by 4%, 9%, and 24%, respectively.[72] Horses with AAEP grade 4 lameness will strike the forelimb with a force as low as 50% of body weight.

MEASUREMENT OF MOVEMENT (KINEMATICS)

Kinematics is the measurement and study of movement. Limb and torso pain alters the normal movement of the horse. If pain predominates in one side of the body, the normal symmetric movement between right and left parts of the stride will become asymmetric. Kinematics, like kinetics, can be used to quantify absolute movement measures that may correlate well with lameness. However, most applications quantify lameness by measuring the asymmetry of movement between left and right sides of the body. Many different motion parameters have been studied and used to detect and evaluate forelimb and hindlimb lameness in horses, including vertical movement of the torso (head bob, pelvic fall and rise),[14] stride and step length and timing,[13] pelvic rotation (hip hike and dip),[44,56] limb and hoof flight pattern,[13] and joint angle extremes and range of motion.[13] Asymmetric vertical movement of the torso, because it is more directly associated with vertical ground reaction force, is the most sensitive kinematic indicator of lameness.[14,16,31]

The body motion changes of lameness are more variable stride by stride than changes in ground reaction force. High stride-by-stride variability of lameness lowers the signal (lameness) to noise ratio, rendering it more difficult to detect small amplitudes of lameness or small changes in amplitude of lameness. Variability can be decreased by strictly controlling conditions of evaluation. Alternatively, or in addition, the adverse effect of stride-by-stride variation can be minimized by collecting data from a high number of contiguous (one after the other) strides. Despite the higher variability compared to the force plate, results of kinematic evaluation of lameness are generally more intuitive and easy to understand for the veterinary practitioner. The practicing veterinarian can more easily apply significant findings in kinematic studies of lameness in their standard lameness evaluation techniques.

The original kinematic technique was camera based. The horse is filmed while moving and body motion is quantified by analyzing trajectories of markers attached to the body of the horse (Figure 2.129). Computer



Figure 2.129. Marking of the horse with reflective spheres (head, right hoof walls, pelvis) for camera-based kinematic evaluation of lameness.

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assistance is used to track, record, segment (into strides), and extract relevant (to lameness) features from the trajectories of the marked body parts. Analysis of the trajectories, most likely the asymmetry of trajectory motion between right and left parts of the stride, can then be used to detect and measure lameness. Although there are many different commercially available camera-based systems, they all depend on unobstructed line-of-site light transmission. The camera-based kinematic technique is more sensitive than subjective evaluation for detecting asymmetry of motion because the sampling rate of the camera can exceed the temporal resolution of the unaided human eye by several orders of magnitude. In order to collect multiple contiguous strides at maximum spatial resolution, kinematic evaluation of lameness in horses is usually performed on the equine treadmill with the horse remaining within the cameras' field of view. Movement of the horse on a treadmill is different than overground with longer stance times, earlier placement of the forelimb, increased retraction of both fore- and hindlimbs, and decreased vertical movement of the hooves and withers.[11] Some lameness that is visible (or measurable) overground may not be seen (or measured) on the treadmill, and vice versa. Horizontal ground reaction forces on the limbs of horses, which are different when trotting on the treadmill, with increased braking (negative horizontal in the first half of stance) and decreased propulsive (positive horizontal in the second half of stance), may be the cause of this discrepancy. Newer kinematic systems with automatic calibration and tracking of markers and user-friendly software to collect and display results greatly simplify use of this technique, but the requirement for dedicated space and multiple cameras to collect multiple strides with adequate spatial resolution limits this practice to research centers and technologically advanced clinics. Most of the early investigations searching for the most sensitive and accurate indicators of pain-induced lameness were carried out using these systems, and it is expected that most of the research into development of models to study equine motion for many different reasons will continue with this type of technology.

Asymmetry of motion can be more readily measured using inertial sensors attached to the horse's body.[6–8,18,21,32,34–36,42,48,54,55,57,58,60,61,64–66,69] Sensor data is wirelessly transmitted to the evaluator, so spatial resolution does not decrease with distance of subject from the evaluator, and line-of-site requirements are relaxed. Motion data from multiple contiguous strides in an overground setting can be collected and evaluated. Wireless transmission of body-mounted inertial sensor data allows clinicians to objectively evaluate lameness in horses in a natural clinical environment. For these reasons, using body-mounted inertial sensors to measure lameness is more intuitive and practical than other methods. In one study inertial sensor-derived lameness measures agreed more closely with blinded subjective evaluation of experts than results from a stationary force plate.[18] In another study, body-mounted inertial sensors identified an induced lameness in horses trotting in a straight line before a consensus of expert evaluators performing full lameness evaluations.[45] The remainder of this section describes one system in use today.

The Q® with Lameness Locator®

The Q® (hardware) and Lameness Locator® (software) was specifically designed as an aid to the practicing equine veterinarian for detection and evaluation of lameness in horses. Equine veterinarians and engineers at the University of Missouri in collaboration with the Hiroshima Institute of Technology in Japan developed the technology.

The Q consists of three inertial sensors, a tablet PC for data collection, analysis, and archiving, a sensor battery charger, and accessories for attaching the sensors to the horse's body (Figure 2.130). The inertial sensors are attached to the head, right forelimb pastern, and dorsum of the pelvis between the tuber sacrale. Each sensor is 1.5 inches by 1.25 inches by 0.75 inches and weighs 28 g. Vertical accelerations of the head and pelvis and angular velocity of the right distal forelimb are measured and wirelessly transmitted (Bluetooth Class 1) in real time to a handheld tablet computer. Range of transmission is up to 100 m, although larger transmission distances can be attained with additional Bluetooth repeater technology or with more sensitive or directional antennas. Sensor data (acceleration) is algorithmically converted to vertical position relative to the ground, which is the data of interest to the evaluator. Additional custom-designed algorithms are used to detect and quantify forelimb and hindlimb lameness when the horse is moving in a symmetrical gait, which is usually the trot. Proper trotting strides are automatically detected by the software when the horse is moving. Non-trotting strides are automatically extracted and discarded before analysis. An additional sensor can be attached to the lower waist of the rider to evaluate rider activity, to determine if the rider is sitting or posting correctly, and to assess how rider activity affects the measurement of lameness. A new research version that tracks movement with 9 degrees of freedom and allows determination of acceleration in the global reference frame is available, allowing flexibility in placement of sensors (number and location) for further kinematic research. Automatic Interpretation and Degree of Evidence (AIDE) algorithms, developed from studies of induced lameness and from contemporary clinical cases, for straight-line evaluation, lunging, flexions, effect of blocking and treatment, under-saddle evaluation, and compensatory lameness, simplify interpretation of kinematic values related to lameness for the practitioner. Mobile Q, with collection of data on smartphone devices and analysis in the cloud, is currently in development.

Lameness Locator® algorithms were developed from previous kinematic research and clinical testing. Best sensor type and locations were determined by data mining of accumulated motion data from groups of sound horses, horses with naturally occurring lameness, and horses with various models of induced lameness. Vertical head and pelvic acceleration are converted to vertical position in space and separated into components by custom error-correction algorithms. Random and nonperiodic movement is discarded, minimizing the negative effect of asymmetry due to nonclinical reasons. Remaining periodic movement is separated into movement due to lameness at stride frequency (the lameness component) and natural vertical motion at twice stride

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Figure 2.130. The Q™ (hardware) with Lameness Locator® (software) on standing horse. (A) Head accelerometer attached to head bumper. (B) Right forelimb gyroscope attached to dorsal pastern in pouch. (C) Pelvic accelerometer attached with foam pad and rubberized grip device. (D) Instrumented horse in motion.

frequency (the expected vertical movement).[2,52] Outlier data is detected and discarded. Simple, experimentally determined reference range estimates between likelihood of "lameness" and "not lameness," and the strength ("weak," "moderate," and "strong") of this evidence, are presented to the veterinary user. Results are standardized to normal expected vertical movement (the periodic component of vertical movement at twice stride frequency) to accommodate for large differences in body size or natural "boundingness" in trot. Lameness is detected and quantified by reporting means and standard deviations of maximum and minimum height differences of the head (for forelimb lameness evaluation) and pelvis (for hindlimb lameness evaluation) position. Location of lameness to limb (or limbs) and timing of peak lameness within the stride phase of a limb are determined by the association of head and pelvic movement to movement (angular velocity) of the right forelimb. All assessment of amplitude of lameness is generated by data from the head and pelvis sensors, and the right forelimb sensor is simply a stride event marker.

Lameness evaluation results are reported in a graphical display that depicts amplitude of impact and propulsion asymmetry in each stride. Lameness can be evaluated with the horse trotting in a straight line, while lunging (at the trot), and trotting under saddle. Response

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to flexion tests and blocking (or other treatment) can be quantified. Compensatory or multiple limb lameness patterns can be determined by studying the distribution of impact and pushoff asymmetry in all four limbs. At the time of this writing, Lameness Locator® is being used in >300 private practice and university veterinary teaching hospital sites around the world. The Q® and Lameness Locator® are sold only to licensed veterinarians. Using data mining techniques on inertial sensor data from large numbers of cases with definitive diagnoses opens the possibility of determining likelihood of site of lameness within the limb derived from inertial sensor results.

Measuring Forelimb Lameness

Forelimb lameness is reported in a graphical display and by calculating lameness values (Figure 2.131). The forelimb lameness display provides a qualitative description of the forelimb lameness by representing each analyzed stride as a single ray directed outward from the plot origin. Each ray on the plot is a stride. The length of the ray is representative of the amplitude of asymmetric head motion for that stride. The direction of the ray on the plot represents the side and timing of lameness for that stride. Using polar coordinate terminology, rays in quadrant 1 (upper right) represent lameness in the right forelimb during the first half of stance, from impact to midstance; rays in quadrant 2 (upper left) represent lameness in the right forelimb during second half of stance, midstance to pushoff; rays in quadrant 3 (lower left) represent lameness in the left forelimb during the first half of stance; and rays in quadrant 4 (lower right) represent lameness in the left forelimb during the second half of stance.

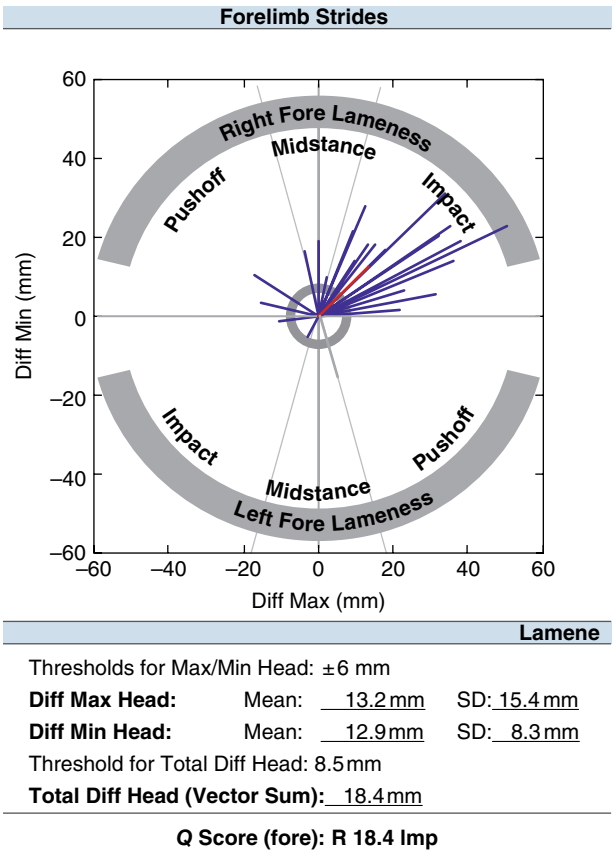


Figure 2.131. Output of Lameness Locator® indicating right forelimb impact lameness. Predominance of rays pointing in quadrant 1 (upper right) have Diff Max Head and Diff Min Head > reference range of ±6 mm and Vector Sum or Total Diff Head > 8.5 mm. Standard deviations of Diff Min Head less than mean Diff Min Head indicating consistent right-sided lameness.

The lameness values provide a more quantitative description of the forelimb lameness. Forelimb lameness is quantified by calculation of the difference in maximum head position between left and right forelimb strides (Diff Max Head) and the difference in minimum head position between left and right forelimb strides (Diff Min Head). Diff Max Head and Diff Min Head are calculated for every stride selected for analysis, and the mean and standard deviation over all strides are reported. The signs of Diff Max Head and Diff Min Head determine side and timing of lameness. The approximate reference range for consideration of "lameness" for these values is ±6 mm. Because forelimb lameness results in a combination of both differences in maximum and minimum heights of the head, the total overall forelimb lameness is a calculated sum called Total Diff Head or Vector Sum (VS). Because Diff Max Head and Diff Min Head take on both positive and negative values depending on side and timing of lameness, the total amplitude of lameness is a vector sum of both measures. Thus, $VS = \sqrt{(\text{Diff min Head})^2 + (\text{Diff max Head})^2}$. Since the approximate reference ranges of Diff Max Head and Diff Min Head are ±6 mm, the approximate reference range of $VS = \sqrt{(6)^2 + (6)^2}$, or 8.5 mm. These reference ranges are actually confidence intervals of the inertial sensor measures that were determined experimentally in a group of >125 horses evaluated for lameness subjectively by at least three experienced veterinarians when the average AAEP score was 0. Variation of lameness side and amplitude, which is related to strength of evidence, is assessed along with amplitude of lameness. The AIDEs used in this system remind the user of the statistical nature of these reference ranges with language indicating weak or strong evidence and reminders to repeat trials when measures are within reference range. Some horses with measures below these reference ranges, but highly consistent, may still have clinical disease. Clinically relevant reference ranges may be higher or lower depending on intended use and specific clinical situation.

When the lameness evaluation is restricted to relatively slow speeds with the horse being led by a handler restrained by a lead rope, the majority of forelimb lameness is measured during the first half of stance (impact to midstance). This is likely because at slow speeds the horse primarily uses the forelimb as a strut with little active forelimb pushoff.

Measuring Hindlimb Lameness

Hindlimb lameness is reported in a graphical display that depicts deficiency of right and left hindlimb impact (first half of stance) or pushoff (second half of stance; Figure 2.132).

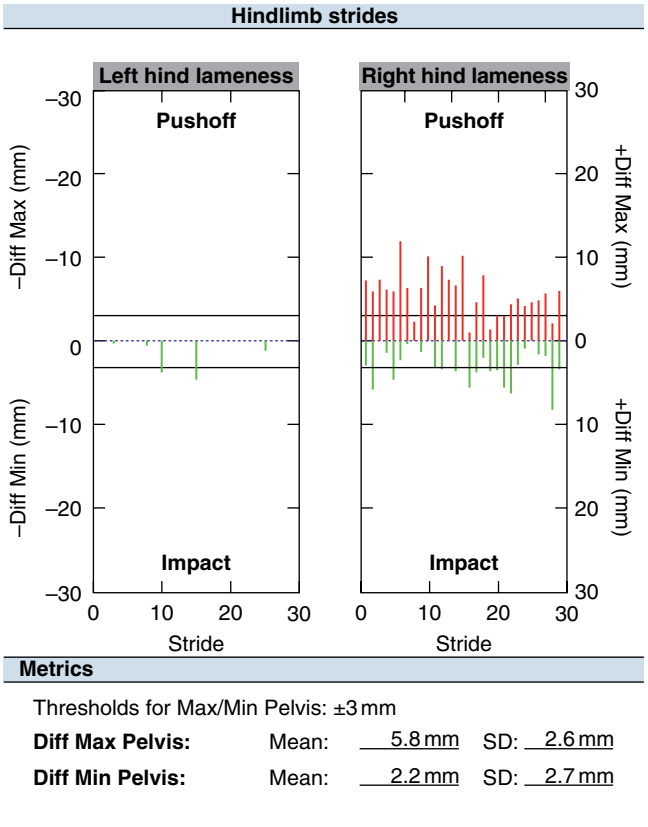


Figure 2.132. Output of Lameness Locator® indicating right hindlimb pushoff lameness. Most red rays (Diff Max Pelvis) are on right side. This indicates most Diff Max Pelvis are + in sign, i.e. the pelvis is thrust up less after pushoff of right hindlimb compared with left hindlimb. Diff Max Pelvis > reference range of ±3 mm. Standard deviation of Diff Max Pelvis less than mean of Diff Max Pelvis, indicating consistent lameness.

The hindlimb lameness display is a qualitative description of the hindlimb lameness. The left side of the display represents qualities of the left hindlimb function. The right side of the display represents qualities of the right hindlimb function. Each vertical line on the display moving from left to right is a measure of either the deficiency of impact (pelvic fall) or the deficiency of pushoff (pelvic rise) for that stride. The length of the line is representative of the amplitude of lameness.

Display of hindlimb lameness, with two subplots, one representing function of the left and the other representing function of the right hindlimb, is different from display of forelimb lameness, as one plot combining Diff Max Head and Diff Min Head. Why? The displays are different because the biomechanical model of VPM with hindlimb pain is different from that of vertical head movement for forelimb pain. VPM is modeled as a single free body falling less due to factors occurring in the first half of stance, which are different from factors causing less pelvic rise during pushoff of a hindlimb in the second half of stance. Diff Max Head and Diff Min Head are dependent quantities caused by the same thing (head moving upward at moment of peak pain), whereas Diff Max Pelvis and Diff Min Pelvis are independent quantities caused by different things.

The lameness values give a more quantitative description of the hindlimb lameness. Hindlimb lameness is quantified by calculation of the difference in maximum pelvic position between left and right hindlimb strides (Diff Max Pelvis) and the difference in minimum pelvic position between left and right hindlimb strides (Diff Min Pelvis). Diff Max Pelvis and Diff Min Pelvis are calculated for every stride collected, and the mean and standard deviation over all strides are reported. The signs of Diff Max Pelvis and Diff Min Pelvis determine side and timing of lameness. Positive values indicate right hindlimb lameness, and negative values indicate left hindlimb lameness. High Diff Max Pelvis absolute values indicate pushoff-type hindlimb lameness. High Diff Min Pelvis absolute values indicate impact-type hindlimb lameness. The approximate reference range for lameness for both measures is ±3 mm, which are confidence intervals of the measure when hindlimb mean subjective AAEP score = 0. These confidence intervals were determined and should be interpreted like the reference ranges of Diff Max Head and Diff Min Head for forelimb lameness.

Detecting Compensatory Lameness

In trotting quadrupeds a primary lameness in the front half of the body will sometimes cause compensatory movements in the back half of the body and vice versa, such that an apparent multiple limb lameness is present. Interpretation of these compensatory movements is sometimes referred to as the "law of sides." The first principle of the "law of sides" states that an apparent ipsilateral lameness, i.e. forelimb and hindlimb lameness on the same side of the body, is likely primary hindlimb lameness and a compensatory but false forelimb lameness. The second principle of the "law of sides" states that an apparent contralateral lameness, i.e. forelimb and hindlimb lameness on opposite sides of the body, is likely primary forelimb lameness and a compensatory but false hindlimb lameness.

Experimental studies have determined that the first principle of the law of sides is, for the most part, true.[38,40,41,70,72] Pain during stance in the hindlimb will cause the horse to shift weight forward onto the simultaneously weight-bearing contralateral forelimb, and the head will fall more than normal. Normal falling of the head during weight-bearing of the opposite forelimb will be less, giving the appearance of an ipsilateral forelimb lameness ("low on sound"). Although there is significant variability from horse to horse, which is likely dependent on the type of primary hindlimb lameness, even slight primary hindlimb lameness may cause compensatory movement in the head that mimics significant forelimb lameness. This suggests the possibility, depending on the sensitivity of detection, of missing the identification of the primary hindlimb lameness because of the more apparent compensatory but false forelimb lameness. The increased sampling frequency of the inertial sensors compared to the unaided human eye decreases the chance of this confusion.

The second principle of the "law of sides" is less true and less straightforwardly explained.[23,38,73,74] Primary forelimb lameness can cause compensatory movements in the vertical movement of the pelvis attributable to

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both hindlimbs such that false hindlimb lameness could be measured in both sides. Primary forelimb lameness causes the horse to shift its center of gravity slightly toward the back half of the body during the stance phase of the affected forelimb. This causes the pelvis to fall more in the contralateral hindlimb compared with the ipsilateral hindlimb, mimicking impact-type lameness in the opposite hindlimb, ipsilateral to the primarily lame forelimb. However, the horse moving forward will push off less from the opposite/diagonal hindlimb (contralateral to the forelimb with primary lameness) to lessen shifting of weight forward onto the primarily lame forelimb, causing the pelvis to rise less and giving the appearance of a contralateral pushoff-type hindlimb lameness. Thus, primary forelimb lameness may cause compensatory movements that mimic ipsilateral hindlimb impact-type lameness but contralateral pushoff-type hindlimb lameness. Generally the ipsilateral impact-type "false" hindlimb lameness is small and less common than the contralateral pushoff-type "false" hindlimb lameness. Compensatory pelvic movement patterns with primary forelimb lameness are regularly measured with the increased sensitivity of the inertial sensors, and these patterns are useful for helping to detect and evaluate forelimb and multiple limb lameness (Figure 2.133). Compensatory pelvic movements with primary forelimb lameness are not commonly observed subjectively.

Compensatory lameness should reduce when the primarily lameness is reduced with blocking. Compensatory lameness patterns are seen when the horse is trotting in a straight line and while lunging. During lunging, compensatory patterns are additive to directional patterns, sometimes exacerbating and sometimes masking multiple limb effects.

Known compensatory lameness patterns were described in the previous section. However, there may be other reasons for an apparent multiple limb lameness. Secondary lameness arises when a primary lameness causes weight shifting away from the primary limb to other limbs. If this weight shifting compensation overloads the other limb(s), supraphysiologic strain may occur, which could damage tissue or cause pain, or both. Although less is known about secondary lameness patterns (because it is difficult to study experimentally), certain patterns in trotting horses have been suggested based on clinical impression. In horses that trot, primary forelimb lameness will most likely overload the opposite forelimb or the opposite hindlimb, but it will unlikely overload the ipsilateral hindlimb. Primary hindlimb lameness will equally likely overload the contralateral fore- or hindlimb, but will unlikely overload the ipsilateral forelimb (Figure 2.134). Secondary lameness should not reduce immediately when the primary lameness is reduced with blocking. Secondary lameness, because it is caused by physiologic overload that may damage anatomic structures that span a length of the limb, for example, the suspensory apparatus or flexor tendons, is also more difficult to block completely with lower limb blocks, and frequently small or partial improvements are seen with successively higher blocks.

Following the logic of Occam's razor (the principle of parsimony), which states that the simplest explanation of competing explanations for a series of observations is usually the most correct, and our knowledge of compensatory (previous section) and secondary lameness patterns, one can make a reasonable and evidence-based guess as to which limb in a multiple limb lameness is the primary source of lameness. In a study of over 1200 horses evaluated for lameness, multiple limb lameness was measured in just over half (57%) of horses with lameness when trotting in a straight line. Therefore, when measuring lameness in horses actually presented for lameness, multiple limb lameness is the norm rather than the exception.

Considering both impact- and pushoff-type forelimb lameness together, but hindlimb impact and pushoff lameness as separate quantities, there are only eight possible "types" of multiple limb lameness:

1. Forelimb lameness with ipsilateral hindlimb lameness that is both decreased impact and decreased pushoff (Figure 2.135).

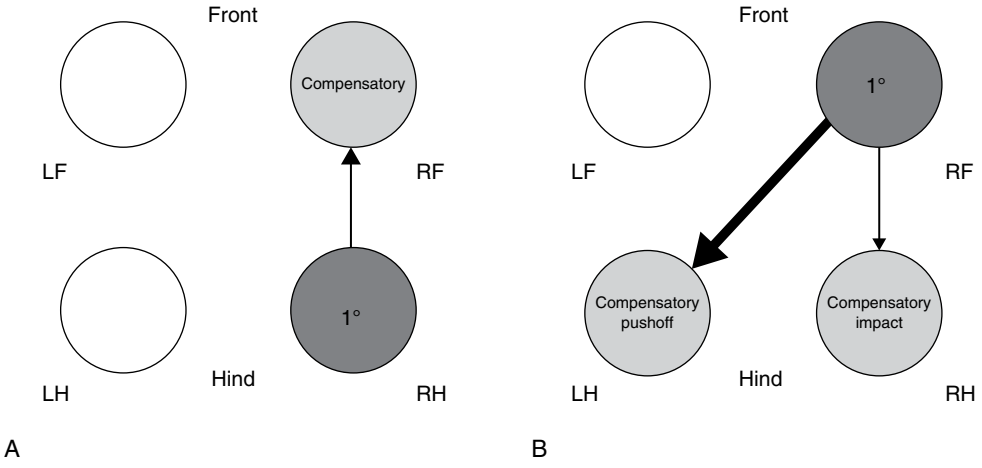


Figure 2.133. Known compensatory lameness patterns. (A) First part of the law of sides. Concurrent apparent ipsilateral forelimb and hindlimb lameness is usually a primary hindlimb lameness and an ipsilateral compensatory (false) forelimb lameness. (B) Second part of the law of sides. Concurrent forelimb and either compensatory contralateral hindlimb pushoff or ipsilateral hindlimb impact (or both) lameness. Compensatory contralateral compensatory hindlimb pushoff lameness is more common and usually more prominent.

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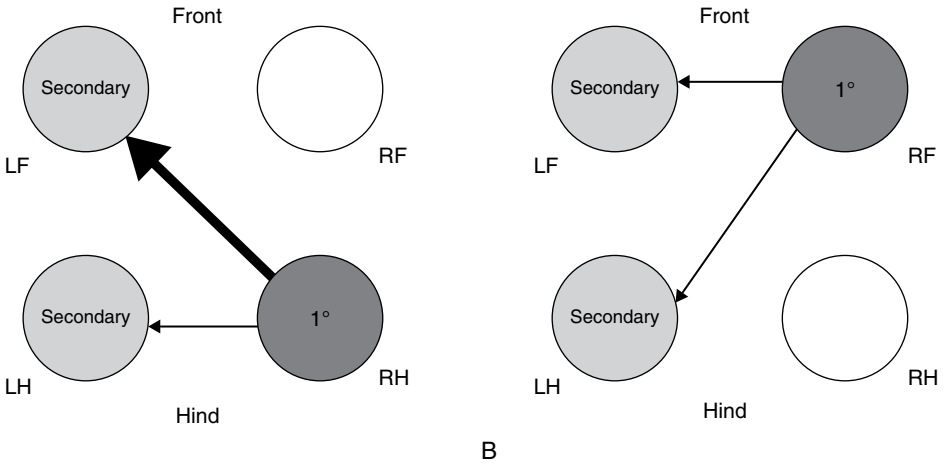


Figure 2.134. Suspected secondary lameness patterns. (A) Primary hindlimb lameness will most frequently overload opposite forelimb, less frequently overload the opposite hindlimb, and infrequently overload the ipsilateral forelimb. (B) Primary forelimb lameness will equally frequently overload the opposite forelimb and/or the opposite hindlimb, but infrequently overload the opposite forelimb.

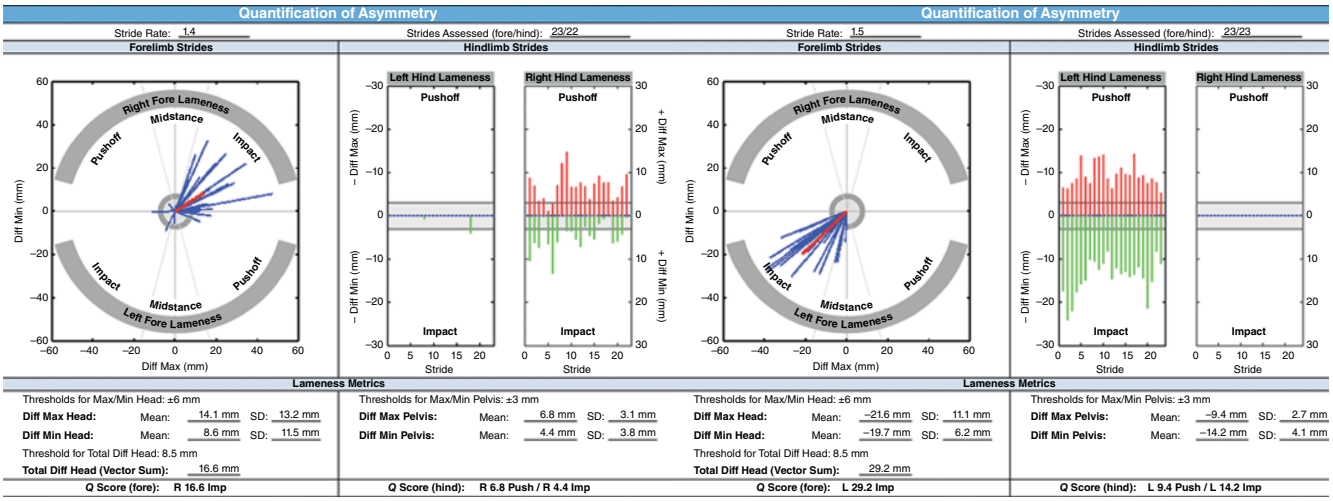


Figure 2.135. Lameness Locator® reports of multiple limb lameness. Left report: Right hindlimb pushoff and impact lameness and ipsilateral (right) forelimb lameness. Right report: Left hindlimb pushoff and impact lameness and ipsilateral (left) forelimb lameness. Both are examples of first part of the law of sides, which is primary hindlimb lameness with compensatory (false) ipsilateral forelimb lameness.

In about 700 horses measured with multiple limb lameness, this was the second most common type, occurring in about 19% of all initial evaluations of multiple limb cases. The simplest explanation of this type of multiple limb lameness is primary hindlimb lameness with compensatory forelimb lameness (the first principle in the "law of sides"). Horses with this type of multiple limb lameness had a high definitive diagnostic rate (82%) with 73% ultimately determined to have only a hindlimb diagnosis and another 14% having diagnoses in both hindlimb and front limb. Less than 11% ultimately were diagnosed with primary forelimb disease/abnormality.

2. Forelimb lameness with ipsilateral hindlimb lameness that is only decreased pushoff (Figure 2.136).

This type was seen in about 10% of all cases of multiple limb lameness. The simplest explanation of this type of multiple limb lameness is primary hindlimb lameness and compensatory forelimb lameness (the first principle in the "law of sides"). Horses with this type of multiple limb lameness had a (62%) definitive diagnostic rate, but with 66% ultimately determined to have only a hindlimb diagnosis and another 20% having both a hindlimb and a forelimb diagnosis. Only 11% ultimately were diagnosed with a primary forelimb lameness.

3. Forelimb lameness with ipsilateral hindlimb lameness that is only decreased impact (Figure 2.137).

This type was uncommon, occurring in less than 10% of all multiple limb lameness cases. Equally legitimate explanations for this type of multiple limb lameness are primary hindlimb lameness with compensatory forelimb lameness, and vice versa, or primary forelimb lameness with compensatory hindlimb lameness. This type of multiple limb lameness had a 78% definitive diagnosis rate, but 4% had only a

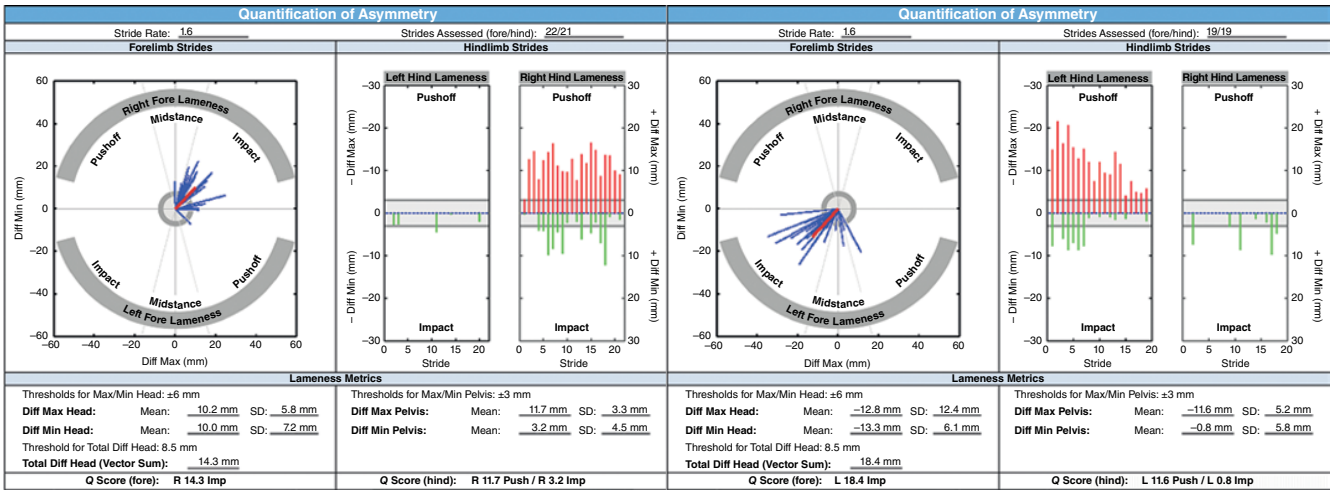


Figure 2.136. Lameness Locator® reports of multiple limb lameness. Left report: Right hindlimb pushoff only, no impact lameness, and ipsilateral (right) forelimb lameness. Right report: Left hindlimb pushoff only, no impact lameness, and ipsilateral (left) forelimb lameness. These are examples of the first part of the law of sides: primary hindlimb lameness with compensatory (false) ipsilateral forelimb lameness.

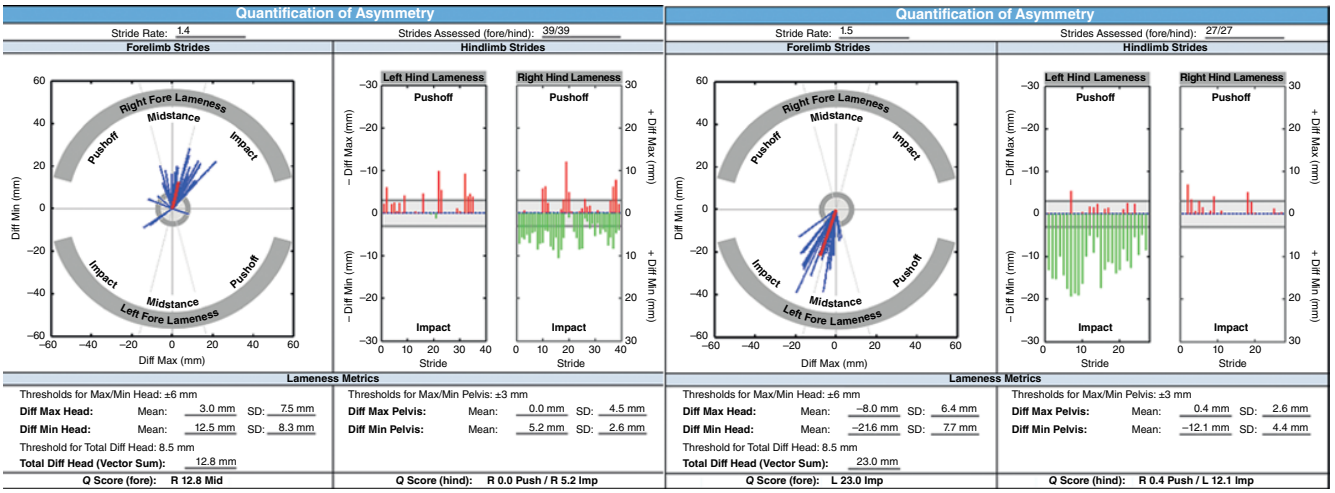


Figure 2.137. Lameness Locator® reports of multiple limb lameness. Left report: Right hindlimb impact only, no pushoff lameness, and ipsilateral (right) forelimb lameness. Right report: Left hindlimb impact only, no pushoff lameness, and ipsilateral (left) forelimb lameness. This multiple limb pattern can be equally explained by either the first or second part of the law of sides, either a primary hindlimb lameness with compensatory ipsilateral forelimb lameness or primary forelimb lameness with compensatory ipsilateral hindlimb impact lameness.

hindlimb diagnosis, 11% had both a forelimb and a hindlimb diagnosis, and 45% had only a forelimb diagnosis, about 1/3 being a bilateral forelimb lameness condition. Also, a relatively high percentage (9%) of these cases had diagnosis of abnormality not located within the limbs. When seeing this type of multiple limb lameness, it was equally likely for the primarily lameness to be in the forelimb or in the hindlimb.

4. Forelimb lameness with ipsilateral hindlimb lameness that is decreased pushoff and contralateral hindlimb lameness that is decreased impact (Figure 2.138).

   This type occurred rarely. It was seen in only 4% of all multiple limb cases. There is no easy, compensatory- or secondary-only explanation of this pattern. A complicated explanation of primary hindlimb lameness, with compensatory ipsilateral forelimb lameness, and secondary contralateral hindlimb lack of impact lameness is required to fully explain this pattern. However, this type of multiple limb lameness had a relatively high rate (71%) of definitive diagnosis, with 35% ultimately diagnosed with only a hindlimb problem, 25% with both a hindlimb and forelimb problem, 20% with only a forelimb problem (but bilateral in 100%), and 20% with a non-limb diagnosis. This type of multiple limb lameness pattern had the highest percentage of "other" diagnosis (problem found but not in the limbs).

5. Forelimb lameness with contralateral hindlimb lameness that is decreased pushoff and decreased impact (Figure 2.139).

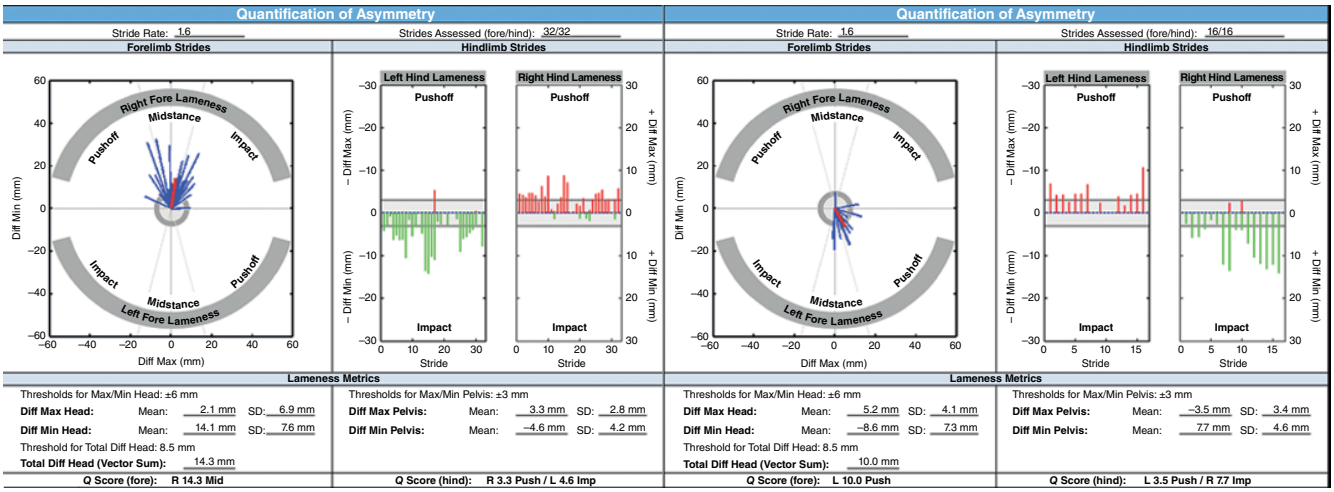


Figure 2.138. Lameness Locator® reports of multiple limb lameness. Left report: Right hindlimb pushoff, left hindlimb impact, and right forelimb lameness. Right report: Left hindlimb pushoff, right hindlimb impact, and left forelimb lameness. No simple single compensatory or secondary multiple limb lameness pattern completely explains this pattern. The best explanation is primary hindlimb lameness, with compensatory ipsilateral forelimb, and secondary contralateral hindlimb lameness exists.

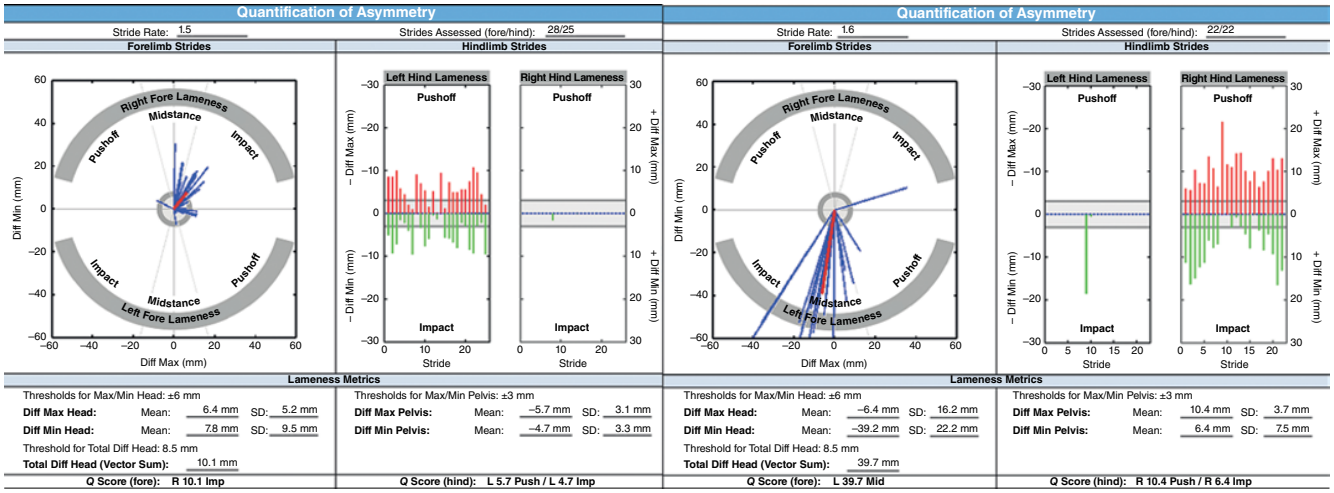


Figure 2.139. Lameness Locator® reports of multiple limb lameness. Left report: Left hindlimb pushoff and impact lameness and contralateral right forelimb lameness. Right report: Right hindlimb pushoff and impact lameness and contralateral left forelimb lameness. The best explanation for this type is primary forelimb with compensatory contralateral hindlimb pushoff lameness and secondary contralateral hindlimb impact lameness.

This type occurred in 17% of multiple limb cases. A possible explanation of this type of multiple limb lameness is primary forelimb lameness with compensatory (or secondary) contralateral hindlimb lack of pushoff and secondary (to the contralateral lack of hindlimb pushoff) ipsilateral hindlimb impact lameness. This type of multiple limb lameness had a high (71%) definitive diagnostic rate, with 66% having a diagnosis only in the forelimbs, with about ¼ (27%) being bilateral, 19% having a diagnosis only in the hindlimbs, and 13% having a diagnosis in both a hindlimb and a forelimb. Only about 2% had diagnosis in an area of the body other than in the limbs.

6. Forelimb lameness with contralateral hindlimb lameness that is only decreased pushoff (Figure 2.140).

This type occurred in 23% of all multiple limb cases, which is the most common multiple limb lameness type. The simplest explanation for this type of multiple limb lameness is primary forelimb with compensatory contralateral lack of hindlimb pushoff. This type of multiple limb lameness had a 70% definitive diagnosis rate, with 78% ultimately having only a forelimb lameness, with about 1 in 5 (22%) being bilateral, 13% having an ultimate diagnosis in both a forelimb and a hindlimb, and only 4% having a diagnosis only in the hindlimb. A diagnosis of "other," or an abnormality localized but not in the limbs, was present in only 4% of cases.

7. Forelimb lameness with contralateral hindlimb lameness that is only decreased impact (Figure 2.141).

This type occurred in less than 10% of cases. No compensatory-only pattern can explain this type of multiple limb lameness. Equally legitimate explanations are primary forelimb lameness and secondary

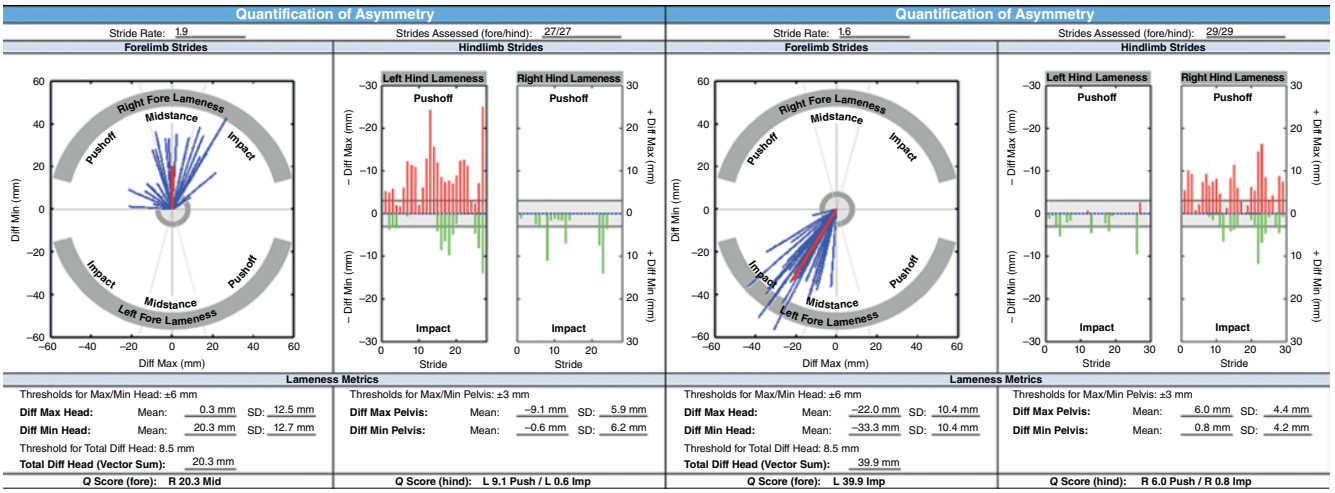


Figure 2.140. Lameness Locator® reports of multiple limb lameness. Left report: Left hindlimb pushoff only, no impact lameness, and contralateral right forelimb lameness. Right report: Right hindlimb pushoff only, no impact lameness, and contralateral left forelimb lameness. Best explanation is second part of the law of sides, or primary forelimb lameness with compensatory contralateral hindlimb pushoff lameness.

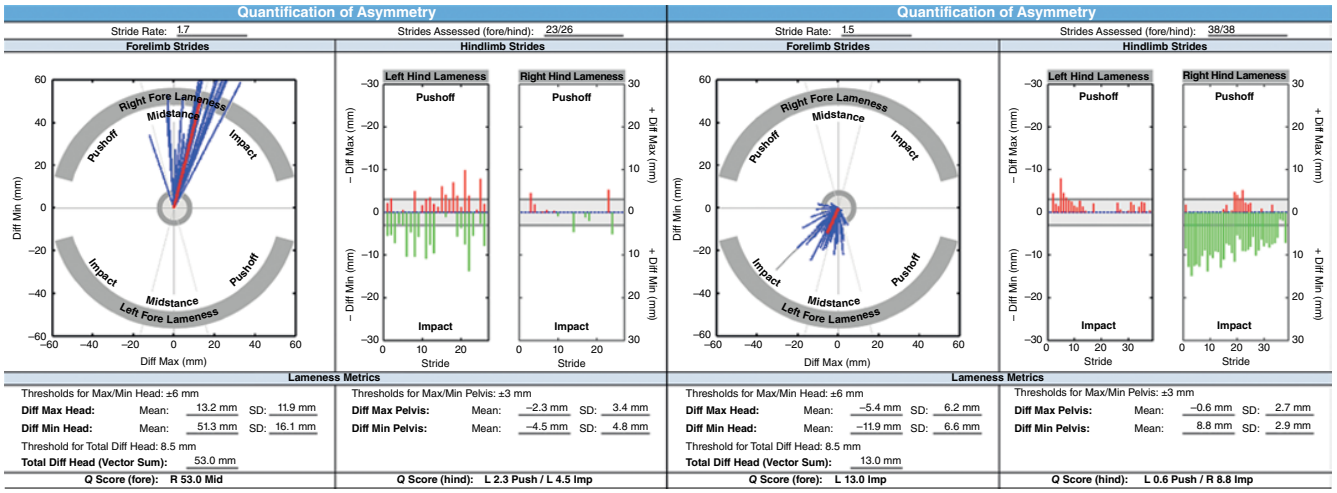


Figure 2.141. Lameness Locator® reports of multiple limb lameness. Left report: Left hindlimb impact only, no pushoff lameness, and contralateral right forelimb lameness. Right report: Right hindlimb impact only, no pushoff lameness, and contralateral left forelimb lameness. No compensatory lameness pattern explains this pattern. The best explanation of this type of multiple limb lameness is primary forelimb lameness and secondary hindlimb lameness or primary hindlimb lameness and secondary forelimb lameness.

hindlimb lameness and primary hindlimb lameness and secondary forelimb lameness. This type of multiple limb lameness had the lowest (54%) definitive diagnostic rate, with 75% ultimately having a diagnosis of only forelimb abnormality, with about 1 in 3 (31%) being bilateral, 11% that ultimately had a diagnosis in both a forelimb and a hindlimb, only 6% with an ultimate diagnosis in only a hindlimb, and an (8%) "other" (diagnosis not in the limbs) diagnostic rate.

8. Forelimb lameness with contralateral hindlimb lameness that is decreased pushoff and ipsilateral hindlimb lameness that is decreased impact (Figure 2.142).

This type occurred in about 7% of all multiple limb cases. It is easily explained by the second principle in the "law of sides," with contralateral hindlimb lack of pushoff and ipsilateral hindlimb lack of impact. This type of multiple limb lameness had a 71% definitive diagnostic rate. Ultimately 61% of cases had only a forelimb diagnosis, with 27% being bilateral forelimb lameness, only 3% with both a forelimb and a hindlimb diagnosis, 28% with a hindlimb only diagnosis, and less than 8% with a diagnosis of "other" (abnormality found but not in the limbs).

Flexion Tests

Lameness Locator® can be used to objectively quantify the effect of flexion tests. Reference ranges and confidence intervals established for trotting in a straight line and collecting at least 25 contiguous strides, however, are not applicable. The effect of flexion tests is usually dissipated after fewer (>25) numbers of strides, and the veterinarian usually evaluates the effects of flexion

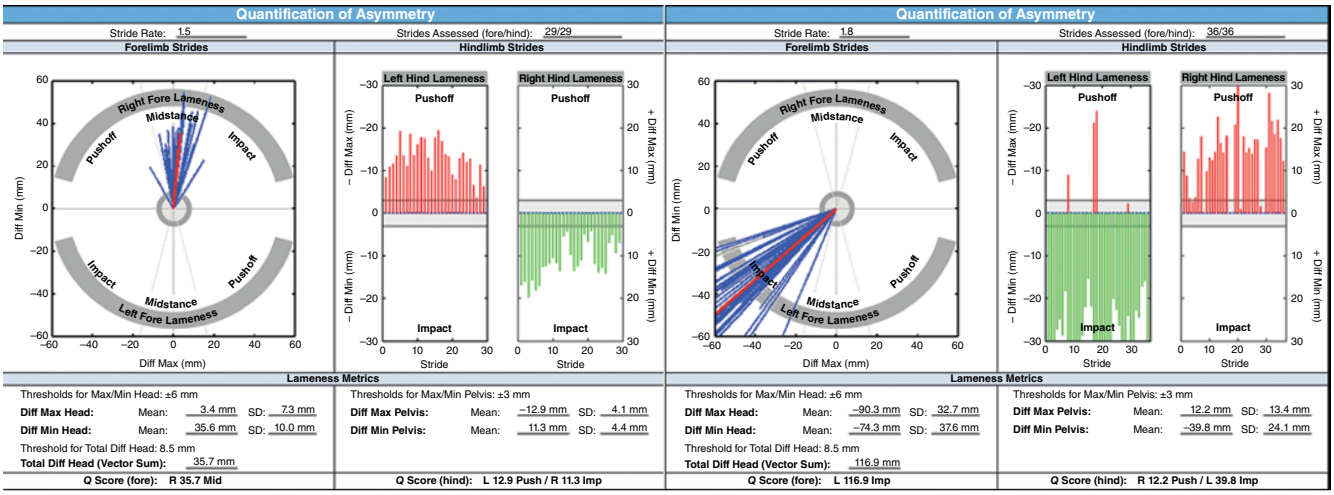


Figure 2.142. Lameness Locator® reports of multiple limb lameness. Left report: Left hindlimb pushoff, right hindlimb impact, and right forelimb lameness. Right report: Right hindlimb pushoff, left hindlimb impact, and left forelimb lameness. The best explanation of this type of multiple limb lameness is the second part of the law of sides, or primary forelimb lameness with compensatory contralateral hindlimb pushoff lameness and ipsilateral hindlimb impact lameness.

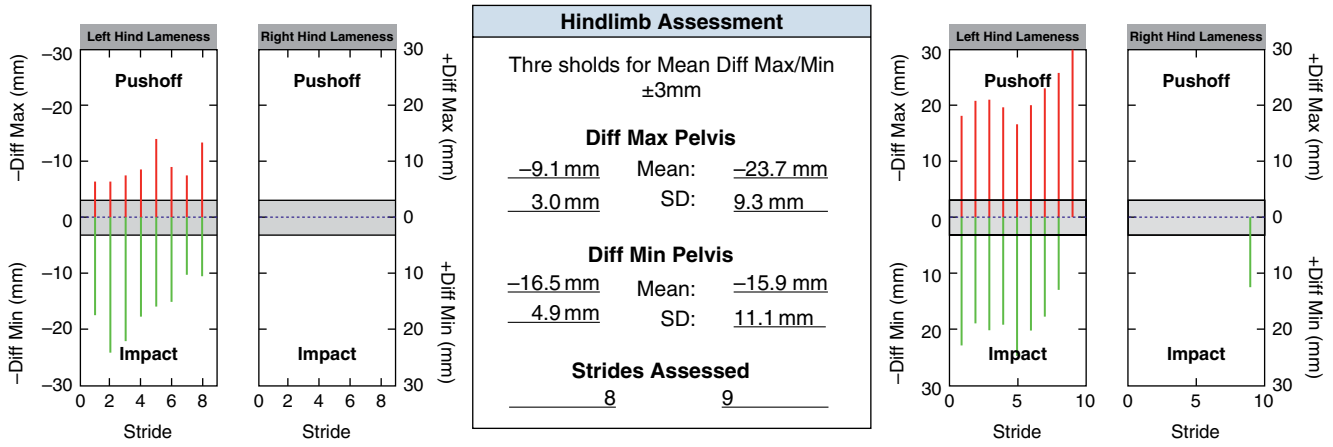


Figure 2.143. Lameness Locator® reports of before (left) and after (right) left hindlimb flexion. Absolute value of Diff Max Pelvis increases from |–9.1| to |–23.7| mm. This is a strong positive left hindlimb flexion test. Eight strides were collected as baseline before flexion and nine strides were collected after flexion.

tests by trotting the horse off in one direction only. Therefore, a baseline trot off in one direction without flexion should be collected as baseline before flexion. A positive response to flexion is then ascertained by simply comparing the amplitude of the lameness values for that limb before and after flexion (Figure 2.143). It is interesting that most hindlimb flexion tests are associated with an increase in Diff Max Pelvis, but not Diff Min Pelvis, or a decrease in hindlimb propulsion and not in a decrease in hindlimb impact.[43,67] In one study a minimum increase in Diff Max Pelvis of about 4.5 mm indicated a positive flexion test.[43]

Evaluation of Lameness Before and After Blocking

One of the most useful applications of using body-mounted inertial sensors is to more precisely and accurately evaluate the effect of blocking for reducing lameness.[56,64–66,69] This protects against the bias inherent in unblinded subjective evaluation. Bias can be positive, believing the lameness to be improved but in reality not, or negative, believing the lameness to be unchanged when in fact it is greater or lesser than before block. A desire for observing improvement in lameness after blocking is made stronger late in the day, after several blocks have been applied with no improvement, and especially in horses that are difficult or dangerous to block. On the other hand, partial improvements in lameness, or a change in the timing of lameness, may be mistaken for no effect. Objective measurements reduce these potential mistakes. Also, intraindividual (between horse) variation in lameness has been shown to be less than interindividual (within horse) variation of lameness; therefore smaller changes in amplitude of lameness and a quantitative percent improvement can be confirmed.

Lameness Locator® calculates percent improvement in lameness in two ways: (1) returning the amplitude of asymmetry (i.e. lameness) down to perfect mean symmetric movement and (2) returning the amplitude of lameness to within the reference range of asymmetry where even experts, performing full lameness evaluations, do not conclude that the horse is "lame." The first

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method is a very conservative assessment where 100% improvement is rare but small changes in amplitude of lameness are unlikely to be misinterpreted. The second method is a more liberal assessment with 100% improvement more common, but where small changes of amplitude of mild lameness may be seem exaggerated.

The first method is calculated as follows:

$$\frac{IS_{\text{before block}} - IS_{\text{after block}}}{IS_{\text{before block}}}$$

where IS *is inertial sensor measure*.

The second method is calculated as follows:

$$\frac{IS_{\text{before block}} - IS_{\text{after block}}}{IS_{\text{before block}} - IS_{\text{threshold}}}$$

where *threshold* is an experimentally determined value of the confidence interval of the inertial sensor measure in a group of horses thought not to be lame by experts during full lameness evaluations.

For example, a horse with a Diff Max Pelvis before block of +12 mm and a Diff Max Pelvis of +6 mm after block would have a 33.3% improvement in lameness ((12 – 8)/12 = 0.33…) using the first method and a 66.7% improvement in lameness ((12 – 8)/(12 – 6) = 0.66…) using the second method. Preference for method will depend on characteristics of the individual case, including beginning amplitude of lameness, stride-by-stride and (if known) trial-by-trial variation of lameness, and whether the lameness is a single or multiple limb lameness. It should be noted that any percent improvement above 100%, or that switches to the other limb, is interpreted as an elimination of lameness, when in reality some pain on weight-bearing may still be present. Also, at the time of this writing, this software considers any improvement below 25% improvement as no improvement.

There is some evidence that regional nerve blocks in non-lame horses do not appreciably change gait, do not make the horse appear subjectively to be lame, and do not significantly alter vertical ground reaction forces.[71] However, regional limb blocks in the front limb have been shown to prolong stance and the transition from braking to propulsion, supportive evidence that some proprioceptive deficit or loss is induced.[19,37] When using inertial sensors to evaluate blocking, users may measure subtle changes in lameness that are less or not apparent when relying on subjective evaluation.[45] For example, it is not uncommon, in fact it is usual, for lameness to get worse (increase in amplitude) when a limb is blocked in an area that is below or does not include the anatomic structure or area with pain. In other words, blocking a normal part of a painful limb will increase lameness. This effect is increased with successive blocks that do not include the focus of pain. Although blocking a normal limb in a horse without lameness has not been shown to significantly affect force plate results,[71] kinematic evidence suggests lower limb loading. If lameness increases with the blocking of normal limbs or with the blocking of normal parts of limbs displaying lameness, then one cannot expect a total removal of lameness when the causative focus is finally found. Having lameness increase after a block is a good sign that the evaluator has picked the correct limb as the primarily lame limb, but has yet to have localized the origin of lameness within the limb. In many studies blocking of the definitive primary source of lameness significantly decreased but did not eliminate the lameness, and the locomotion of the horse after diagnostic analgesia is not always identical to that of a non-lame horse. This suggests that it is more appropriate than to compare the after block evaluation to the evaluation done immediately before the block evaluated, instead of comparing to the trial before any blocks were applied.

When using inertial sensors to evaluate lameness, one will soon realize that the level of lameness, especially in the beginning of the evaluation, may not be stable. Lameness can increase, decrease, switch limbs, or change in timing at the beginning of a lameness evaluation. Beginning a series of blocking procedures before the lameness is stabilized risks misinterpretation and misdiagnosis. When using inertial sensors to evaluate response to blocking, it is recommended to first stabilize the lameness by some movement (walking) or exercise (light lunging on soft surface) and to avoid strong joint flexions or deep palpations of limb structures before assessing for lameness. Lameness can be considered stabilized when the amplitude (±8.5 mm for head and ±3 mm for pelvic movement asymmetry), limb(s) involved, and timing of lameness are consistent in back-to-back trials conducted within a few minutes of one another.

Evaluation of Lameness During the Lunge

Lameness Locator® can be used to objectively quantify lameness while the horse is trotting in a circle. Some lameness conditions in horses are more apparent when the horse is moving in a circle. Reference ranges and confidence intervals established for trotting in a straight line and collecting at least 25 contiguous strides, however, are not applicable. When the horse is trotting in a circle, the torso and limbs are tilted toward the center of the circle.[10] Depending on the horse, its speed of movement, the radius of the circle it is moving in, and the characteristic of the surface, this tilt can be significant. Also, on soft surfaces, the circular movement, centripetal forces, angle, and inclination of the limbs relative to the ground cause less efficient outside limb pushoff, and this creates normal asymmetric movement. However, the effects of tilt and surface characteristics can be individually predicted and accounted for.[59]

On soft surfaces, because the outside forelimb is less efficient during pushoff (the surface gives way to the forelimb pushing off), the horse will throw its head upward and inward to assist in turning. Likewise the outside hindlimb is less efficient at pushoff with the outside hindfoot digging into the surface. Subsequent pelvic rise from this less efficient pushoff is less than after pushoff of the inside limb. Also, the torso tilting toward the inside of the circle results in less downward fall of the head during inside forelimb stance and of the pelvis during inside hindlimb stance (decreased downward movement of the inside head and pelvis is independent of type of surface). So, on soft surfaces, there is an appearance of an outside forelimb pushoff-type lameness (right going to left and left going to right), an outside hindlimb pushoff-type lameness, and an inside hindlimb impact-type lameness. These patterns of asymmetry should be expected when lunging on soft surfaces (Figure 2.144).

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In contrast, on hard surfaces, torso tilt is the primary and most significant factor creating asymmetric head and pelvic vertical movement, so an inside forelimb impact-type forelimb (less head fall on the inside forelimb) and an inside hindlimb impact (less pelvic fall) and pushoff (less pelvic rise) lameness are frequently seen and should not be unexpected (Figure 2.145).

Surfaces with characteristics that are intermediate between hard and soft may have hybrid vertical head and pelvic height asymmetries. However, certain asymmetric movements are abnormal regardless of direction or surface characteristics. Impact-type lameness on the outside forelimb or pushoff-type forelimb lameness on the inside forelimb should not be expected on any surface. Impact-type lameness on the outside hindlimb should also not be seen in any direction or on any type of surface.

Evaluation of Lameness with Sedation

Examination of the horse in motion for lameness is more complicated in anxious and misbehaving horses. Random movement of the head, frequent and abortive gait transitions from trot to canter, rearing, jumping, attempting to bite the handler, all of these behaviors make it more difficult to detect small asymmetries (mild lameness) in vertical head and pelvic movement. Also, anxiety of the horse may mask the display of lameness. Lameness amplitude will vary more from stride to stride and confidence in results is sacrificed. This is a problem with both subjective and objective lameness evaluation. Some of the variable movement can be removed when using objective measurement methods with algorithms that extract and discard random movement, and presumably this is done also subjectively, and collecting data on higher numbers of multiple contiguous strides can mitigate some of the negative consequences of high stride-by-stride variability. However, stride-by-stride variability can be noticeably reduced with mild sedation, increasing the consistency of measurement and confidence in interpretation. Also, most studies have shown that mild sedation, although it may significantly affect limb movement parameters (e.g. stride length) sometimes used to assess lameness, does not as a rule artificially lower amplitude of head vertical movement asymmetry, and does not artificially lower amplitude of VPM asymmetry, as might be expected if sedation caused significant orthopedic analgesia.[3,15,61] Some protocols tested include 0.1–0.3 mg/kg xylazine and 0.025 mg/kg acepromazine. The author also believes that mild sedation in anxious or misbehaving horses allows are more thorough and particular limb and torso palpation. Of course, mild sedation may also facilitate blocking procedures in some horses. There are some circumstances of lameness evaluation (prepurchase examination, substance use before competition, owner preference) that do not allow the use of mild sedation, but if possible the clinician can mildly sedate without adversely affecting blocking results.

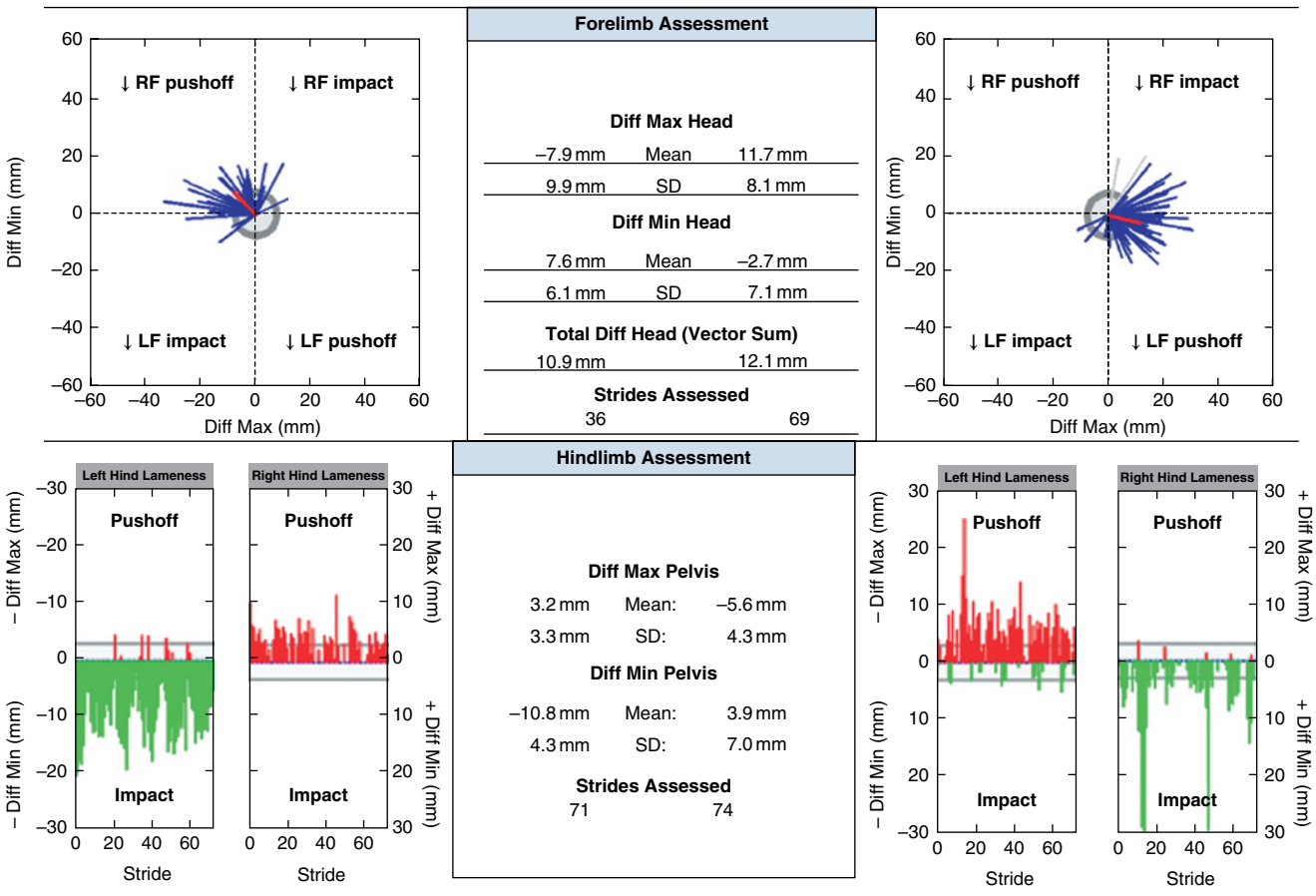


Figure 2.144. Lameness Locator® report of a non-lame horse lunging on a soft sand surface. Outside forelimb pushoff lameness, outside hindlimb pushoff lameness, and inside hindlimb impact lameness are measured.

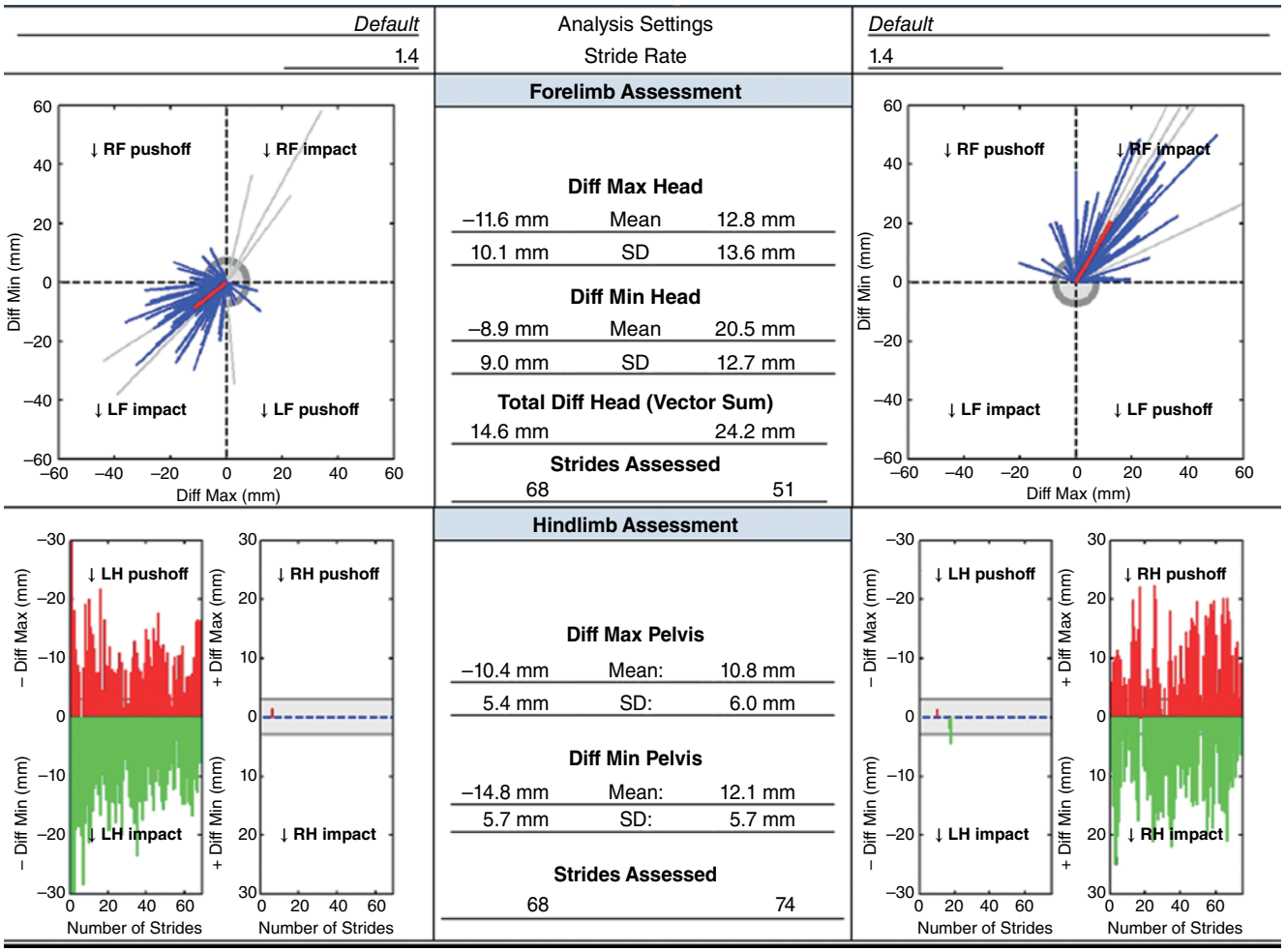


Figure 2.145. Lameness Locator® report of a normal horse lunging on a hard (asphalt) surface. Inside forelimb impact and inside hindlimb impact and pushoff lameness are measured.

Measuring Bilateral Lameness

No method that uses asymmetry as the measure of lameness can detect a perfectly bilateral lameness, and no measurement method that does not have a reasonable absolute known normal for the particular measurement for the individual horse can detect the existence of a perfectly bilateral pathological condition.[12] However, most bilateral conditions that cause lameness will have one side that is more affected than the other so that some lameness can be measured. Therefore, a bilateral lameness measured in a horse traveling in straight line will be an underestimation of the amplitude of true lameness. "Lateralization" of the lameness by measuring while lunging in both directions and then comparing both directional trial results can assist in estimating the true amplitude of lameness in both limbs, but until the pain in one limb is totally eliminated with blocking can the total lameness in one limb be determined. This fact should be realized when assessing treatments in studies using subjects with bilateral pathological conditions. In quadrupeds, the state of the two limbs in the other half of the body must also be determined.

Evaluation of Lameness Under Saddle

The Q® can be used to evaluate lameness under saddle with or without instrumentation of the rider with a separate inertial sensor. If used with the rider sensor, rider activity is detected, monitored, and assessed, and lameness is interpreted in light of this activity. If used under saddle without the rider sensor, rider activity should be known and noted by the evaluator so that any expected effects on lameness measurement can be properly interpreted. Rider activity affects lameness measurement under saddle.[39,62,63] Because there is no need for a handler to run back and forth multiple times leading the horse overground, assessment under saddle facilitates collection of data from multiple contiguous strides over longer periods of time (several minutes) compared to in-hand straight-line evaluation. Also, many horses behave better with less variable movement when under saddle. The collection of larger numbers of contiguous strides with lower stride-by-stride variability aids in detecting mild lameness or finding small changes in lameness severity. In most assessment, environments (arena and round pens) collecting unbroken under-saddle data for several minutes assumes that the horse will be ridden in a circular path.

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The Q® includes an optional Lameness Locator® module that collects and evaluates data from an inertial sensor attached to the rider at waist level in a belt with a pouch. Additional stabilization of the sensor to prevent independent wiggling, which creates motion noise, may be necessary. The rider inertial sensor measures and assesses vertical and horizontal (backward and forward) acceleration of the rider when the horse is trotting. The data is used in conjunction with that from the horse-mounted sensors to determine if the rider is sitting the trot, sitting the trot correctly, performing the rising trot (posting), and posting on the correct limb given the direction of motion selected for collection. The rider posting on the incorrect limb given the direction of movement may exacerbate lameness, but this is not currently evaluated.

Turning the rider sensor on initiates Lameness Locator®'s software for under saddle collection and analysis. Four synchronous channels of data (horse head, horse right forelimb, horse pelvis, and rider) are collected. Because it is assumed that the horse will be ridden in a circular path, and direction of movement affects vertical head and pelvic movement asymmetry, the user must assign direction of movement as either "ride to left" or "ride to right." Riding in one direction, the rider is asked to either sit the trot or post on the correct (outside) limb, or both, each for the desired duration. The desired trial length most conveniently includes one full rotation around a large arena or multiple rotations for riding in smaller circles. Strides collected with the rider sitting the trot and strides collected with the rider posting on the correct limb are automatically detected and separated. Improper technique when sitting the trot with either asymmetrical (right vs. left strides) or asynchronous (to the horse's vertical movement) vertical rider movement is flagged and will not be analyzed. Proper riding technique is verified, and the number of strides collected during each activity in one direction is displayed for analysis. Data collection for under-saddle evaluation while riding in the other direction is collected and analyzed in a separate trial. Riding to the left and riding to the right are then compared for each rider activity collected in a dual trial report similar to that for evaluating lunging in either direction (Figure 2.146).

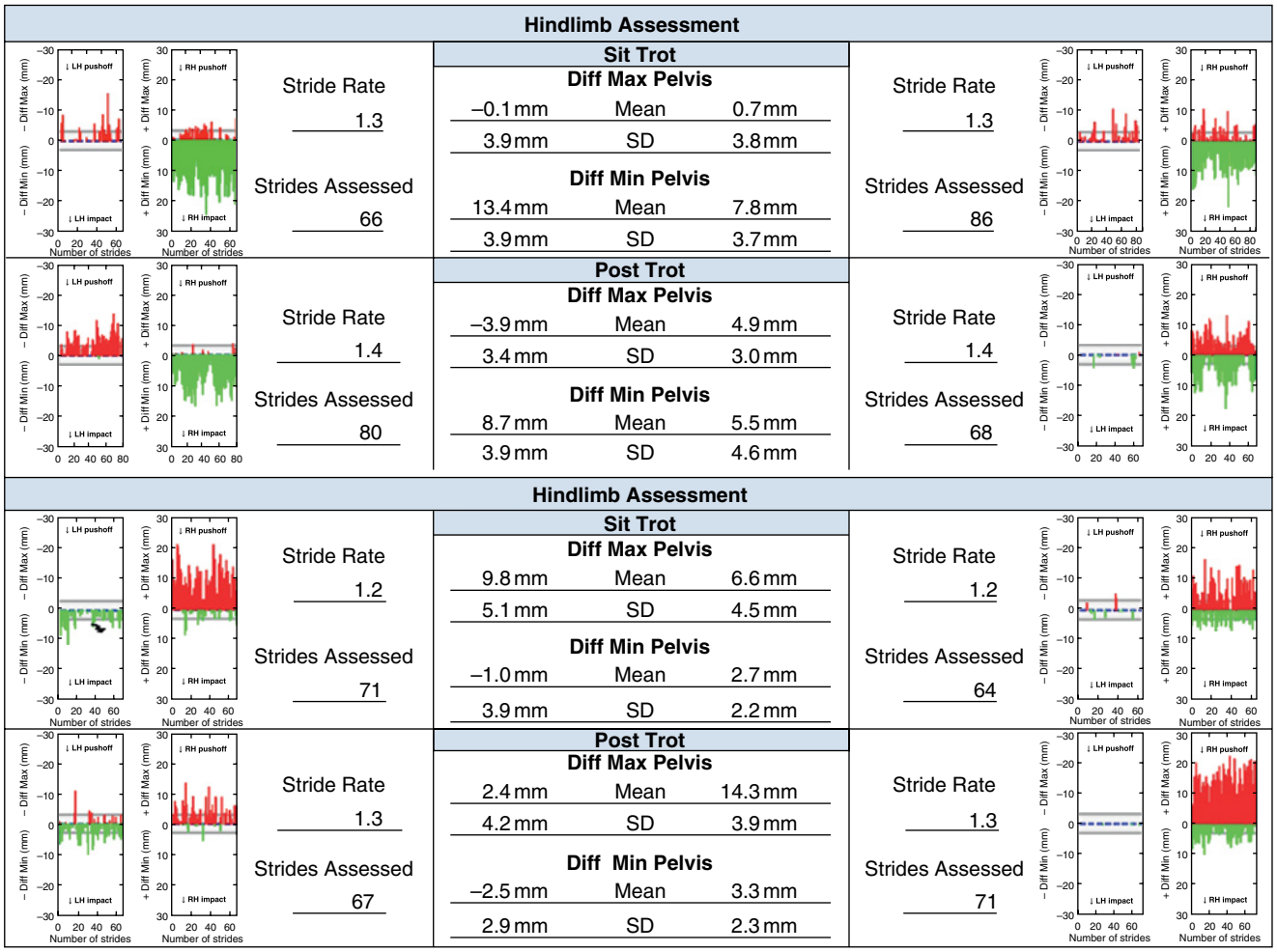


Figure 2.146. Lameness Locator® hindlimb lameness dual reports for ride to left versus ride to right. Results of sit trot are at top of report and results of posting are at bottom of report. Top report: This horse has a right hindlimb impact lameness riding in both directions under both activities that is unaffected by activity. However, note the artifactual production of a left hindlimb pushoff lameness when riding to the left and posting on the right forelimb. Bottom report: This horse has a right hindlimb pushoff lameness riding in both directions under both activities that is strongly affected by posting. Note the decrease in right hindlimb pushoff lameness when riding to the left (posting on the right forelimb) and the increase in right hindlimb pushoff when riding to the right (posting on the left forelimb).

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Sitting the trot, when performed correctly, has the least artifactual effect on assessment of lameness. A rider riding to the left and posting on the right forelimb moves up and forward when the inside (left) forelimb is weight bearing. This movement can exacerbate an existing painful focus in the inside forelimb, making lameness more visible and easily measured. Subsequently, the rider falls downward and backward during pushoff of the inside hindlimb. This rider movement opposes the upward thrust of the horse's inside hindlimb, preventing or lessening pelvic rise. Less pelvic rise after pushoff on the inside hindlimb will be measured as a hindlimb pushoff-type lameness. This later effect of posting is highly predictable and definitive. Horses without hindlimb pain during pushoff will frequently measure with inside hindlimb pushoff-type (lack of impulsion) lameness. Horses with existing pushoff lameness in the inside hindlimb will measure with greater lameness. Horses with existing pushoff lameness on the outside hindlimb (ipsilateral to the posting forelimb) will measure with lesser lameness or the existing pushoff lameness will be completely masked. The effect of posting on lameness will be additive to the effect of direction of movement and to surfaces characteristics, which can be significant when riding in small circles. Lameness Locator® AIDES, taking into consideration both direction of movement, surface characteristics, and rider activity, remind the user about these artifactual effects. Amount of artifactual effect will depend on amplitude of rider posting, which, depending on the individual rider, may differ when riding in opposite directions. The combination of artifactual effects from direction of movement, surface characteristics, and rider activity complicates lameness evaluation under saddle. However, similar to evaluation of lameness while lunging, when lameness is consistently measured during in-hand straight-line evaluation, under-saddle evaluation with the rider sitting the trot uncommonly brings out a lameness in a different limb entirely, and for lameness measured with the rider sitting the trot, evaluation for lameness with the rider posting uncommonly brings out non-artifactual lameness in a different limb. Nevertheless, especially when lameness is not consistently measurable in hand, evaluation of lameness under saddle is a valuable technique to assess some lameness conditions in certain situations.

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PERINEURAL AND INTRASYNOVIAL ANESTHESIA

Gary M. Baxter

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Local anesthesia is commonly used during a lameness examination to confirm or identify the site or sites of pain where obvious pathology may not exist.[9,17,63,66] It can also be useful to prove the location of a lameness to a client who may be suspicious of another site. Local anesthesia may be accomplished by perineural infiltration (local nerve block), ring block, direct infiltration of a painful region, or intrasynovial injection (joints, tendon sheaths, and bursae). The reader is referred to the accompanying website for individual video clips that demonstrate many of the various techniques.

Perineural infiltration and ring blocks are used to localize the source of pain to a specific region and, therefore, should be performed in a systematic manner starting with the distal extremity and progressing proximally. Direct infiltration and intrasynovial anesthesia are used to identify the involvement of a specific structure or synovial cavity and do not necessarily have to be performed in a systematic manner.[9] It is not uncommon to perform several different types of local anesthesia in the same horse (perineural together with intrasynovial), depending on the specific clinical signs and problem. With lameness, local anesthesia is important to document the specific site of the problem so that diagnostic imaging (radiography, ultrasonography, or magnetic resonance imaging) can be used to determine the cause of the problem. Other uses of local anesthesia include providing analgesia during and after surgery and pain control for other painful conditions.

TYPES OF LOCAL ANESTHETICS

The local anesthetics most frequently used are 2% lidocaine hydrochloride (Xylocaine hydrochloride) and 2% mepivacaine hydrochloride (Carbocaine). These solutions are potent and rapidly effective, but can be locally irritating. Because mepivacaine is longer lasting, less irritating, and more reliable than lidocaine, it is used most frequently.[5,33,48,66] Lidocaine is thought to last only 60 minutes with the maximum effect at 15 minutes.[73] It was also recently reported to not resolve lameness despite the loss of skin sensation.[33] One study using force plate evaluations indicated that mepivacaine was also only fully effective for 15–60 minutes after a palmar digital (PD) nerve block was performed.[5] The effect of the block began to subside between 1 and 2 hours, but gait characteristics persisted beyond 2 hours. A more recent crossover study indicated that the mean duration of action was 366 minutes with skin sensation returning before the return of lameness.[33] These variables in skin desensitization and duration of action are very important to remember when performing multiple nerve blocks on any given horse over a prolonged period of time. Bupivacaine hydrochloride (Marcaine) may be used if the goal is to provide a longer duration of analgesia (4–6 hours), such as following surgery.[48]

SKIN PREPARATION AND RESTRAINT

The only skin preparation necessary for most sites of regional anesthesia is scrubbing/wiping the area with gauze soaked in alcohol until clean. However, there are a few sites for perineural anesthesia that are adjacent to synovial structures where during injection the needle may inadvertently enter a synovial cavity or the solution may diffuse into these structures. These exceptions include the low four-point block (digital tendon sheath or fetlock joint), the high palmar/plantar blocks (carpometacarpal or tarsometatarsal [TMT] joints, carpal or tarsal sheaths), and the lateral palmar block (middle carpal joint or carpal sheath). A more thorough skin preparation of these sites is recommended to avoid potential complications. A minimum 5-minute scrubbing of the skin and hair with an antiseptic and alcohol is recommended for intrasynovial injections.[29] The horse may be clipped if the hair is unusually long or soiled, but this is not necessary unless utilizing spinal needles with stylets.[1] In all cases, the least amount of local anesthetic possible should be used to reduce tissue irritation and to prevent local diffusion of anesthetic that may complicate the interpretation of the block.

When performing local anesthesia, the horse should be haltered and restrained by an attendant who is standing on the same side of the horse. For intrasynovial anesthesia, a twitch is usually applied so that there is minimal limb movement during the insertion of the needle and injection of the anesthetic. When using local anesthesia in the hindlimb, the practitioner should always be in a position so that minimal bodily harm will result if rapid movement occurs. In most cases the needle is inserted rapidly, and the syringe applied just tight enough to prevent loss of local anesthetic solution during injection.

PERINEURAL ANESTHESIA

Perineural anesthesia is used when the lame limb has been identified, but the exact region affected cannot be determined by other methods. Even if a suspicious region is identified, it is often useful to anesthetize the region to confirm that the lameness is emanating from a single location. It is not uncommon to find several regions on one limb or find that other limbs may be contributing to the overall lameness problem. In these cases local anesthesia allows one to interpret the percentage that each region is contributing to the lameness. In addition, the degree of improvement with perineural blocks may aid in the interpretation of the findings on imaging.

Perineural anesthesia is typically performed in a stepwise manner starting from the distal limb and progressing proximally. In general, the accuracy of desensitizing a nerve is greater in the distal limb (distal to the carpus and tarsus) than more proximally where the nerves are deeper and covered with soft tissue. Also, the more distal

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the nerve, the more specific or smaller the region is that is anesthetized. In most cases perineural anesthesia is thought to desensitize the skin and all deep structures distal to the site of injection. However, aberrant nerves exist and should be remembered when interpreting the response to a block. Some feel that ring blocks are more reliable to completely desensitize the skin than local perineural blocks.[75] However, ring blocks are not commonly performed when attempting to accurately locate the site of lameness. Guidelines for performing perineural anesthesia are given in Table 2.5.

A thorough knowledge of the neuroanatomy of the involved region and a good understanding of the limitations of perineural anesthesia are necessary to properly apply and interpret perineural anesthesia. Accurately determining whether the nerve has been completely desensitized by the block is often the first step in interpreting the result. Complete desensitization of the nerve is often evaluated by checking the skin sensation distal to the point of injection. This can be performed with a blunt object such as a pen, hemostat, or needle cap. These objects should not be jabbed into the skin, but applied gently at first with a gradual increase in pressure. Most horses are receptive to this technique and will quietly respond if the nerves are not totally desensitized. However, some horses are difficult to read, and skin sensation may persist even with an effective block. In contrast, one recent study indicated that lidocaine blocked skin sensation without resolving an experimentally induced lameness in 3/8 horses.[33] This difficulty of using skin sensation to indicate the success of a block is especially amplified for those performed more proximally in the limb (above the fetlock). Other manipulative tests that previously caused pain (such as hoof tester examination, deep palpation, and flexion) may need to be repeated to accurately determine if the block worked. The ultimate test is whether lameness is no longer present, but for those horses that have not improved,

Table 2.5. Guidelines for perineural local anesthesia.

Specific block	Needle size	Volume of anesthetic	Sterile skin prep recommended (Yes or No)	Location
Palmar/plantar digital (PD)	25 g, 5/8″	1–1.5 mL	No	Just above collateral cartilages
Basisesamoid (high PD)	25 g, 5/8″	1.5–2 mL	No	At the base of the proximal sesamoid bone
Pastern ring block	22 g, 1–1.5″	2–3 mL	No	Above collateral cartilages and directed dorsally
Abaxial sesamoid	25 g, 5/8″	1.5–2 mL	No	Abaxial surface of proximal sesamoid bone
Low palmar or 4-point	22–25 g, 5/8–1″	2–3 mL/site	Yes	Distal metacarpus (above buttons of splint bones)
High palmar or 4-point	25 g 5/8″ and 20–22 g 1.5″	3–4 mL/site	Yes	Proximal metacarpus
Lateral palmar (lateral approach)	20–22 g 1″	4–6 mL	Yes	Distal to accessory carpal bone
Lateral palmar (medial approach)	25 g, 5/8″ or 22 g 1″	3–4 mL	No	Medial aspect of accessory carpal bone
Ulnar	20 g 1.5″	10 mL	No	4″ above accessory carpal bone
Median	20–22 g 1.5–2.5″	10 mL	No	Caudal to radius below pectoralis muscle
Medial cutaneous antebrachial	22–25 g 1–1.5″	5–10 mL	No	Mid-radius near cephalic and accessory cephalic veins
Low plantar or 6-point	25 g, 5/8″ or 22 g 1″	2–3 mL/site	Yes	Distal metatarsus and each side of long digital extensor tendon
High plantar, high 4-point or subtarsal	25 g 5/8″ and 20–22 g 1.0″	3–4 mL/site	Yes	Proximal metatarsus
Deep branch of lateral plantar	20–22 g 1.0″	3–5 mL	Yes	Lateral aspect of proximal metatarsus
Tibial/peroneal	20–22 g 1.5″	10–20 mL/site	No	4″ above point of hock on lateral and medial aspects of limb

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accurate interpretation of complete nerve desensitization is important. Therefore, it is recommended that multiple approaches be used before deciding whether perineural anesthesia was or was not successful.

The Forelimb

Palmar Digital (PD) Block

The medial and lateral PD nerves are located just palmar to their respective artery and vein and lie along the dorsal border of the SDFT proximal to the pastern joint and along the DDFT distal to the pastern joint. The injection is done with the foot elevated in most cases. Some prefer to stand with their backs toward the animal's rear end while holding the hoof between their knees. Others prefer holding the pastern with one hand while injecting with the other and assume either a lateral or frontal position in relation to the limb (Figure 2.147; Video 2.7). The PD nerves should be anesthetized just distal to or at the proximal border of the collateral cartilages (Figures 2.147 and 2.148). Blocking the nerves at this location reduces the risk of anesthetizing the dorsal branches of the PD nerve.[64,66] If the PD block is performed 2–3 cm above the collateral cartilages, the proximal interphalangeal (PIP) joint is often desensitized in addition to the foot.[65] The PD nerve and neurovascular bundle are easily palpable at the level of the collateral cartilage just behind the DDFT. A 25-gauge, 5/8-inch (1.5-cm) needle is inserted into the subcutaneous tissue in a proximal to distal direction over the nerve, and 1–1.5 mL of local anesthetic solution is injected perineurally.[66,75] The needle is retracted slightly and redirected if excessive pressure is needed to inject. Since there are several tissue planes, it is advisable to inject a small amount of local anesthetic as the needle is being withdrawn. Each nerve can be injected individually (preferred by the author) or the needle can be directed across midline under the skin to inject the contralateral nerve (Figure 2.148).



Figure 2.147. This image illustrates the positioning to perform a PD nerve block when facing the back of the horse and holding the limb with one hand. The needle is directed toward the hoof and is inserted at or below the level of the collateral cartilages.

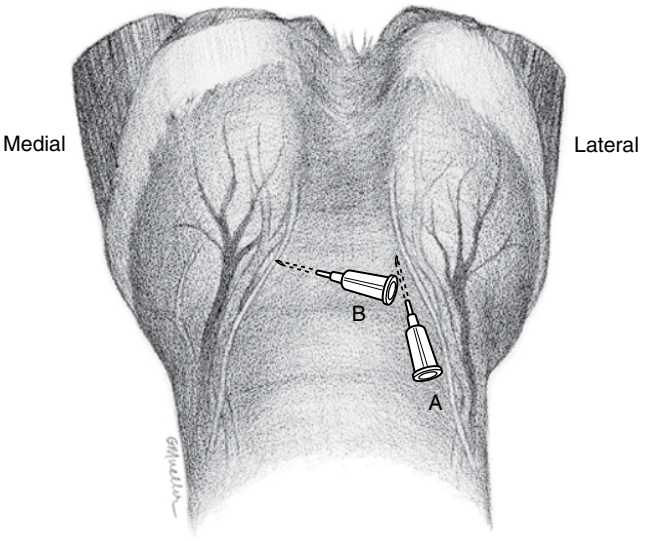


Figure 2.148. Injection sites for a PD nerve block. In (A), the needle is inserted parallel to the lateral PD nerve, while in (B), the needle enters just off midline and is inserted in the subcutaneous tissues to approximate the medial PD nerve.

Loss of skin sensation at the coronary band in the heel region and loss of deep sensation between the heel bulbs after 5–10 minutes are a reliable indication that the block was successful.[48] Previous hoof tester pain should also be eliminated following the block. Lameness assessment of the horse following the block should be performed in a similar manner as was done prior to the block. In cases of many foot-related lameness conditions, the lameness will often shift to the opposite forelimb. Structures that are desensitized with a biaxial PD nerve block include the entire sole, the navicular apparatus and soft tissues of the heel, the entire DIP joint of the forelimb, the distal portion of the DDFT, and some of the distal sesamoidean ligaments.[21,48,63,66] Partial desensitization of the PIP joint is also thought to occur in some horses.[75]

Pastern Ring or Semi-ring Block

A pastern ring block is performed just above the collateral cartilages of the distal phalanx at the same site as the PD block (Figure 2.149A). A 20- to 22-gauge needle is used to inject 3–4 mL of anesthetic subcutaneously laterally and medially from the site of the respective PD block. The needle is directed dorsally perpendicular to the long axis of the pastern to about the level of the medial and lateral collateral ligaments. This will anesthetize the dorsal branches of the PD nerve and will desensitize all the deep structures distal to the block. However, because the dorsal branches of the digital nerves are thought to contribute little to sensation within the foot, the pastern ring block is unlikely to improve lameness that has not been eliminated with a PD nerve block.[21,48] The pastern ring block can be performed with the limb in a full weight-bearing position but is easier to perform with the foot off the ground. Horses tend to resent this block more than the PD or basisesamoid nerve blocks.

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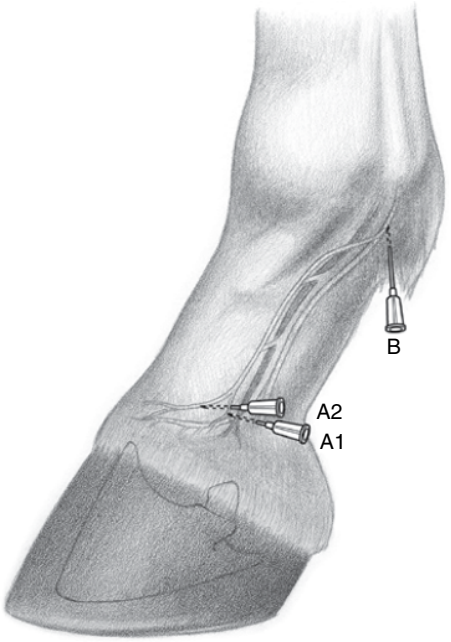


Figure 2.149. Injection sites for local anesthesia. (A1 and A2) Sites for the pastern ring block. (B) Site for the digital nerve block at the base of the sesamoid bones (basisesamoid block).



Figure 2.150. Needle location to perform an abaxial sesamoid nerve block in the forelimb.

Basisesamoid or High PD Block

An alternative to the pastern ring block is the basisesamoid block. This block is performed similarly to the PD block, only it is more proximal on the limb at the base of the proximal sesamoid bones (often referred to as a high PD block). The PD nerves can be palpated at this location, and 1.5–2 mL of anesthetic is deposited directly over the nerves using a 25-gauge, 5/8-inch (1.5-cm) needle (Figure 2.149; Video 2.8). The basisesamoid block will desensitize the dorsal branch and the PD nerve at a more proximal location in the pastern. This block desensitizes the palmar/plantar soft tissue structures of the pastern, PIP joint, and all structures of the foot. Because it is performed at the base of the sesamoid bones, it is unlikely to desensitize any of the fetlock joint.

Abaxial Sesamoid Block

The neurovascular bundle is easily palpable along the abaxial surface of the proximal sesamoid bone. With the limb elevated and holding the fetlock in the palm of the hand, isolate the palmar nerve by rolling it away from the artery and vein with the thumb or forefinger. A 5/8-inch (1.5-cm), 25-gauge needle is used to inject 1.5–2 mL of anesthetic perineurally (Figure 2.150; Video 2.9). Alternatively, the block can be performed with the horse standing (Video 2.10). It is best to use a small volume of anesthetic and direct the needle distally to avoid partial desensitization of the fetlock joint.[66] The biaxial block desensitizes the foot, middle phalanx, PIP joint, distopalmar aspects of proximal phalanx, distal portions of the SDFT and DDFT, distal sesamoidean ligaments, and digital annular ligament.[48,66] Loss of skin sensation at the coronary band in the toe region together with loss of skin sensation on the palmar pastern is used to determine the success of the block.[48] While it is common to find that skin sensation still exists over the dorsal surface of the phalanges, this does not mean that the phalanges and all the deep structures are not desensitized.

Low Palmar Block (Low 4-Point Block)

The low palmar block is often referred to as the low 4-point block because both palmar and palmar metacarpal nerves are anesthetized at the distal aspect of the metacarpus/metatarsus (Figure 2.151; Video 2.11). The lateral and medial palmar nerves lie between the suspensory ligament and the DDFT. Since they assume a vein–artery–nerve relationship, these nerves are located closer to the DDFT and lie on its dorsal edge.[47] These nerves are relatively deep but can be reached in most cases with a 5/8-inch (1.5-cm), 25-gauge needle (a 1-inch, 22-gauge needle may also be used), after which 2–3 mL of local anesthetic is deposited. It is best to perform these blocks 1 cm proximal to the distal ends of the splint bones to avoid injection into the digital flexor tendon sheath.[34,48,68]

Blocking the palmar nerves alone will not completely desensitize the fetlock joint. Two additional nerves, the medial and lateral palmar metacarpal nerves, innervate the deep structures of the fetlock.[56,75] These nerves course parallel and axial to the second and fourth metacarpal bones. A 5/8-inch (1.5-cm), 25-gauge needle or a 1-inch (2.5-cm), 22-gauge needle is used to inject 2–3 mL of anesthetic around these nerves as they emerge from the distal edges of the second and fourth metacarpal bones (Figure 2.151b). However, because the palmar pouches of the fetlock joint can be inadvertently entered at this location, these nerves can also be anesthetized more proximally.[48,68]

Both the palmar and palmar metacarpal nerve blocks can be performed while the horse is bearing full weight or the limb can be held with the opposite hand. It is usually easier to perform in the standing position. Anesthesia of these four nerves effectively desensitizes the deep structures of the fetlock region and all structures

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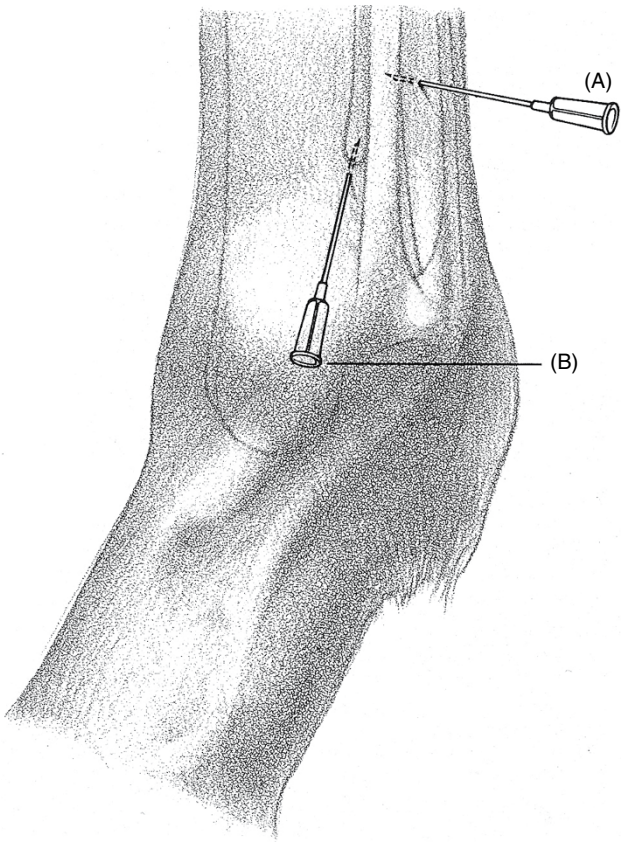


Figure 2.151. Low palmar or 4-point block. (A) Site for palmar nerve block, but it is recommended to go 1 cm proximal to the distal end of the small metacarpal bones. (B) Site for palmar metacarpal nerve block at the distal end of the splint bones.

distally.[48] Anesthesia of the skin over the dorsal aspect of the pastern and fetlock indicates that the block was successful. Some skin sensation may be present over the dorsal surface of the fetlock joint as a result of the sensory supply from the medial cutaneous antebrachial distribution.[56]

High Palmar (High 4-Point Block)

The high 4-point or high palmar block is analogous to the low 4-point block because the same four nerves are anesthetized in the proximal aspect of the metacarpus just below the carpometacarpal joint. However, the high palmar block is more difficult to perform because the soft tissue structures are more closely confined to the metacarpus and the palmar metacarpal nerves are located deeper within the axial borders of the second and fourth metacarpal bones. In addition, the distal outpouchings of the carpometacarpal joint extend approximately 2.5 cm distal to the joint in close proximity to the nerves and can be entered when blocking the palmar metacarpal nerves.[23,34] Therefore, aseptic preparation of the injection sites is recommended when blocking the palmar metacarpal nerves.

The proximal palmar nerves are anesthetized in the groove between the suspensory ligament and the DDFT. The nerves lie under heavy fascia, palmar to the vein and artery, and rest against the dorsal, lateral, and medial

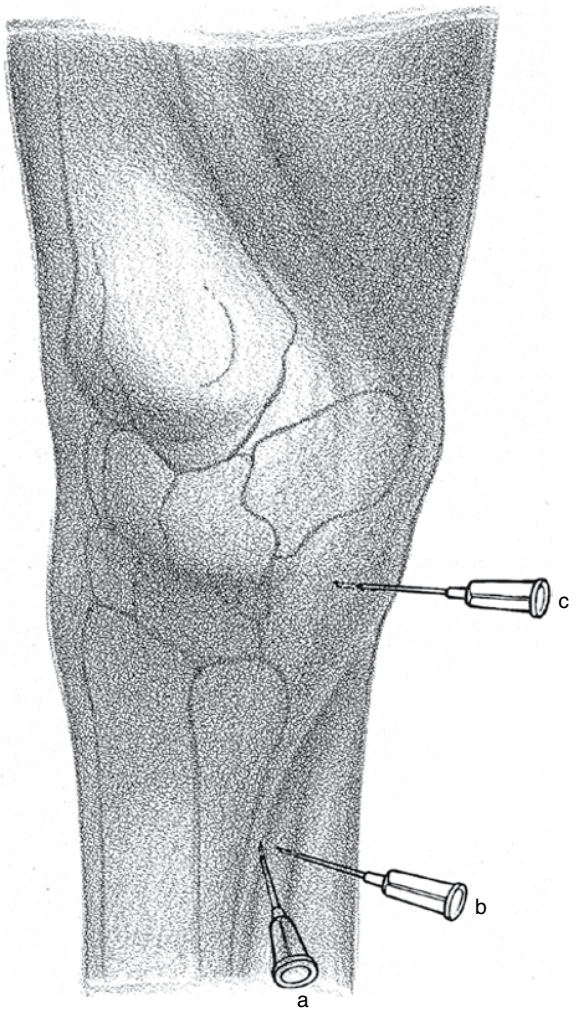


Figure 2.152. High 4-point block. (a and b) Needle positioned lateral and medial to block the palmar nerves. (c) Needle positioning to perform the lateral approach to block the lateral palmar nerve. Needle positioning to block the palmar metacarpal nerves is not shown but is located axial to the heads of the splint bones.

aspects of the deep digital flexor tendon (DDFT). A 5/8-inch (1.5 cm), 25-gauge needle is inserted through the heavy fascia, and 3–4 mL of anesthetic is deposited (Figure 2.152a and b).[48] Blocking just the palmar nerves will not completely desensitize the deep structures of the metacarpus.[75] The palmar metacarpal nerves run parallel and axial to the second and fourth metacarpal bones, and each can be desensitized by infiltration of 3–4 mL of local anesthetic along the axial surfaces of the metacarpal bones (Figure 2.153b_1 and b_2). A 20- to 22-gauge, 1.5-inch needle is directed toward the palmar metacarpus along the axial borders of the splint bones until bone is contacted. The needle is withdrawn slightly and aspirated to be certain that the needle is not within the carpometacarpal joint before the anesthetic is deposited. Blocking the palmar metacarpal nerves is usually performed with the limb held, whereas anesthesia of the palmar nerves is often easier with the limb weight-bearing.[48]

These four nerve blocks will effectively desensitize the deep structures of the metacarpus with the exception of the origin of the suspensory ligament.[34] The medial

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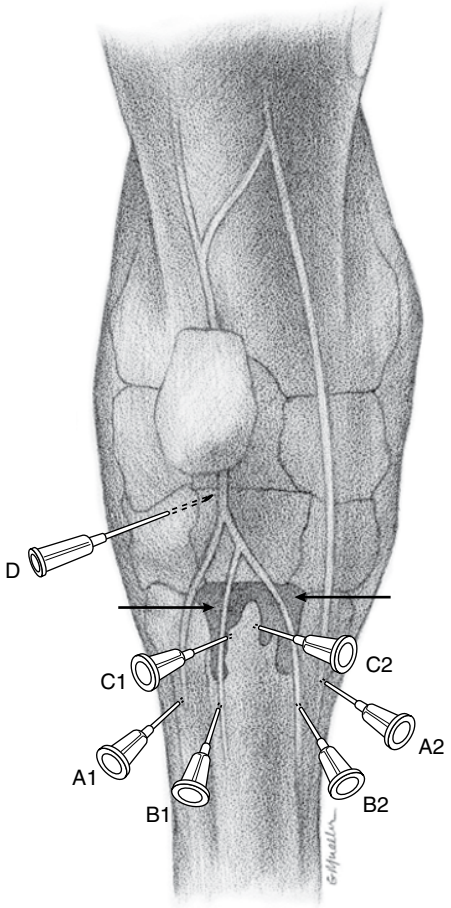


Figure 2.153. Palmar view of the carpometacarpal region of the left forelimb showing the synovial outpouchings of the carpometacarpal joint (arrows). (A1 and A2) Sites for injection of the palmar nerves. (B1 and B2) Sites for injection of the palmar metacarpal nerves. (C1 and C2) Sites for direct infiltration of the origin of the suspensory ligament. (D). Site for injection of the lateral palmar nerve using the lateral approach.

and lateral palmar metacarpal nerves innervate the interosseous ligaments of the second and fourth metacarpal bones, the interosseous lateralis and medialis muscles, and the suspensory ligament (interosseous muscle).[56] The palmar nerves innervate the flexor tendons and the inferior check ligament.[48] Horses that become sound after this block warrant diagnostic imaging of the metacarpal region. Lack of improvement with a high palmar block does not necessarily rule out a problem at the origin of the suspensory. Because of the difficulty of performing this block and its lack of specificity, use of the high palmar block has fallen out of favor with many clinicians. The lateral or medial approach to the lateral palmar nerve is usually performed instead especially if a proximal suspensory problem is suspected. Alternatively, a high 2-point block can be performed to desensitize the metacarpal region.

Lateral Palmar Block (Lateral Approach)

The lateral palmar nerve originates at a variable distance proximal to the carpus and represents a continuation of the median nerve plus the palmar branch of the ulnar nerve. The lateral palmar nerve courses in a dorsolateral direction distal to the accessory carpal bone and runs along the palmar distal aspect of the accessoriometacarpal ligament (Figure 2.153d). At the proximal end of the fourth metacarpal bone, the lateral palmar nerve gives off its deep branch that detaches branches to the origin of the suspensory ligament and divides into the lateral and medial palmar metacarpal nerves (Figure 2.153b_1 and b_2).[75] The lateral palmar nerve can be anesthetized just below the accessory carpal bone (lateral approach) or axial to the accessory carpal bone in a more proximal location (medial approach). This block desensitizes the origin of the suspensory ligament and other deep structures of the palmar metacarpus. Performing this block avoids the necessity of direct infiltration of the suspensory ligament and anesthesia of the palmar and palmar metacarpal nerves independently.[24,34]

The lateral palmar nerve is anesthetized with 4–6 mL of anesthetic administered through a 1-inch (2.5-cm), 20-gauge needle midway between the distal border of the accessory carpal bone and the proximal end of the fourth metacarpal bone on the palmar border of the accessoriometacarpal ligament (Figures 2.152c and 2.153d).[3,75] The needle is directed in a palmarolateral-to-dorsomedial direction and must penetrate the 2- to 3-mm thickness of the flexor retinaculum of the carpus (Video 2.12).[3,34] This block may be performed with the horse standing or with the carpus slightly flexed.[3,34] Skin sensation is not useful to evaluate the effect of the block. Instead, lack of any response to deep palpation of the proximal suspensory ligament often suggests an effective block.

Lateral Palmar Block (Medial Approach)

The lateral palmar nerve may also be blocked medial to the accessory carpal bone.[10] This medial technique is thought to reduce the risk of inadvertent injection into the carpal canal, which may occur with the lateral approach to the lateral palmar nerve.[24,48,49] The site of injection is a longitudinal groove in the fascia palpable over the medial aspect of the accessory carpal bone, palmar to the insertion of the flexor retinaculum that forms the palmaromedial aspect of the carpal canal.[10,48] With the limb weight-bearing, a 25-gauge, 5/8-inch needle is inserted into the distal third of the groove in a mediolateral direction perpendicular to the limb. The needle should contact the bone and 3–4 mL of anesthetic is injected (Figure 2.154). In some cases the injection may be difficult until the needle is withdrawn slightly or redirected. The author uses a 22-gauge, 1-inch (2.5 cm) needle and 3–4 mL of anesthetic for this technique (Video 2.13). A recent contrast study indicated that anesthetic may be distributed proximally to the distal third of the antebrachium following this block.[49] Skin sensation is not useful to evaluate the effect of the block, and lack of pain on palpation of the suspensory ligament is often the best indicator of success.

High 2-Point Block

The high 2-point block is a combination of the lateral palmar block and the high medial palmar block (1 nerve

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Figure 2.154. The medial approach to desensitize the lateral palmar nerve is located on the axial border of the accessory carpal bone.

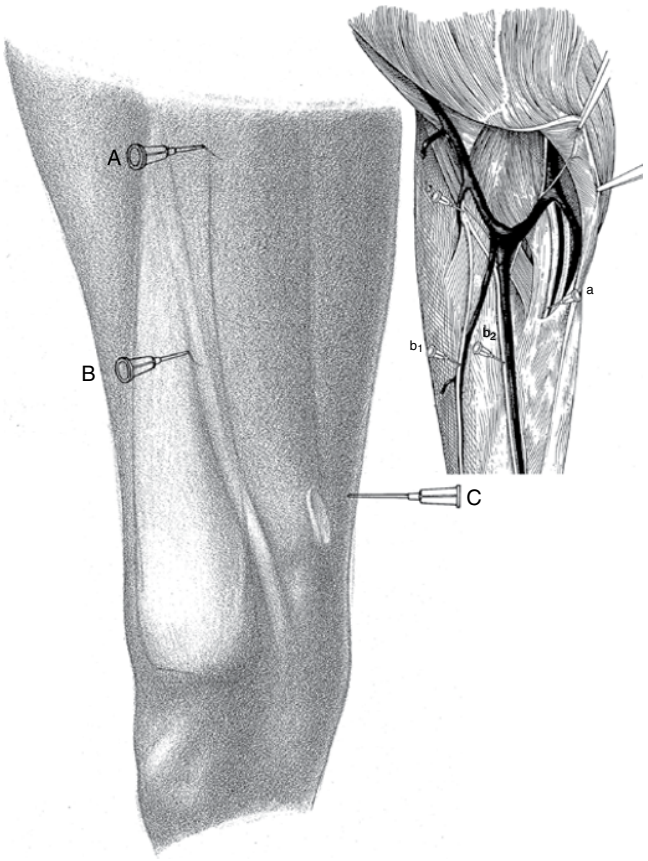


Figure 2.155. Forelimb blocks. (A) Site for median nerve block. (B) Site for medial cutaneous antebrachial nerve block. (C) Site for ulnar nerve block. Inset: (a) Site for median nerve block. (b) Site for medial cutaneous antebrachial nerve block as nerve crosses the lacertus fibrosus, which blocks both the cranial (b1) and the caudal (b2) branches.

of the high 4-point block). When performed, all deep and superficial structures on the palmar aspect of the metacarpus distal to the block will be desensitized. This includes the proximal aspects of the second and fourth metacarpal bones and the origin of the suspensory ligament.

This block can be used instead of the high 4-point and is easier to perform with less risk of complications. However, some clinicians have found it unnecessary to block the medial palmar nerve in conjunction with blocking the lateral palmar nerve.[75]

Ulnar, Median, and Medial Cutaneous Antebrachial Blocks

Perineural anesthesia is used most frequently up to the level of the carpus because the nerves lie superficial and the injection techniques are relatively easy to perform. Above this region intrasynovial anesthesia is often used to identify the site of lameness. However, the carpus and distal aspect of the limb can be desensitized by blocking the ulnar, median, and medial cutaneous antebrachial nerves. The medial cutaneous antebrachial nerve innervates only the skin, so it is primarily used to anesthetize the limb for a surgical procedure.[48] The median and ulnar nerve blocks may be used to locate a painful condition in the distal limb during a lameness examination. For instance, this procedure could be used to rule out lameness of the distal limb if lameness in the proximal forelimb was suspected.

The ulnar nerve is anesthetized approximately 4 inches (10 cm) proximal to the accessory carpal bone on the caudal aspect of the forearm (Figure 2.155C). Careful palpation reveals a groove between the flexor carpi ulnaris and ulnaris lateralis muscles. A 20-gauge, 1.5-inch (3.8-cm) needle is inserted through the skin and fascia perpendicular to the limb. Although the depth of this nerve varies, it is usually about 0.25–0.5 inches (1–1.5 cm) below the skin surface. The local anesthetic (10 mL) is infused both superficially and deeply in this region. Because the palmar branch of the ulnar nerve gives rise to the lateral palmar and palmar metacarpal nerves, anesthesia desensitizes the lateral skin of the forelimb distal to the injection site down to the fetlock.[48] Also, the accessory carpal bone and surrounding structures, palmar carpal region, carpal canal, proximal metacarpus, SDFT, and suspensory ligament are partially blocked by this technique. Lame horses with lesions in the very proximal aspect of the SDFT may only improve after an ulnar block.[11]

The median nerve is anesthetized on the caudomedial aspect of the radius, cranial to the origin of the flexor carpi radialis muscle (Figure 2.155A). The injection site is located just below the elbow joint where the ventral edge of the posterior superficial pectoral muscle inserts in the radius.[48] At this point the nerve is superficial and lies directly on the caudal surface of the radius. A 2- to 2.5-inch (5- to 6.2-cm), 20-gauge needle is inserted obliquely through the skin and fascia to a depth of 1–2

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inches (2.5–5 cm). The needle should be kept as close to the radius as possible to avoid the median artery and vein which lie caudal to the nerve, and approximately 10 mL of anesthetic is usually injected.[75] Blocking this nerve alone accomplishes little more than a medial and lateral palmar nerve block. However, blocking the median nerve in conjunction with the ulnar nerve effectively anesthetizes most of the important areas of lameness distal to the blocks.

The two branches of the medial cutaneous antebrachial nerve are blocked on the medial aspect of the forearm halfway between the elbow and the carpus, just cranial to the cephalic vein and just cranial to the accessory cephalic vein (Figure 2.155b_1 and b_2).[75] The nerve is usually just below the skin; however, its location can vary. It is best to block the subcutaneous tissues both cranial and caudal to the cephalic vein. A 22-gauge, 1-inch (2.5-cm) needle is used to deposit 5 mL of anesthetic solution. Alternatively, the medial cutaneous antebrachial nerve may be blocked as it crosses the lacertus fibrosus before it branches (Figure 2.155b).[48,75]

The Hindlimb

The neuroanatomy of the distal hindlimb below the tarsus is somewhat similar to that of the forelimb below the carpus. The majority of perineural techniques described previously for the forelimb are similar in the hindlimb. However, limb positioning, restraint, and the technique may vary slightly. One difference in the neuroanatomy is that lateral and medial dorsal metatarsal nerves from the deep peroneal (fibular) nerve course over the dorsolateral and dorsomedial surfaces of the third metatarsal bone and digits.[75] Therefore, it is recommended to anesthetize the dorsal metatarsal nerves dorsally when performing plantar digital nerve blocks at the pastern and proximal (abaxial) sesamoid bones and low and high 4-point plantar nerve blocks. This is accomplished by injecting 2–3 mL of anesthetic subcutaneously, lateral and medial to the long digital extensor tendon using a 5/8-inch (1.5-cm), 25-gauge needle.

Intrasynovial anesthesia is performed most frequently proximal to (above) the metatarsus in the hindlimb. However, perineural anesthesia of the tibial and peroneal nerves can be used to desensitize the tarsal region. The tibial and peroneal nerve blocks can also be used to determine whether the pain from a severe lameness without clinical findings is located proximal or distal to the hock region. Horses exhibiting subtle lameness are generally not good candidates for tibial and peroneal anesthesia because blocking the peroneal nerve may affect the horse's ability to extend the digit, thus making interpretation of the results difficult.[9]

When dealing with the hindlimb, proper restraint and body positioning are important to prevent bodily harm. In most cases a twitch is applied, and the handler should stand on the same side as the veterinarian. All blocks should be performed with the veterinarian facing toward the back of the horse. The authors routinely begin diagnostic nerve blocks at the level of the proximal sesamoid bones unless there is uncertainty regarding foot involvement. If the foot is suspected, a high PD nerve block in the pastern is usually performed because often there is less concern about anesthetizing more proximal structures. In addition, a low PD block is difficult to perform in the hindfeet because the fetlock flexes when the limb is picked up.

The PD and abaxial sesamoid blocks are best performed with the limb extended behind the horse in a position similar to that when performing a fetlock flexion test or applying a horseshoe. The point of the hock is held fast by cradling it with the inside of the arm and axilla. This position reduces the ability of the horse to withdraw the limb to kick. Perineural blocks performed proximal to the abaxial sesamoid block are usually performed while standing close to the horse and with the limb on the ground. If the horse is prone to kicking, the limb can be held fast by grasping the foot, after which the limb is brought forward (similar to that done with a spavin test) to perform the block.[9]

High Plantar Block

The high plantar block anesthetizes the medial and lateral plantar and plantar metatarsal nerves just below the tarsus analogous to the high palmar block of the forelimb. Anesthesia of the dorsal metatarsal nerves just below the TMT joint may be included in this block to provide complete analgesia to structures in the metatarsal region and below.[48] The plantar metatarsal nerves can be blocked using a 1.5-inch (3.8-cm), 20-gauge needle inserted axial to the second and fourth metatarsal bones and directed dorsally toward the plantar aspect of the metatarsus. Three to four mL of local anesthetic is injected into the region of the origin of the suspensory ligament. Inadvertent administration of anesthetic into the tarsal sheath or the TMT joint can occur when performing anesthesia of the plantar metatarsal nerve.[20,34,48] Because of the potential for intrasynovial injection, careful skin preparation prior to performing this block is recommended.[20,34]

The medial and lateral plantar nerves can be anesthetized by placing 3–4 mL of anesthetic through the heavy fascia adjacent to the dorsal surface of the DDFT in the proximal metatarsal region using a 25-gauge, 5/8-inch needle. If a large volume of anesthetic is used at the site of the lateral plantar nerve block, the deep branch of the lateral plantar nerve (DBLPN) can also be anesthetized.[48] This will block both the medial and lateral plantar metatarsal nerves, negating the need to block these nerves individually. The high plantar block effectively desensitizes the second and fourth metatarsal bones, the suspensory ligament and its origin, and the flexor tendons in the metatarsal region. One study concluded that the high plantar nerve block cannot be used to differentiate between flexor tendon and suspensory ligament lesions because horses with both conditions improved after the block.[40] Because the high plantar block is difficult to perform and interpret and can inadvertently block the tarsal sheath or TMT joint, it is not commonly performed. Most clinicians will perform the DBLPN block instead.

Deep Branch of the Lateral Plantar Nerve (DBLPN) Block

The DBLPN innervates the proximal suspensory in the hindlimb and is removed to treat some horses with hindlimb proximal suspensory desmitis. This nerve can

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be selectively desensitized to aid more accurate diagnosis of this condition (Figure 2.156). Two different techniques have been described (Figure 2.157). With the first approach, a 20–22-gauge, 1-inch (2.5-cm) needle is inserted 15 mm distal to the head of the fourth metatarsus and directed perpendicular to skin between the axial border of the fourth metatarsus and the SDFT to a depth of approximately 25 mm (Figure 2.156–2.158).[12,35] This single-injection technique was considered to be 95% accurate to block the DBLPN in a cadaver and live horse study. Alternatively, a 20–22-gauge, 1.5-inch (3.8-cm) needle can be inserted 20 mm distal and plantar to the head of the fourth metatarsus and directed proximodorsally and axial to the bone (Figure 2.157).[25] The needle is advanced to a depth of 1–2 cm and anesthetic is deposited.[25] It is usually best to hold the limb to perform either of these techniques, and it is the author's opinion that the perpendicular approach is easier to perform (Figures 2.156 and 2.157). The single-injection technique for the DBLPN is thought to provide a reliable method for perineural analgesia of the DBLPN for diagnosis of proximal suspensory desmitis of the pelvic limb with a minimal risk of inadvertently desensitizing structures within the tarsal sheath and the TMT joint.[25,35] However, two different *in vivo* studies have shown that

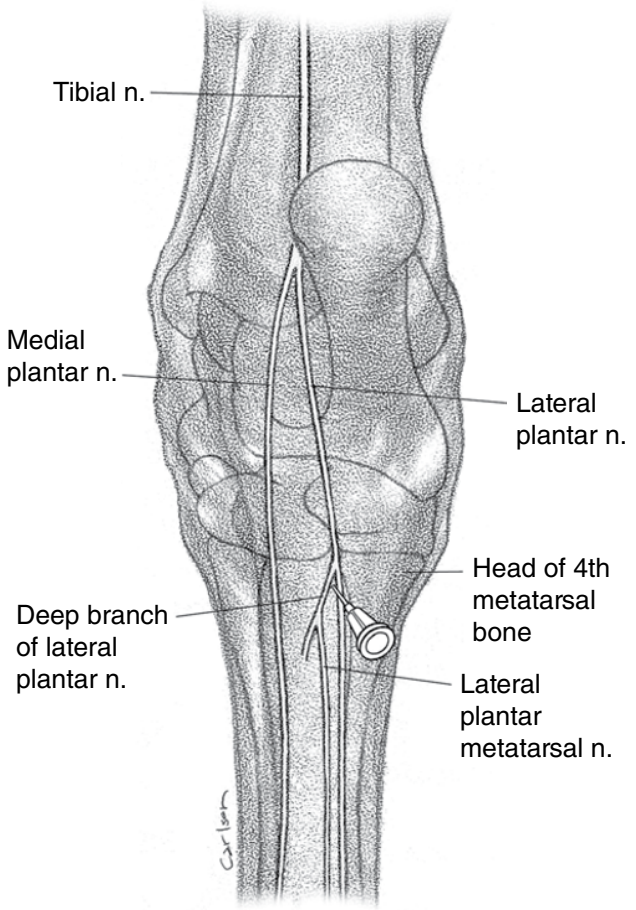


Figure 2.156. Innervation of the proximal suspensory ligament in the hindlimb and the location and positioning of the needle following the perpendicular approach to block the DBLPN just below the tarsus.

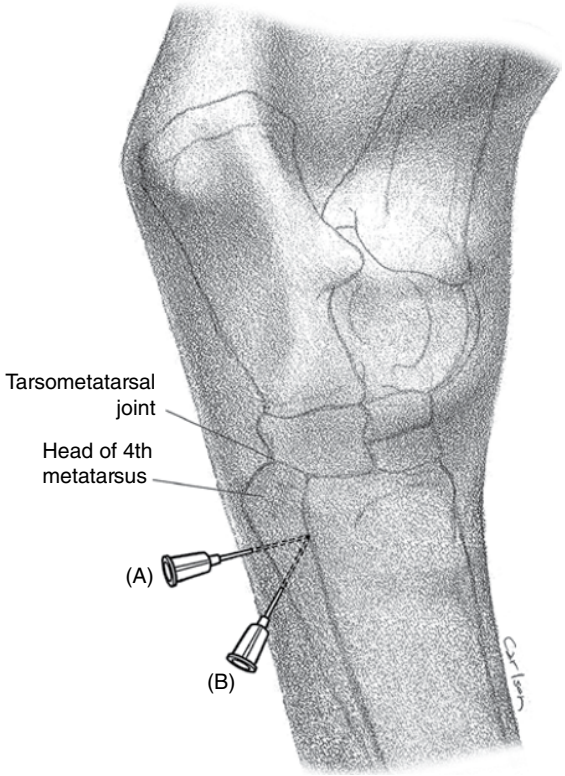


Figure 2.157. The deep branch of the lateral plantar nerve (DBLPN) can be desensitized by inserting a needle 15 mm below the head of the lateral splint bone directed perpendicular to the limb (A) or 20 mm below the head of the lateral splint and directing the needle proximodorsally and axial to the bone (B).

anesthetic can enter the TMT joint and the tarsal sheath following this block regardless of the technique used.[12,13] Diffusion of anesthetic both proximally and distally in the limb can also occur following injection, and the amount of diffusion is volume dependent. Therefore, a small volume of anesthetic (no more than 5 mL) is recommended when performing the DBLPN block to prevent inadvertent desensitization of other structures.[2,12]

Tibial and Peroneal Block

Anesthetizing the tibial and deep and superficial peroneal nerves above the point of the hock desensitizes the entire distal limb.[48] These blocks can be helpful to diagnose some horses with hock lameness, or they can be used to rule out whether the pain causing the lameness is located within the hock or distal limb. However, blocking the peroneal nerve, particularly the common trunk, can affect the ability of the horse to extend the limb and may cause dragging of the toe or knuckling of the fetlock.[9,75] This may complicate the ability to assess improvement in the lameness and injure the horse during the lameness evaluation.

The site for injection of the tibial nerve is approximately 4 inches (10 cm) above the point of the hock on the medial aspect of the limb between the Achilles tendon and the deep digital flexor muscle (Figure 2.159). When the horse is weight bearing, the nerve lies close to the caudal edge of the deep digital flexor muscle.

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Figure 2.158. (a) Blocking the DBLPN by inserting the needle 15 mm distal to the head of the lateral splint bone perpendicular to the limb. Some clinicians cradle the limb and pull the tendons medially (b) to facilitate the injection (see Figure 2.156 for needle location).

The nerve (6 mm in diameter) can be palpated caudal to the deep flexor muscle by unweighting the limb and grasping firmly cranial to the Achilles tendon with the thumb and forefinger.[3,75] The block may be performed



Figure 2.159. Image illustrating the locations to block the tibial and peroneal nerves. The site for injection of the tibial nerve is approximately 4 inches (10 cm) above the point of the hock on the medial aspect of the limb between the Achilles tendon and the deep digital flexor muscle. The location to block the peroneal nerves is approximately 4 inches (10 cm) above the point of the hock on the lateral aspect of the limb in the groove formed by the muscle bellies of the lateral and long digital extensor muscles.

by either standing on the lateral side of the limb to be blocked or by reaching across from the opposite limb to access the medial aspect of the limb. A small amount of anesthetic placed in the skin and subcutaneous tissues may minimize the horse's reaction to the block. A 20- to 22-gauge, 1.5-inch (3.8-cm) needle is used to deposit 15–20 mL of anesthetic in several tissue planes in the fascia that overlies the deep digital flexor muscle. Blocking the tibial nerve provides anesthesia to the plantar tarsus, metatarsus, distal Achilles tendon, calcaneus, suspensory ligament, and most of the foot.[9,75]

To completely desensitize the hock and limb distal to the hock, the deep and superficial peroneal (fibular) nerves must be anesthetized.[48,75] The deep peroneal nerve lies near the lateral edge of the cranial tibial muscle close to the tibia. The superficial peroneal nerve lies slightly caudal to the septum of the two extensor muscles and more superficial. The location of injection is approximately 4 inches (10 cm) above the point of the hock on the lateral aspect of the limb in the groove formed by the muscle bellies of the lateral and long digital extensor muscle (Figure 2.159). To block the deep peroneal nerve, a 1.5- to 2-inch (3.8- to 5-cm), 20-gauge needle is inserted in a slightly caudal direction until the needle contacts the caudal edge of the tibia. Ten to 15 mL of anesthetic is injected on the lateral border of the cranial tibial muscle close to the tibia. The needle is then retracted and another 10–15 mL of local anesthetic is injected more superficially in several planes to be sure

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that the superficial peroneal nerve is blocked. The depth of the superficial peroneal nerve can vary, so the more superficial injection should include a region from 0.6 to 2.5 cm deep.[75]

An alternative to blocking the superficial and deep peroneal nerves individually is to block the common peroneal nerve proximal to its division.[9] This can be accomplished by blocking the nerve near the origin of the long digital extensor tendon. The nerve can be palpated at this point, and it is anesthetized using a 1.5-inch (3.8-cm), 20-gauge needle to inject 15–20 mL of anesthetic.

Direct Infiltration of Anesthetic

Direct infiltration of anesthesia can be used anywhere a sensitive area is identified. However, it is used most often at sites of insertions of ligaments and tendons (e.g. the proximal interosseous muscle) or at bony prominences (i.e. splints or swellings). The region is infused directly with local anesthetic instead of performing perineural anesthesia. This approach often permits the clinician to be more definitive regarding whether a painful region is contributing to the lameness. The amount of local anesthetic administered depends on the location and dimensions of the area involved.

The origin of the suspensory ligament can be desensitized by direct infiltration (Figure 2.153c_1 and c_2), but perineural anesthesia using the lateral palmar nerve block in the forelimb and the DBLPN block in the hindlimb is preferred. Direct infiltration of the proximal suspensory is best performed with the limb held with the opposite hand. A 1-inch (2.5-cm), 20- to 22-gauge needle is inserted between the attachments of the suspensory ligament and the inferior check ligament in the forelimb or between the fourth metatarsus and the SDFT in the hindlimb.[75] The needle is directed toward the origin of the suspensory ligament and 4–6 mL of anesthetic is injected. Both the lateral and medial sides can be blocked in the same manner, but this is usually unnecessary. Inadvertent injection into the carpometacarpal joint in the forelimb and the TMT and tarsal sheath in the hindlimb can occur with these techniques.[2,3,75]

INTRASYNOVIAL ANESTHESIA

The use of intrasynovial anesthesia plays an important role in the diagnosis of equine lameness.[28,34,43,47,79] In most cases, it is more specific and efficient to anesthetize the specific synovial structure (joint capsule, tendon sheath, or bursa) that is thought to be the cause of the lameness than performing local perineural anesthesia. This is especially true in horses that tend to have joint problems (racehorses) or if the clinical findings suggest involvement of a synovial structure. In addition, intrasynovial anesthesia is commonly performed above the carpus and tarsus where perineural anesthesia becomes more difficult. If intrasynovial anesthesia needs to be performed after perineural anesthesia, it is best to wait at least 3–4 hours for sensation to return.[5,33,75] There is also evidence to suggest that the return of skin sensation does not coincide with the actual return of lameness in some horses.[33] In addition, gait changes may persist beyond 3–4 hours, and it is probably safest to perform intrasynovial anesthesia on a different day.[5,33]

Intrasynovial anesthesia is thought to be more specific than perineural anesthesia because if the lameness improves, the synovial cavity is considered the site of the problem. The three major exceptions to the specificity of intrasynovial blocks are the distal interphalangeal (DIP) joint, middle carpal joint, and the TMT joint. These exceptions are usually related to regional nerves being close to synovial outpouchings of the synovial cavities or passive diffusion of local anesthetic from the synovial cavity.[25,61,63,64,66,67] Intrasynovial anesthesia of the DIP joint can anesthetize the nerves innervating the foot,[6–8,17,18] and intrasynovial anesthesia of the middle carpal joint or TMT joint can provide analgesia of the proximal palmar metacarpal/plantar metatarsal regions, respectively.[14,23,24] In general, the more anesthetic that is used, the greater the likelihood of inadvertent analgesia of surrounding structures.[62,66] Less commonly, nerves that lie close to the site of intrasynovial injection also may be inadvertently desensitized. For example, the lateral plantar nerve may be desensitized when injecting local anesthetic into the TMT joint,[20] and the lateral palmar/plantar nerve above the fetlock may be desensitized when injecting anesthesia into the digital tendon sheath using the proximal approach.[37]

General Technique: Site Preparation, Restraint, and Interpretation

There are several different anatomic approaches that can be used for intrasynovial injections in most synovial cavities. Knowledge of the anatomic landmarks for intrasynovial injections is imperative to be able to competently perform these injections. Practice on cadaver limbs can be very beneficial to improve the proficiency in performing the injection techniques because repeated attempts to locate the synovial cavity are not well tolerated by most horses.

Proper preparation of the site for injection is necessary to prevent subsequent infection within the synovial cavity. Iatrogenic infection appears very rare following anesthesia of a synovial cavity but is more likely to occur when synovial cavities are treated with medications, especially corticosteroids. However, this risk is still quite low with a reported occurrence of 1/1,279 injections (0.08%).[76] Clipping the hair is usually unnecessary because there is no appreciable difference in bacteria-forming units in clipped and haired skin after 5 minutes of preparation with povidone iodine scrub followed by an alcohol rinse.[29] However, if the hair is very long and soiled, it is best to clip the hair overlying the injection site, and clipping is recommended when using spinal needles with stylets.[1] In general, the authors tend to clip a very small area (1–2 cm square) over the site unless the owners/trainers request otherwise. Clipping the hair also has the advantage of marking the site of injection so that a helper knows exactly where to prep the skin. A 5-minute sterile skin preparation is recommended using either povidone iodine or chlorhexidine and alcohol.

An experienced helper makes performing intrasynovial injections much easier and safer. Proper restraint of the horse is also required to prevent injury to personnel and damage to the articular cartilage and to reduce the risk of needle breakage. Be sure to always keep in mind that the handler and any person observing becomes your assumed responsibility.[47] Twitch

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restraint is recommended by the authors for all intrasynovial injections unless it is not tolerated by the horse. Horses being treated by intrasynovial injections are often routinely tranquilized, but this is not usually possible for injections used for diagnostic purposes. Local anesthetic (1–2 mL) at the site of injection also may aid in the injection process, especially for synovial cavities in the proximal aspect of the limbs. In addition, the smallest possible gauge needle (usually 20 gauge or smaller) should be used to minimize objection by the horse.

Sterile gloves are recommended to permit careful palpation of the anatomic landmarks and to be able to handle the shaft of the needle without contamination. The injection should be done carefully but also as rapidly as possible. Once the needle has penetrated the synovial space, synovial fluid may be observed draining from the needle hub. The synovial fluid is allowed to run freely until its ejection pressure is reduced to a slow drip. The syringe is then inserted on the finger-stabilized needle hub, and the anesthetic is injected as rapidly as possible. If synovial fluid is not observed, a small syringe can be attached to the needle to withdraw fluid. However, lack of synovial fluid does not necessarily mean that the needle is not within the synovial cavity. To confirm correct needle placement, one can inject a small amount of sterile solution; if there is little or no plunger pressure, it is reasonable to assume that the needle is within the synovial space. However, the only definitive method to confirm that the needle is in the correct location is to obtain synovial fluid. Mepivacaine is the anesthetic of choice for intrasynovial anesthesia because there is some evidence that it is less irritating than lidocaine after intra-articular injection.[8,48] Guidelines for performing diagnostic intrasynovial anesthesia, including suggested volumes of anesthetic to use, are given in Table 2.6.

Assessment of intrasynovial blocks is usually performed 5–30 minutes after completion of the injection. If there is no improvement after 30 minutes, it is unlikely that waiting longer will change the response. In one study, lameness was not apparent after 5 minutes, and the improvement lasted for 55 minutes.[3] In addition, improvement in lameness within 5–8 minutes of injection is often seen after intra-articular injection of the DIP joint in horses with navicular disease or experimentally induced navicular bursal pain.[18,52,63] Evaluation of the effectiveness of the block should include repeating the exercise that resulted in the most significant signs of lameness and possibly re-performing the manipulative/flexion test that made the examiner suspicious that this region was involved. It is important to remember that structures superficial to the synovial cavity may retain their sensitivity.[75] In addition, false-negative intrasynovial blocks have been reported but tend to be uncommon. One report identified a failure of intra-articular anesthesia of the radiocarpal joint to abolish lameness associated with chip fracture of the distal radius.[71] Diffusion of anesthetic to local structures, inadvertent anesthesia of peripheral nerves closely associated with the synovial cavity outpouchings, and the possibility that the injection was not in the synovial cavity should all be considered when assessing the response to intrasynovial anesthesia.

Distal Interphalangeal (DIP) Joint

The DIP joint can be entered using three dorsal approaches (perpendicular, parallel, or dorsolateral) and one lateral approach. These approaches are usually best performed in the standing patient, and a maximum of 4–6 mL of anesthesia is recommended.[66] The dorsolateral and dorsal parallel approaches are used most commonly by the author. The site of injection for the dorsolateral approach is 0.5 inch (1 cm) above the coronary band and 0.75–1 inch (2–3 cm) lateral (or medial) to midline. A 1.5-inch (3.8-cm), 20-gauge needle is inserted from a vertical position and directed distally and medially toward the center of the foot at approximately a 45° angle (Figure 2.160; Video 2.14). The needle should enter the DIP joint capsule at the edge of the extensor process. If entry into the joint is uncertain, the needle can be directed at a more acute angle (more horizontal) to the skin and inserted until the needle contacts the distal end of P2.[26,79] It then is "walked" distally until the joint is penetrated.

Some prefer to enter the joint on the dorsal midline using the proximal outpouching of the DIP joint above the extensor process.[48,63,66] The injection site is just above the coronary band 0.25–0.5 inches (8–12 mm) above the edge of the hoof wall on the dorsal midline of the foot. With the dorsal perpendicular approach, the needle is directed downward perpendicular to the bearing surface of the foot (Figure 2.161; Video 2.15).[48] With the dorsal parallel approach, the needle is directed parallel or slightly downward (hub of the needle is moved proximally) to the ground to a depth of approximately 0.5 inches (12–15 mm) (Figure 2.162; Video 2.16). The dorsal parallel approach is usually easier to perform and is recommended by many clinicians.[48,63,66]

The site for injection for the lateral approach is bounded distally by a depression along the proximal border of the collateral cartilage approximately midway between the dorsal and palmar/plantar border of P2 (Figure 2.163; Video 2.17). A 1-inch (2.5-cm), 20-gauge needle is directed downward at a 45° angle toward the medial weight-bearing hoof surface.[81] Most horses appear to tolerate this technique very well. However, the specificity of the lateral approach is thought to be less than the dorsolateral approach. In one study using cadavers and live horses, contrast material entered the DIP joint in 100% of the cases injected using the dorsolateral approach and 85% of the cases in which the palmar/plantar lateral approach was used.[81] Importantly, with the lateral approach, only 65% of the limbs had contrast exclusively in the DIP joint, 20% had contrast in the digital sheath, and 5% had contrast in the subcutaneous tissues.[81] Because of this and the ease of performing any of the dorsal approaches to the DIP joint, the lateral approach is rarely used.

Several studies have documented that injection of the DIP joint with a local anesthetic is not selective for the joint and it will cause analgesia of the podotrochlear apparatus, navicular bone, and navicular bursa.[7,8,17,39,52,61,64] In addition, injection of the DIP joint may cause partial, and often complete, analgesia of the sole, toe, and heel regions of the foot.[61,62] The analgesic effect increased with time, and 10 mL of anesthetic was more effective than 6 mL in alleviating pain in the foot.[62]

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Table 2.6. Guidelines for intrasynovial anesthesia.

Synovial cavity	Needle size	Volume of anesthetic	Approaches and limb position (standing or held)
Coffin joint	20–22 g, 1–1.5″	4–5 mL	Dorsal approaches: standing Lateral approach: standing or held
Pastern joint	20–22 g, 1.5″	4–5 mL	Dorsal and dorsolateral approaches: standing Palmar/plantar approach: held
Fetlock joint	20–22 g, 1–1.5″	8–12 mL	Proximal palmar/plantar approaches: standing or held Collateral sesamoidean approach: held Distal palmar/plantar approach: standing Dorsal approach: standing
Carpal joints	20–22 g, 1–1.5″	8–10 mL	Doral approaches: held Palmar approaches: standing
Elbow	20 g, 1.5″ or 20 g, 3.5″	20–30 mL	All approaches: standing
Shoulder	18–20 g, 3.5″	20–40 mL	All approaches: standing
Tarsometatarsal joint	20 g, 1–1.5″	4–6 mL	All approaches: standing
Distal intertarsal joint	25 g, 5/8″ or 22 g, 1″	3–5 mL	All approaches: standing
Tarsocrural joint	20–22 g, 1.5″	15–20 mL	All approaches: standing
Femoropatellar joint	20 g, 1.5–3.5″	30–40 mL	All approaches: standing
Medial femorotibial joint	20 g, 1.5″	20–30 mL	All approaches: standing
Lateral femorotibial joint	20 g, 1.5″	20–30 mL	All approaches: standing
Coxofemoral joint	16–18 g, 6–8″ spinal	30–60 mL	All approaches: standing
Sacroiliac joint	15–16 g, 10″ spinal	7–10 mL	All approaches: standing
Digital flexor tendon sheath	20–22 g, 1–1.5″	8–15 mL	Proximal approach: standing All other approaches: held
Carpal sheath	20 g, 1.5–3.5″	15–30 mL	Medial approach: standing Lateral approach: held
Tarsal sheath	20 g, 1.5″	15–20 mL	Medial approach: standing
Extensor carpi radialis sheath	20 g, 1.5″	10–20 mL	All approaches: standing or held
Calcaneal bursa	20 g, 1.5″	10–15 mL	Distal approach: standing Proximal approach: standing or held
Bicipital bursa	18–20 g, 3.5–5″ or 20 g, 1.5″	20–30 mL	All approaches: standing
Trochanteric bursa	18–20 g, 1.5–3.5″	7–10 mL	All approaches: standing
Cunean bursa	20–22 g, 1″	2–3 mL	Medial approach: standing

To improve the selectivity of DIP joint anesthesia, a maximum of 5–6 mL of anesthesia is recommended, and assessment of the block should be performed within 10 minutes of injection.[63,66]

Podotrochlear (Navicular) Bursa

The traditional approach to access the navicular bursa is through the heel bulbs.[51,60,62,66] With this approach, a 18-20-gauge, 3.5-inch (8.9-cm) spinal needle is inserted between the heel bulbs just above the coronary band (Figure 2.164; Video 2.18). Local anesthesia at the site of the injection or perineural anesthesia above the heel bulbs may be used to provide skin analgesia prior to the injection. The needle is advanced along a sagittal plane aiming for a point 1 cm below the coronary band, midway between the toe and the heel.[60] The needle is advanced until bone is contacted. Only 2–4 mL or anesthetic or medication can usually be injected, and flexing the lower limb will usually decrease the resistance to injection. Some clinicians prefer to use a special wooden block that unweights the heel and flexes the distal limb to

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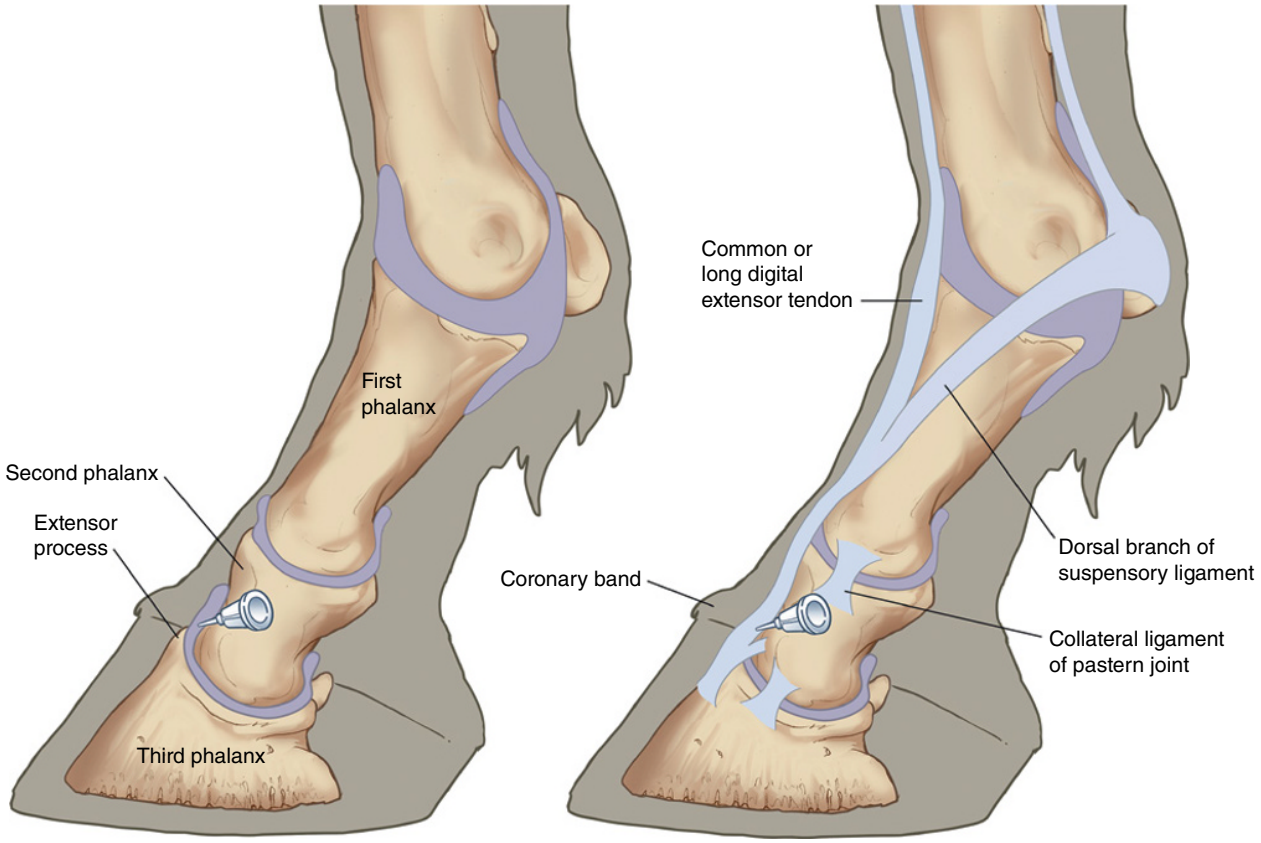


Figure 2.160. Injection site for the dorsolateral approach to the DIP joint. The injection site is 1 cm above the coronary band just off midline and the needle is directed at a 45° angle.

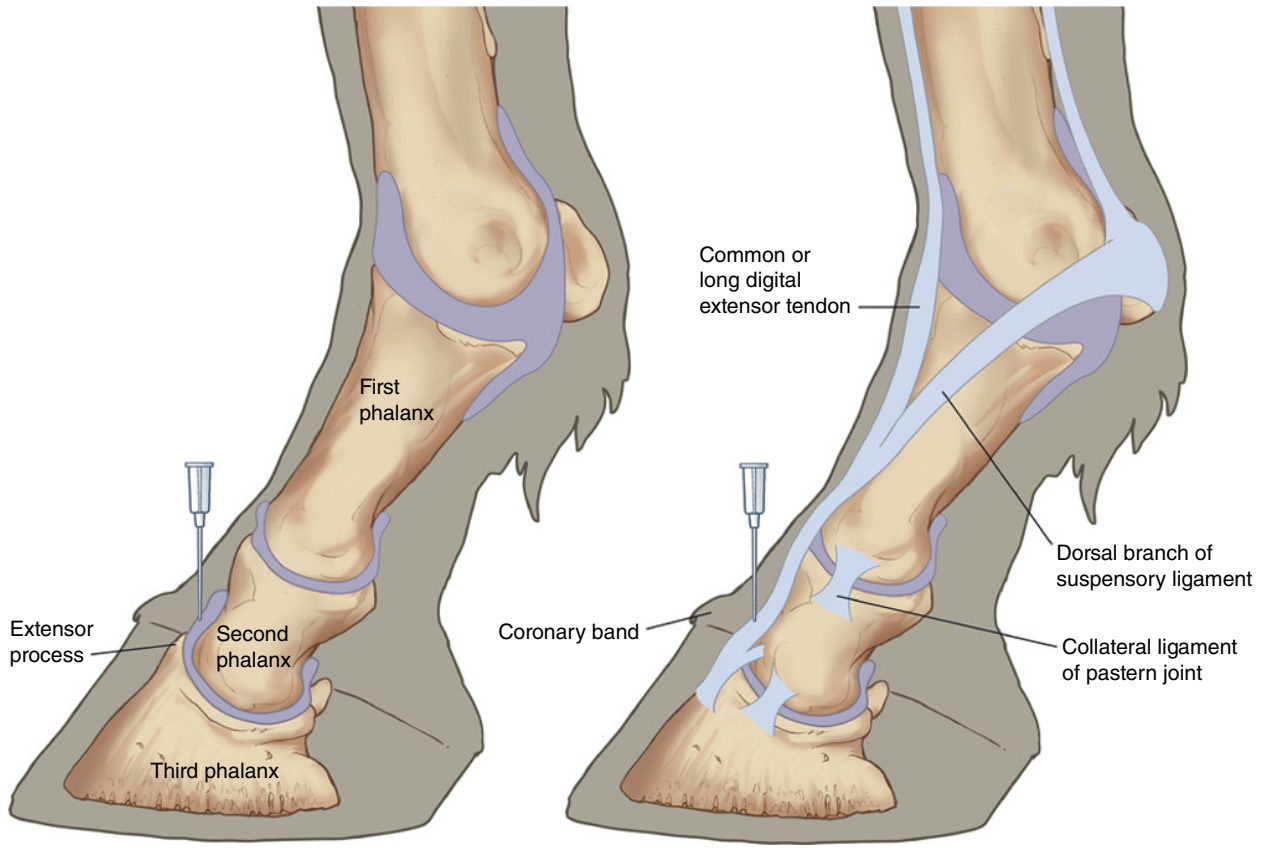


Figure 2.161. The dorsal perpendicular approach to the DIP joint.

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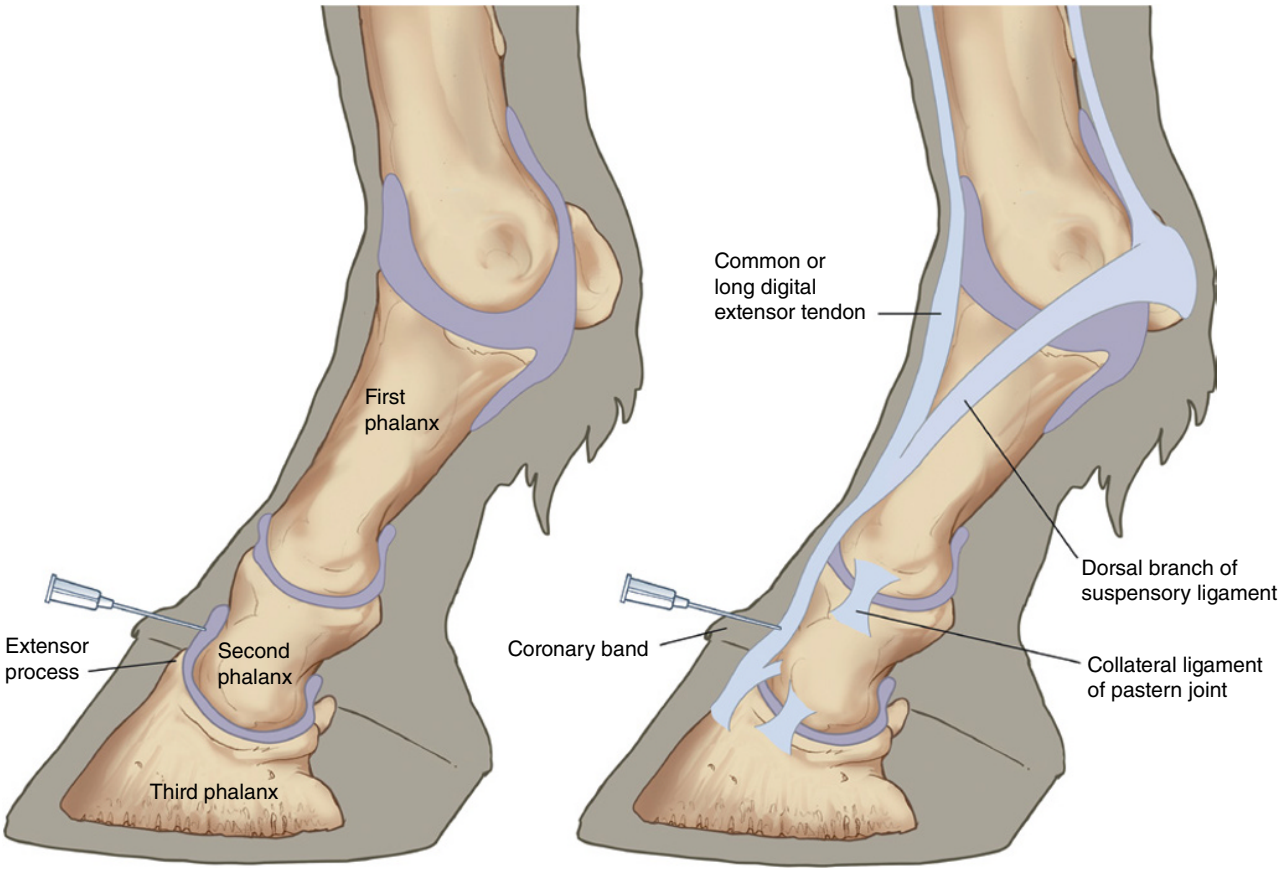


Figure 2.162. The dorsal parallel approach to the DIP joint. The needle is inserted just above the coronary band directly into the proximal pouch of the joint.

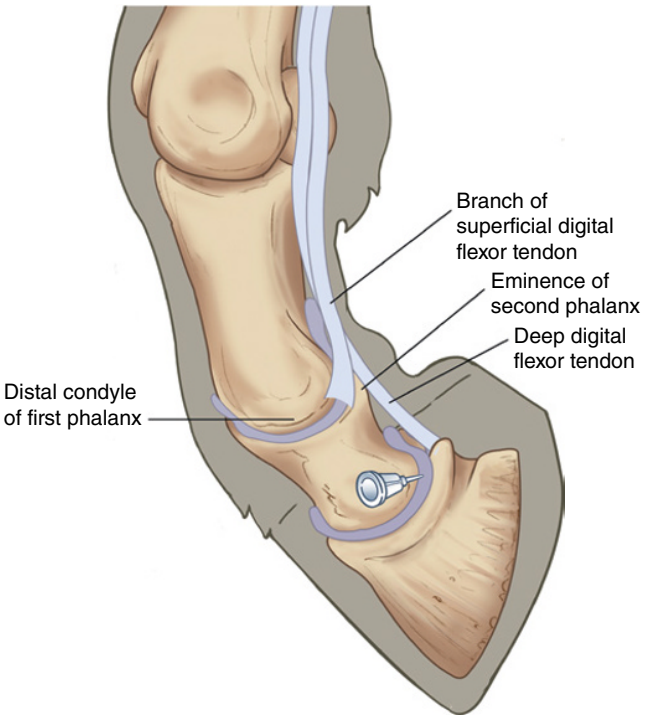


Figure 2.163. The lateral approach to the DIP joint. This technique can be performed in the horse standing or with the limb slightly flexed.

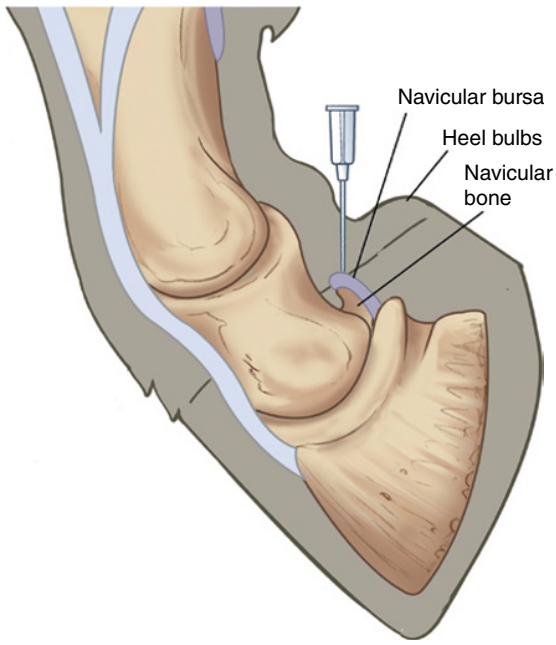


Figure 2.164. Lateral view of the foot demonstrating the approximate location of the spinal needle to enter the navicular bursa using the palmar approach between the heel bulbs with the distal limb slightly flexed.

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perform this block (Figure 2.164). Alternatively, the forelimb can be suspended with the metacarpus placed within a padded hoof stand to help facilitate the injection. Radiographic or fluoroscopic documentation of the needle's location is recommended in most cases because it is easy to pass the needle over to the proximal border of the navicular bone into the DIP joint.[28,48] Including radiographic contrast medium into the injection solution and taking a radiograph immediately after the injection can also be used to document a successful injection. The disadvantage of this technique is that the needle passes through the DDFT to gain access to the bursa and is difficult to perform in the standing horse.

The navicular bursa can also be entered from the lateral or medial sides (abaxial or lateral position) just proximal to the collateral cartilage of the distal phalanx. Two lateral techniques that do not penetrate the DDFT have recently been described.[14,50] One technique was performed in the standing horse using radiographic guidance, and the other used ultrasound to help guide the needle into the bursa with the foot placed in a navicular block. Needle entry was along the proximal and dorsoproximal margin of the collateral cartilage, and an 18- to 20-gauge, 3.5-inch (8.9-cm) needle was angled approximately 45° to the horizontal plane toward the 10 o'clock (right front) or 2 o'clock (left front) position (Figure 2.165).[14] The needle was directed beneath the DDFT and digital tendon sheath to enter the bursa. This technique can also be performed with the limb unweighted (Video 2.19). In one study needle placement was confirmed with radiography,[14] while ultrasound was used in the other study.[50] The primary advantage of these techniques is that the needle does not penetrate the DDFT, which may decrease the morbidity associated with the injection. The disadvantages include possible entry into the tendon sheath and DIP joint and the difficulty of placing the needle at the correct angulation. As with all navicular bursal injections, confirmation of correct needle placement is recommended with some type of imaging either before or after the injection.

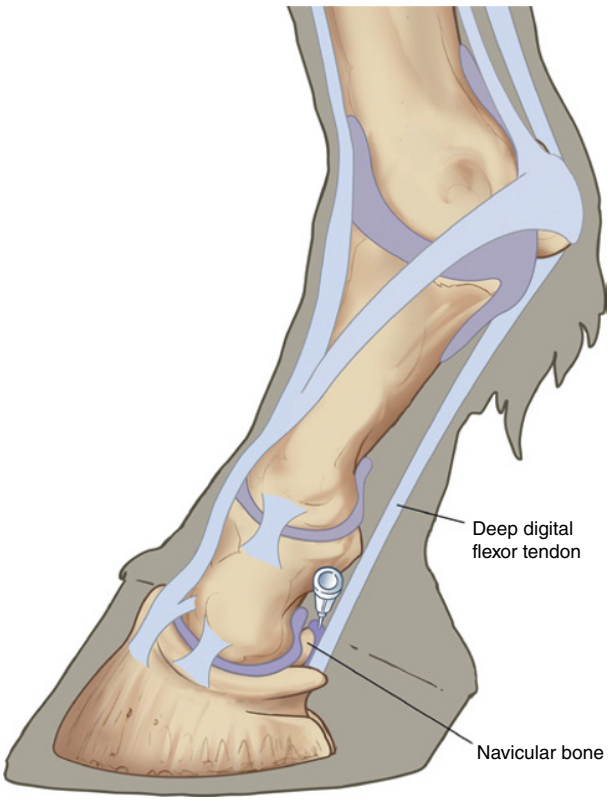


Figure 2.165. Lateral approach to the navicular bursa that avoids penetrating the DDFT. The spinal needle is inserted just above the collateral cartilage and angled at approximately 45° to the horizontal plane to enter the bursa.

A positive response to administration of local anesthesia into the navicular bursa may indicate problems of the navicular bursa, navicular bone and/or its supporting ligaments, sole and/or toe, or distal aspect of the DDFT.[17,48,63,66] Even though diffusion of local anesthetic into the navicular bursa occurs following DIP joint injection,[6–8,39,52] the converse does not occur, and analgesia of the navicular bursa does not result in analgesia of the DIP joint.[66] Pain from the DIP joint can likely be excluded as a cause of lameness if analgesia of the navicular bursa improves the lameness within 10 minutes.[66] In addition, a positive response to intra-articular analgesia of the DIP joint together with a negative response to navicular bursa analgesia incriminates pain within the DIP joint as the cause of lameness.[18,66]

Proximal Interphalangeal (PIP) Joint

There are primarily two approaches (dorsolateral and palmar/plantar) for arthrocentesis of the PIP joint. A dorsal approach has been described but is more difficult to perform[53] and is not recommended by the author. The dorsolateral approach is usually performed with the horse standing but can be done with the limb extended and the sole supported on the knee. The condylar eminences of the distolateral aspect of P1 are identified, and a 1.5-inch (3.8-cm), 20-gauge needle is inserted parallel to the ground surface 0.5 inches (1.2 cm) distal to the palpable eminence.[48,75] The needle is directed beneath the edge of the extensor tendon to enter the joint at a depth of 0.5 inches (Figure 2.166; Video 2.20).

The palmaro/plantaroproximal approach is best performed with the distal limb in a flexed position. A 1.5-inch (3.8cm), 20-gauge needle is inserted perpendicular to the limb into the palpable V-depression formed by the palmar aspect of P1 dorsally, the distal eminence of P1 distally, and the lateral branch of the SDFT as it inserts on the eminence of P2 palmarodistally (Figure 2.167; Video 2.21).[45] This corresponds to the transverse bony prominence on the proximopalmar/plantar border of P2 that is usually easily palpable. The author prefers to angle the needle slightly dorsally to contact P1, and then direct the needle along the palmar/plantar aspect of the bone. This ensures that the needle is just behind P1 where it will enter the PIP joint capsule at a depth of approximately 1 inch (2.5 cm). The main disadvantage of this approach is the inadvertent injection of the digital flexor tendon sheath (DFTS).[53]

Metacarpophalangeal/Metatarsophalangeal (Fetlock) Joints

Three palmar/plantar approaches and one dorsal approach can be used for arthrocentesis of the fetlock joint. The proximal palmar/plantar approach can be

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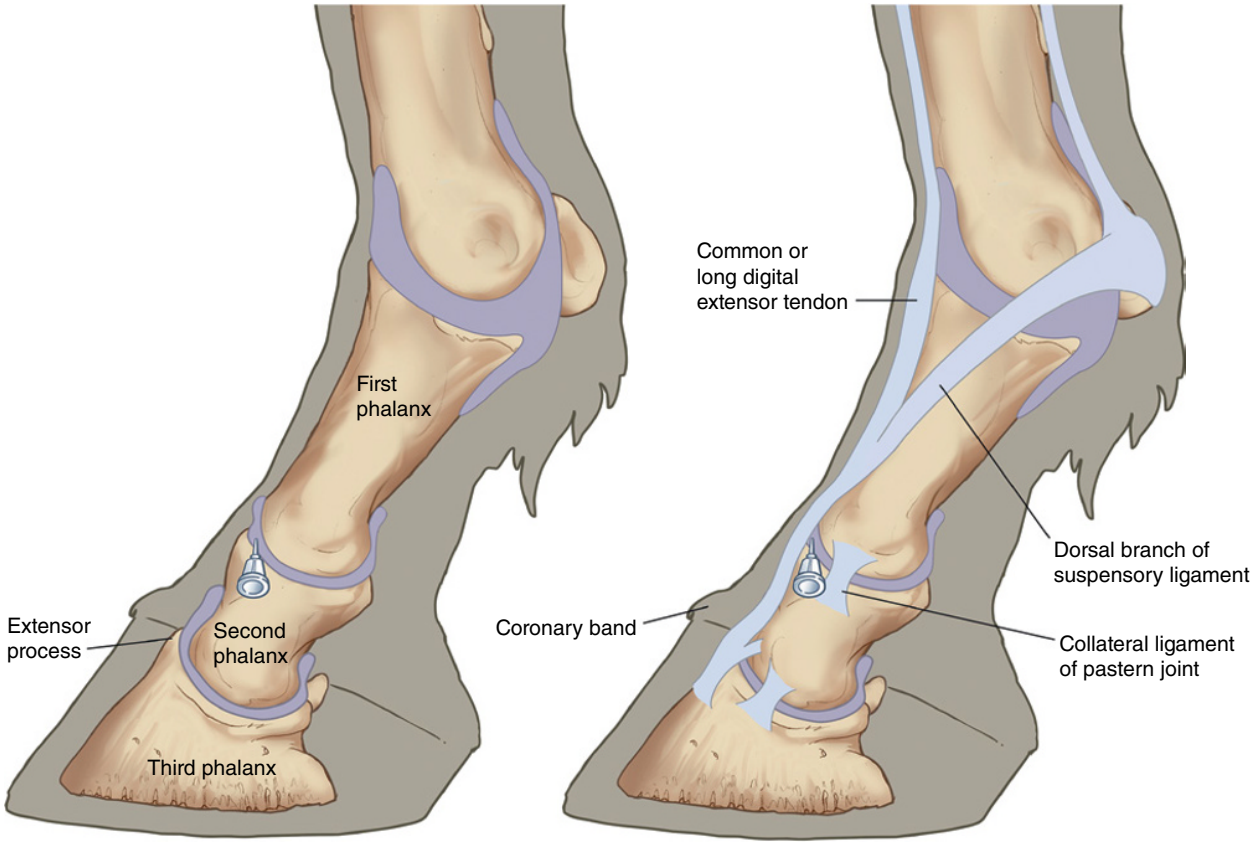


Figure 2.166. Dorsolateral approach to the PIP joint. The needle is inserted at distal border of the P1 condyle beneath the extensor tendon.

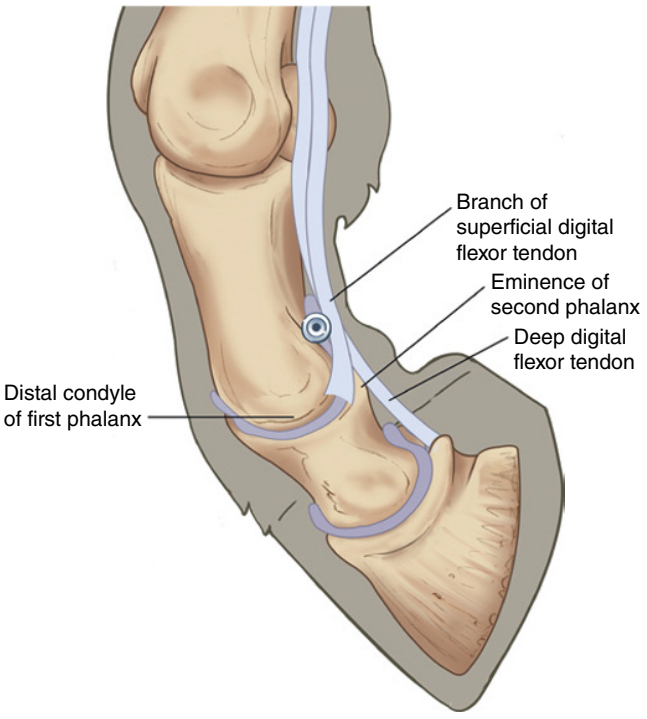


Figure 2.167. Palmar/plantar approach to the PIP joint. The limb is held to relax the palmar structures, and the needle is inserted in a V-shaped depression formed by the palmar aspect of P1 dorsally, the distal eminence of P1 distally, and the lateral branch of the SDFT as it inserts on the eminence of P2 palmarodistally.

performed with the limb weight-bearing or held, the approach through the collateral sesamoidean ligament must be performed with the fetlock joint flexed, and the distal palmar/plantar and dorsal approaches are usually performed in the standing limb. The dorsal and proximal palmar/plantar approaches are usually reserved for horses with significant effusion to avoid damage to the articular cartilage and inadvertent hemorrhage, respectively. Which technique to use is often based on personal preference, although some are easier to perform in the forelimb than the hindlimb.

Proximal Palmar/Plantar Pouch

The boundaries of the palmar/plantar pouches of the fetlock joint are the apical border of the proximal sesamoid bones distally, the distal ends of the splint bones proximally, the third metacarpal/metatarsal bone dorsally, and the branch of the suspensory ligament palmar/plantarly (Figure 2.168). In the normal horse, these pouches appear as a depression, and attempts to retrieve synovial fluid may be difficult. However, they are usually easily identified in horses with fetlock effusion. When performing this approach in the standing patient, a 1-inch (2.5-cm), 20-gauge needle is inserted from lateral to medial and directed distally at a 45° angle to the long axis of the limb (Video 2.22). The disadvantages of this approach are the possibility of contaminating the synovial fluid sample with blood because of the highly vascular synovial membrane and the inability to aspirate

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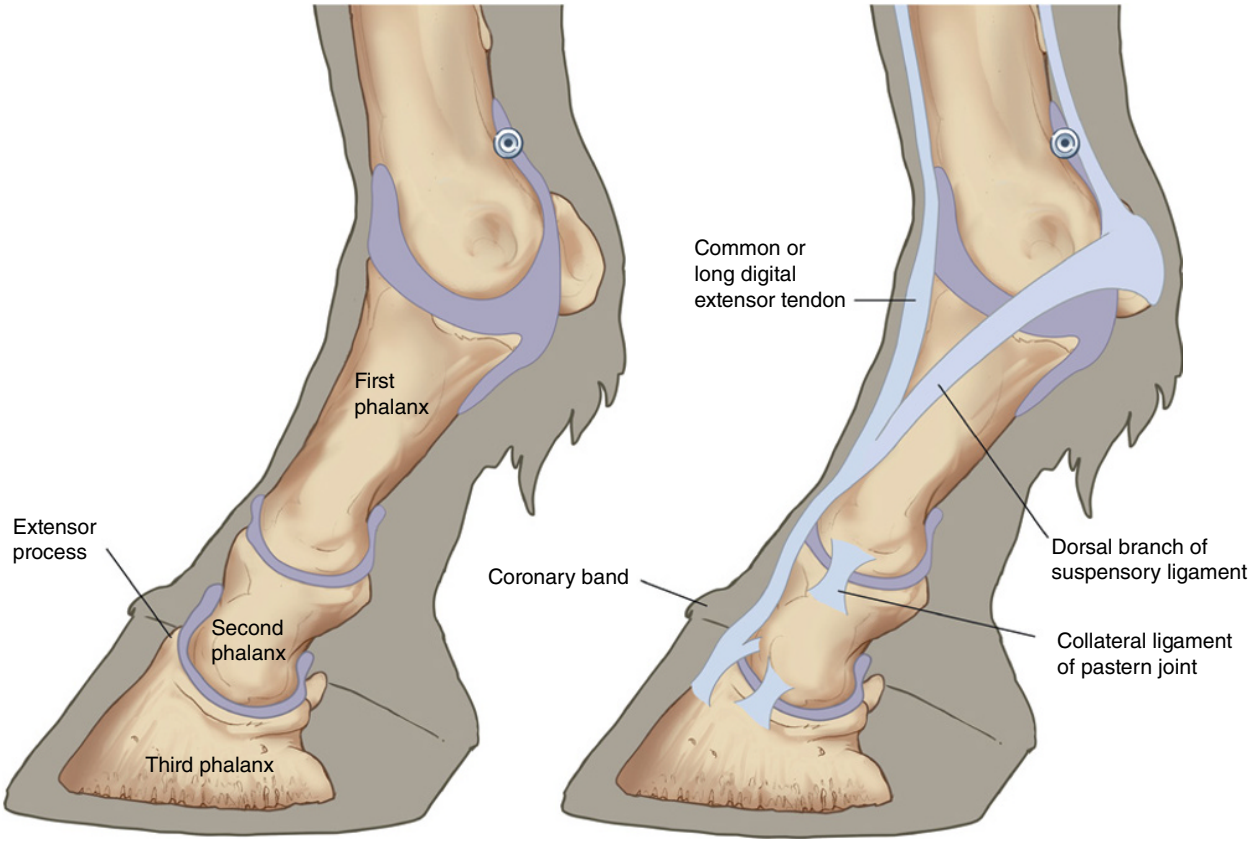


Figure 2.168. Location for injecting the proximal palmar/plantar pouch of the fetlock joint in the standing horse.

synovial fluid because the synovial villi plug the needle.[34,46]

Performing the palmar/plantar approach with the fetlock flexed can potentially minimize these complications (Figure 2.169; Video 2.23). With the fetlock flexed, there is a very palpable depression at the very distal aspect of the pouch just above the branch of the suspensory ligament. A 1-inch (2.5-cm), 20-gauge needle is inserted at this location and directed distally at a 45° angle. The more distal location in the palmar/plantar pouch reduces the risk of iatrogenic hemorrhage.

Collateral Sesamoidean Approach

Arthrocentesis of the fetlock through the lateral collateral sesamoidean ligament is probably the best approach to obtain a hemorrhage-free synovial fluid sample. The fetlock is flexed to increase the space between the articular surfaces of the proximal sesamoid bones and the metacarpus/metatarsus. The depression between the bones is palpated, and a 1-inch, 20-gauge needle inserted through the collateral sesamoidean ligament perpendicular to the limb (Figure 2.170; Video 2.24).[46] If the needle fails to advance, it is most likely contacting bone and will need to be redirected to enter the joint space.

Distal Palmar/Plantar Approach

The distal palmar/plantar approach is performed in the palpable depression formed by the distal aspect of the proximal sesamoid bone and the proximopalmar/plantar eminence of P1. The landmarks are the distal aspect of the proximal sesamoid bone and collateral sesamoidean ligament proximally, the proximal palmar/plantar eminence of P1 distally, and the digital vein, artery, and nerve palmar/plantarly.[72] A 1.5-inch (3.8-cm), 20-gauge needle is inserted in the depression and directed slightly dorsally (10°–20°) and proximally (10°) until the joint is entered (Figure 2.171; Video 2.25). To avoid penetration of the digital sheath, it is important that the needle be inserted dorsal to the PD artery, vein, and nerve. The advantages to this approach are that the landmarks are easily palpable, synovial fluid is often obtained, it can be performed in the standing horse, and horses tolerate the procedure well.[72]

Dorsal Approach

The dorsal approach is usually performed with the limb weight-bearing. The needle is inserted proximal to the proximodorsal limits of P1 in the palpable joint space in a slightly oblique manner, either lateral or medial to the extensor tendon (Figure 2.172; Video 2.26). The fetlock joint capsule is thicker in this location than in the palmar/plantar pouch and appears to cause greater discomfort to the horse than the other techniques.

Digital Flexor Tendon Sheath (DFTS)

There are several outpouchings of the DFTS that may be used for synoviocentesis. In general, the proximal pouch palmar/plantar to the suspensory and the distal

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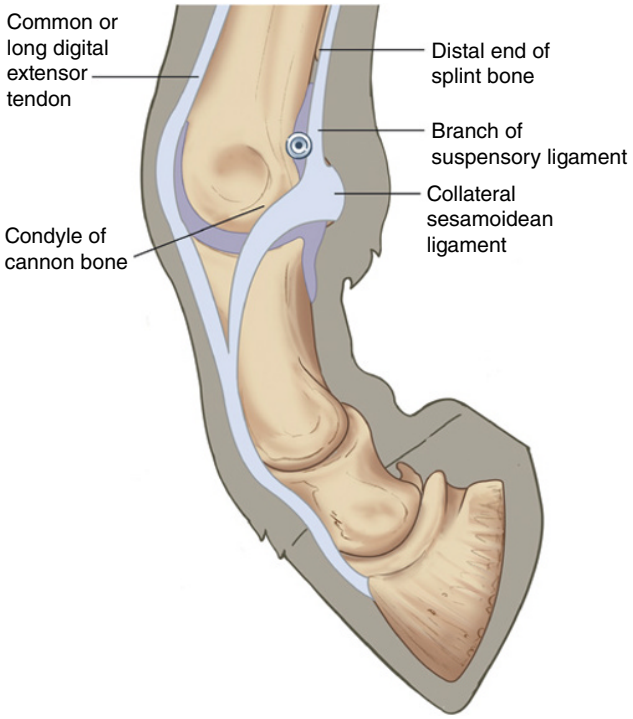


Figure 2.169. Injection of the proximal palmar/plantar pouch of the fetlock joint with the limb flexed.

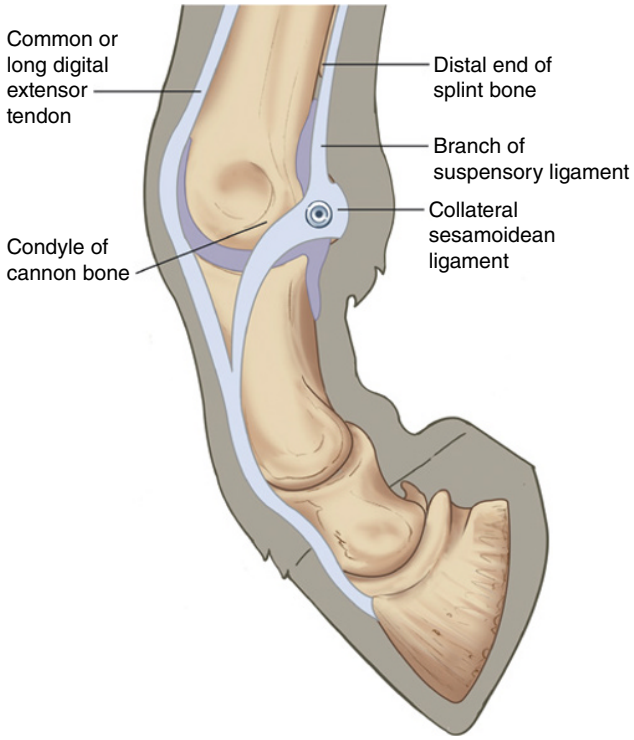


Figure 2.170. Lateral view of the injection site through the collateral sesamoidean ligament into the fetlock joint. Synovial fluid is reliably obtained with this technique.

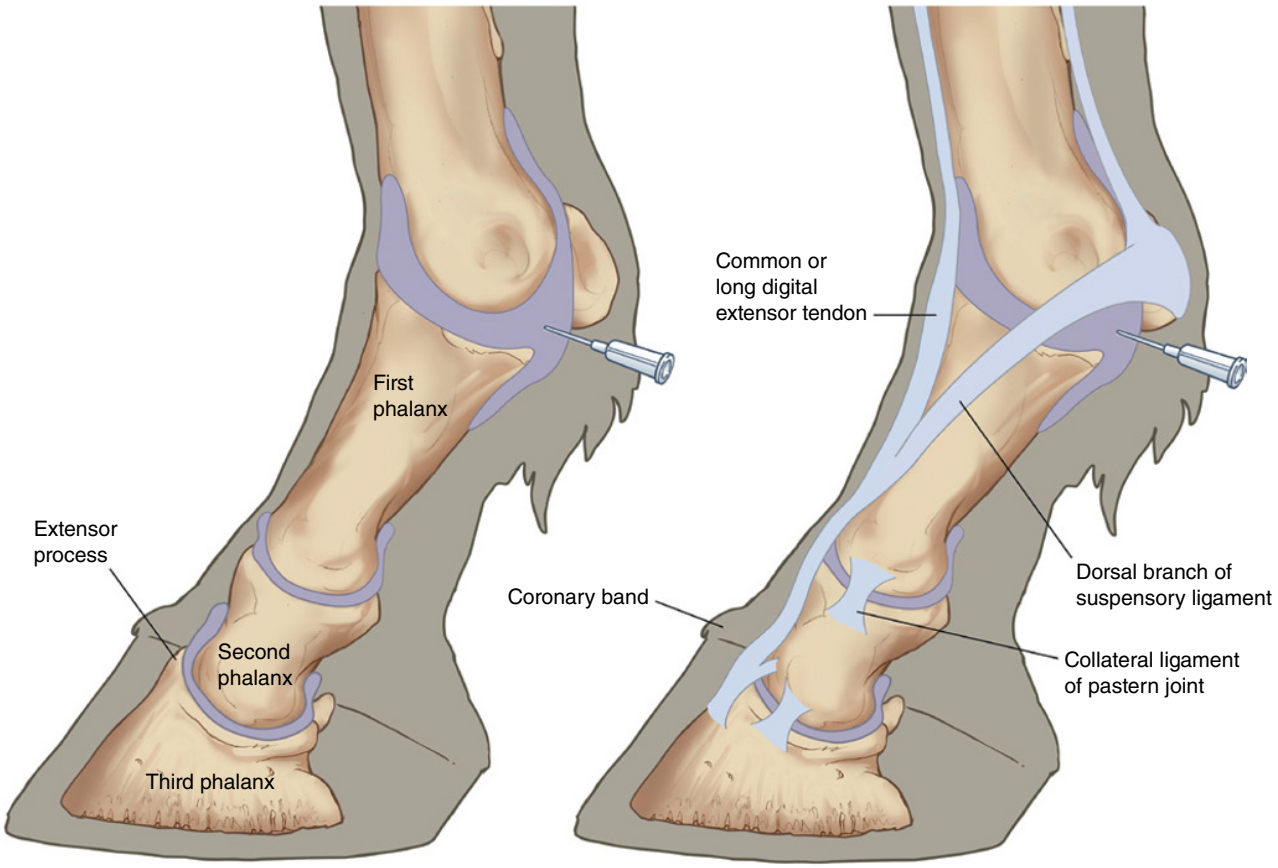


Figure 2.171. Location of injection for the distal palmar/plantar approach to the fetlock joint in the standing horse.

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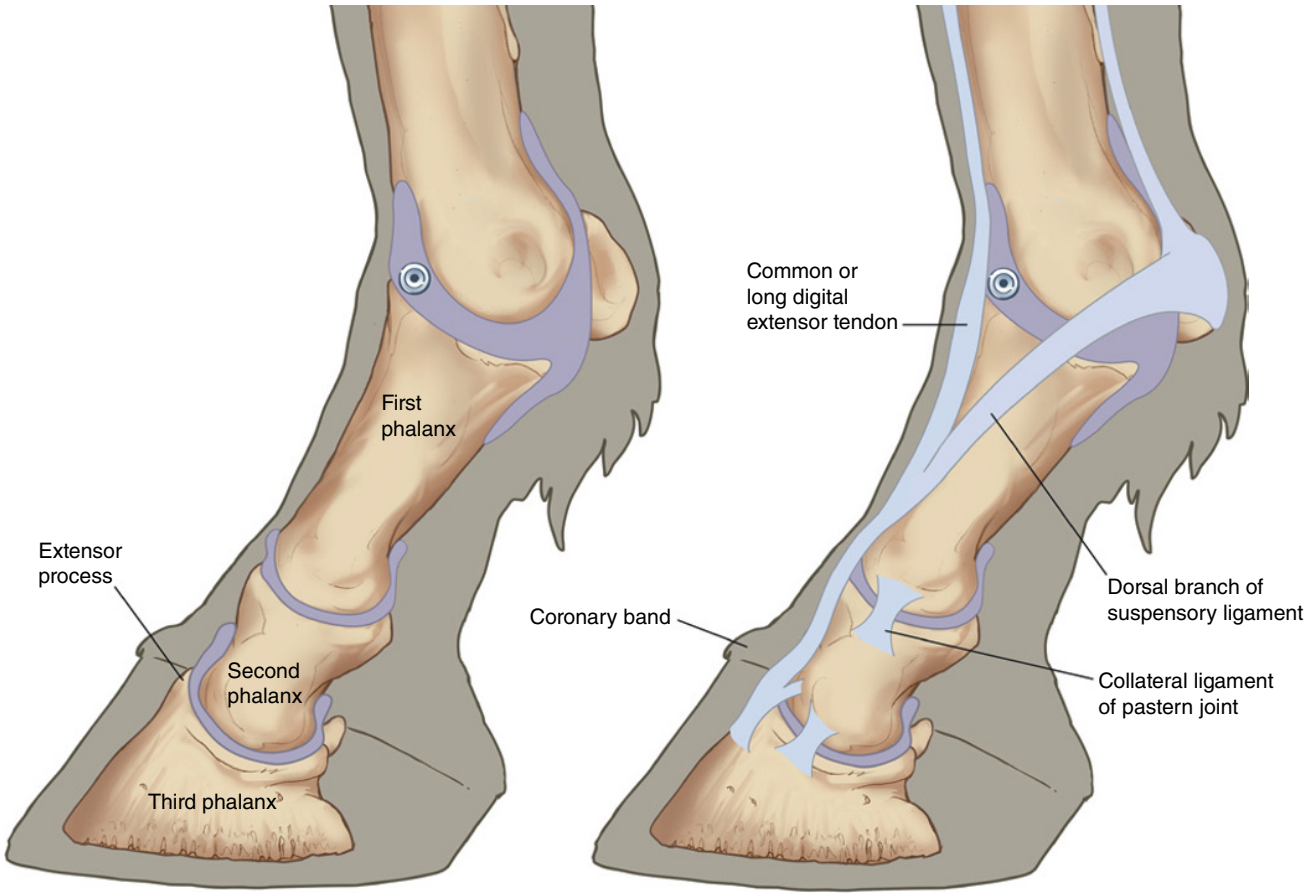


Figure 2.172. Dorsal view of the injection site for the dorsal approach to the fetlock joint in the standing horse.

recess in the pastern superficial to the DDFT are difficult to penetrate without sheath effusion. However, they can often be the easiest approaches to perform if effusion is present. The site for injection of the proximal pouch of the DFTS is 1 cm proximal to the palmar/plantar annular ligament and 1 cm palmar/plantar to the lateral branch of the suspensory ligament (Figure 2.173; Video 2.27). A 1- to 1.5-inch, 20-gauge needle is directed slightly distally until the sheath is penetrated.

The distal outpouching of the DFTS in the pastern region is often palpable as a distinct "bubble" when effusion is present. It is located between the proximal and distal digital annular ligaments and between the diverging branches of the SDFT where the DDFT lies close to the skin.[48] A 20-gauge, 1-inch needle is directed in a lateral to medial direction just beneath the skin so as not to penetrate the DDFT (Figure 2.174; distal needle; Video 28). Alternatively, the needle can be inserted into the outpouching of the DFTS abaxial and distal to the sesamoid bones between the annular and proximal digital annular ligaments. The needle is inserted in a distal to proximal direction at approximately a 45° angle to the sagittal plane (Figure 2.174; proximal needle). This approach is referred to as the basilar sesamoidean approach and can be performed in the standing horse. It has been reported to be more reliable to obtain synovial fluid than the axial sesamoidean approach.[55]

The axial sesamoidean approach at the level of the fetlock and the medial or lateral approach between the annular ligament and proximal digital annular ligament can be performed in the distended and non-distended DFTS. Both approaches are best performed with the limb held with the fetlock slightly flexed. The axial sesamoidean approach is performed 3 mm axial to the palpable border of the midbody of the lateral proximal sesamoid bone using a 1–1.5-inch (2.5–3.8-cm), 20-gauge needle (Figure 2.175; Video 2.29).[31] The needle is directed at a 45° angle to the sagittal plane to a depth of approximately 1.5–2 cm. Ten to 15 mL of anesthetic is recommended for diagnostic purposes for the DFTS. Improvement of lameness in horses after intrasynovial analgesia of the DFTS is usually due to attenuation of pain within the structures contained in the DFTS. Analgesia of the DFTS has little effect on lameness caused by pain originating in the sole, DIP joint, or the navicular bone.[30]

Carpal Joints

Arthrocentesis of the radiocarpal and middle carpal joints can be performed using either a dorsal or palmar approach. The dorsal approach is performed with the carpus flexed, and the palmar approach is performed in the weight-bearing limb and can be somewhat difficult in the non-distended joint. Approximately 10 mL of anesthetic is recommended. Because the carpometacarpal joint and the middle carpal joint communicate, anesthetics injected into the middle carpal joint also desensitize the carpometacarpal joint. Additionally, the carpometacarpal joint has palmar pouches that extend

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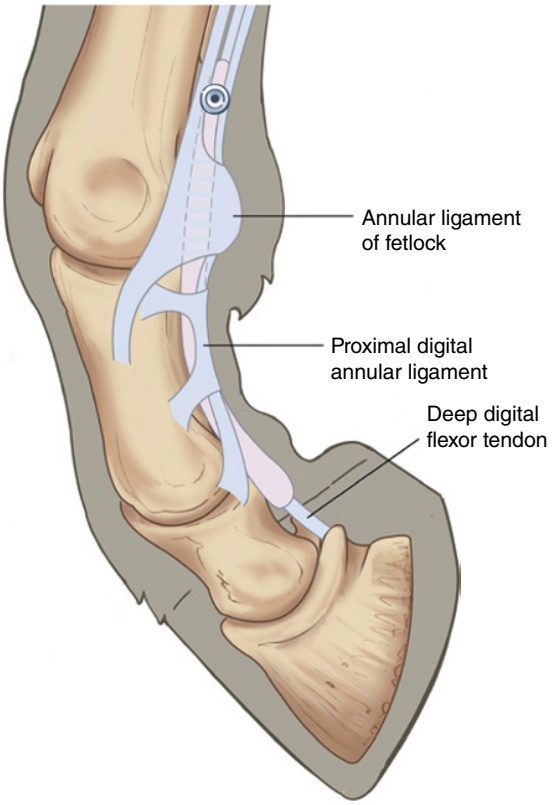


Figure 2.173. The proximal outpouching of the digital flexor tendon sheath can be used for synoviocentesis when the DFTS is distended. It is usually performed with the horse weight-bearing.

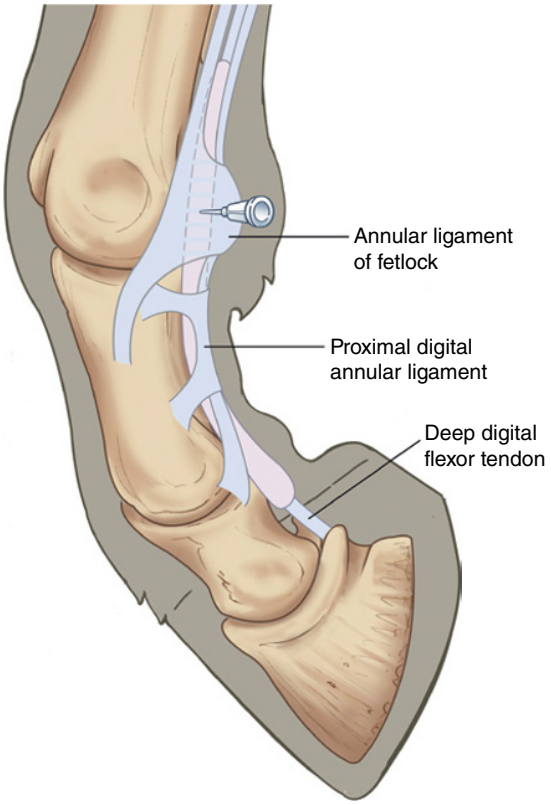


Figure 2.175. Injection site for the axial sesamoidean approach to the DFTS. The needle is inserted on the axial border of the sesamoid bone through the annular ligament.

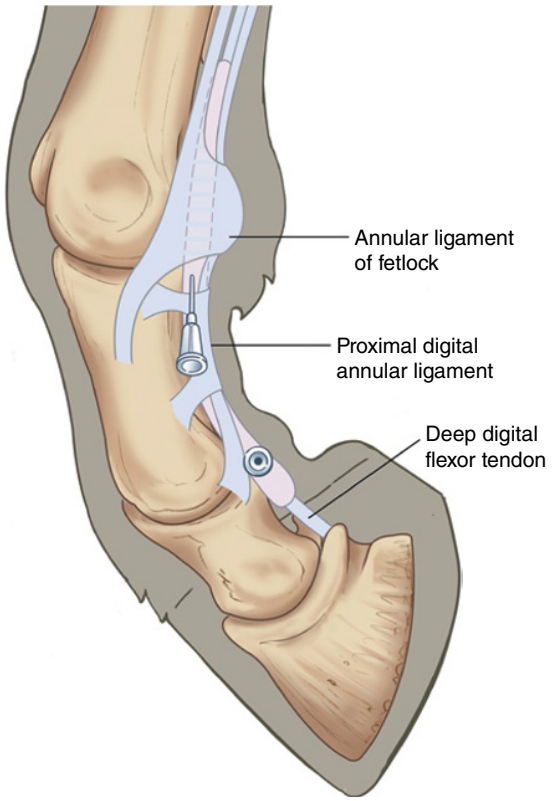


Figure 2.174. Injection site for the distal outpouching of the DFTS (distal needle) and the outpouching of the sheath located abaxial and distal to the sesamoid bones between the annular and proximal digital annular ligaments (proximal needle).

palmarodistally adjacent to the origin of the suspensory ligament. Anesthetic injected into the middle carpal joint may also desensitize the proximal suspensory ligament and proximal palmar metacarpal region.[23,24]

The sites of injection for the radiocarpal and middle carpal joints are located in palpable depressions lateral or medial to the extensor carpi radialis tendon on the dorsal aspect of each joint (Figures 2.176 and 2.177). The injection is made with a 1-inch (2.5-cm), 20-gauge needle midway between the distal radius and proximal row of carpal bones for the radiocarpal joint (Figure 2.177; Video 2.30) or between the proximal and distal rows of carpal bones for the middle carpal joint (Figure 2.176; Video 2.31). Because the surfaces of the carpal bones are at an angle, the needle should be directed slightly proximally to avoid hitting the articular cartilage.[3,75]

The landmarks for the palmarolateral approach to the radiocarpal joint are the palmarolateral aspect of the radius, proximolateral aspect of the accessory carpal bone, and palmarolateral aspect of the ulnar carpal bone (Figure 2.178).[47,79] A 1-inch (2.5-cm), 20-gauge needle is inserted in this palpable depression at 90° to the long axis of the limb, and the needle is directed dorsomedially (Video 2.32). Another palmarolateral approach is at the midaccessory carpal bone level in a palpable "V" between the tendons of the ulnaris lateralis and the lateral digital extensor.[41,48] The needle is inserted perpendicular to the skin in a small depression 0.5–1 inch (8–12 mm) distal to the "V" in the space between the distal lateral aspect of the radius (vestigial ulna) and the proximal lateral aspect of the ulnar carpal bone.[47,48]

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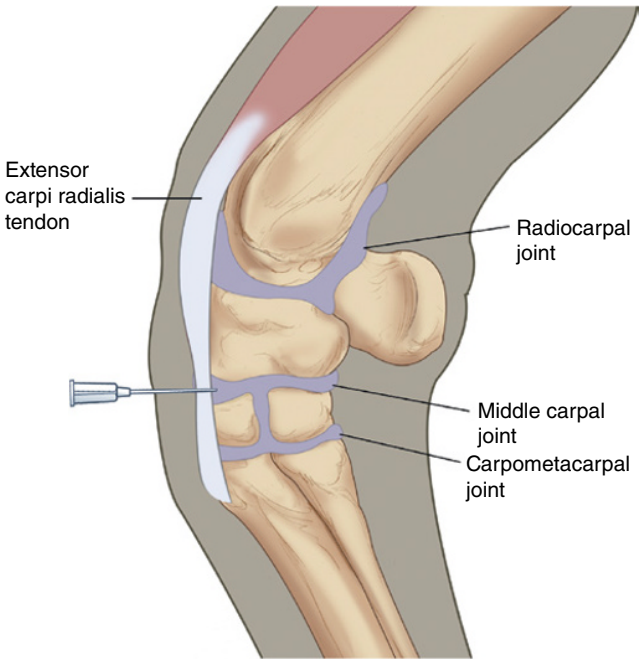


Figure 2.176. Dorsal approach to the middle carpal joint with the carpus flexed. Needles can be placed either lateral or medial to the extensor carpi radialis tendon.

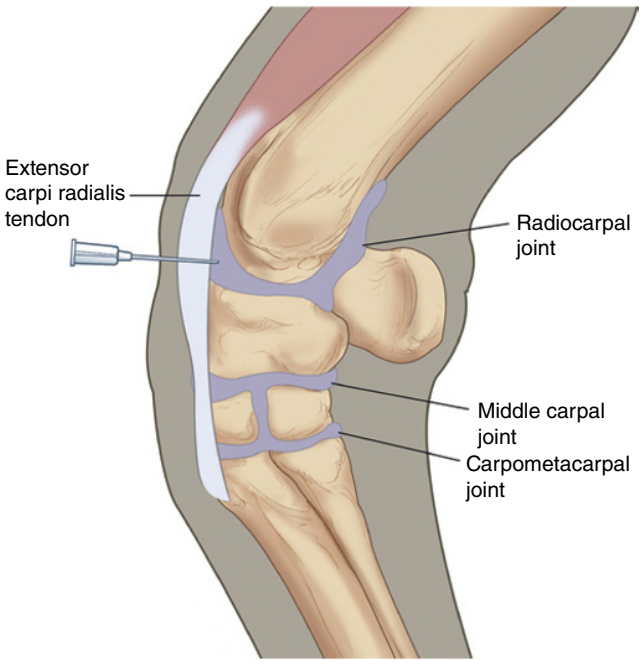


Figure 2.177. Dorsal approach to the radiocarpal joint with the carpus flexed. Needles can be placed either lateral or medial to the extensor carpi radialis tendon.

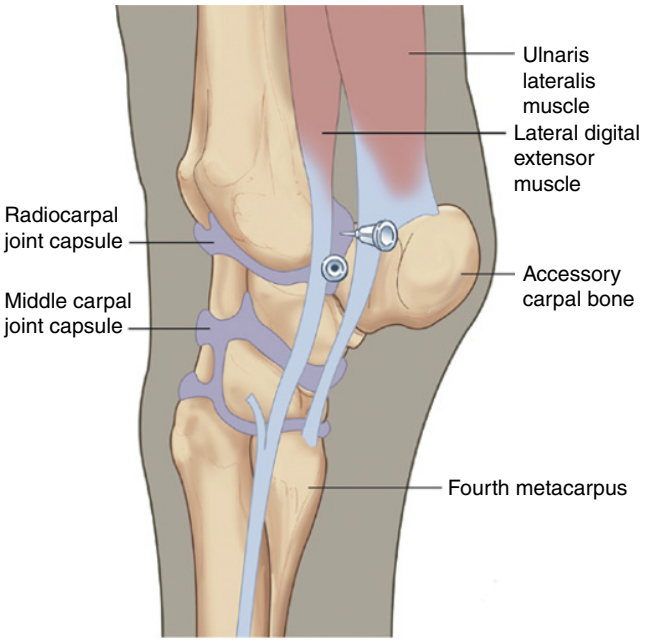


Figure 2.178. Lateral view of the palmarolateral injection sites for the radiocarpal joint in the standing horse.

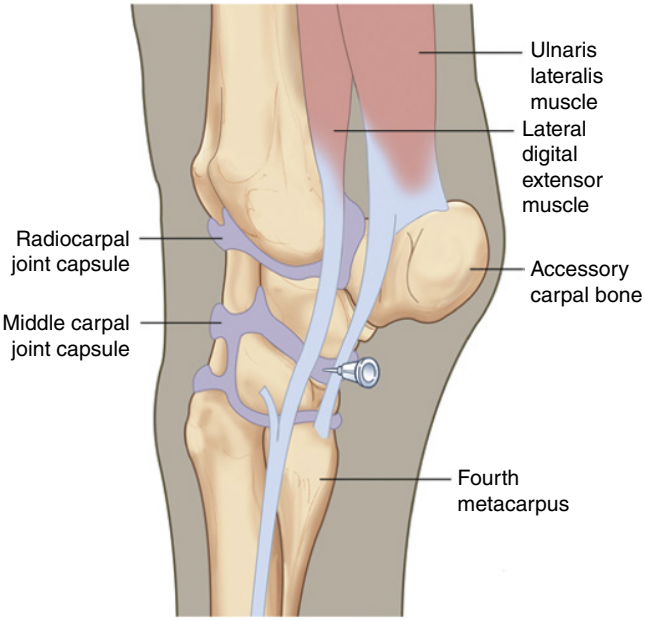


Figure 2.179. Lateral view of the palmarolateral injection site for the middle carpal joint in the standing horse. The palmar joint pouch can be difficult to palpate without the presence of synovial effusion.

The palmarolateral approach to the middle carpal joint is best used if the joint is distended (Figure 2.179; Video 2.33). With distension the joint capsule is superficial and protrudes palmar and lateral to the ulnar and fourth carpal bones distal to the accessory carpal bone. The injection site is approximately 1 inch (2.5 cm) distal to the site of injection of the radiocarpal joint.[2,75] A 1-inch, 20-gauge needle is inserted perpendicular to the skin to a depth of about 0.5 inches (1.2 cm).

Elbow Joint

There are three major approaches to the elbow joint: lateral, caudolateral, and caudal. The lateral approach is within the radiohumeral articulation, whereas the caudolateral and caudal approaches are within the humeroulnar articulation. All three techniques are best performed

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while the horse is standing, but can be done with the limb flexed.[75] Injection of the elbow is more difficult than many other joints in the horse, and approximately 20–30 mL of anesthetic is needed for diagnostic purposes.[48]

Lateral Approach

The landmark for the lateral approach is the lateral collateral ligament that extends across the joint from the lateral epicondyle of the humerus to the lateral tuberosity of the radius. Both of these bony landmarks are easily palpated. The elbow joint can be entered either cranial or caudal to the collateral ligament. The site for injection is two-thirds the distance distally measured from the lateral epicondyle of the humerus to the lateral tuberosity of the radius (Figure 2.180).[34,75] A 1.5-inch (3.8-cm), 20-gauge needle is inserted at a 90° angle to the skin just cranial or caudal to the lateral collateral ligament to a depth of 1 inch.[34,48] If injected cranially, it is important to verify that the needle is within the joint because periarticular anesthetic may desensitize the distal branches of the radial nerve, causing temporary paralysis of the extensor carpi radialis and common digital extensor muscles. This will cause the horse to be unable to lock its carpus in extension. If injected caudally, the needle may enter the bursa of the ulnaris lateralis muscle, which is thought to communicate with the elbow joint.[58] However, communication between the bursa and the elbow joint occurred in only 9 of 24 (37.5%) of the joints examined.[58] To avoid the ulnaris lateralis bursa, the needle may be inserted more caudally in the elbow in a palpable depression formed by the caudal epicondyle of the humerus, the caudal proximal tuberosity of the radius, and the anconeal process (Figure 2.181).[75]

Caudolateral Approach

The caudolateral approach is an alternative to placing the needle directly caudal to the collateral ligament using the lateral approach. The injection site is caudal to the palpable humeral epicondyle in the anconeal notch within the humeroulnar joint. This palpable V-shaped depression is usually just below the triceps muscles and 6–8 cm cranio-distal from the point of the olecranon process.[2,3,75] A 1.5- to 3.5-inch (3.8- to 8.8-cm), 20-gauge needle is inserted at a 45° angle to the skin and directed craniomedially (Figure 2.181).

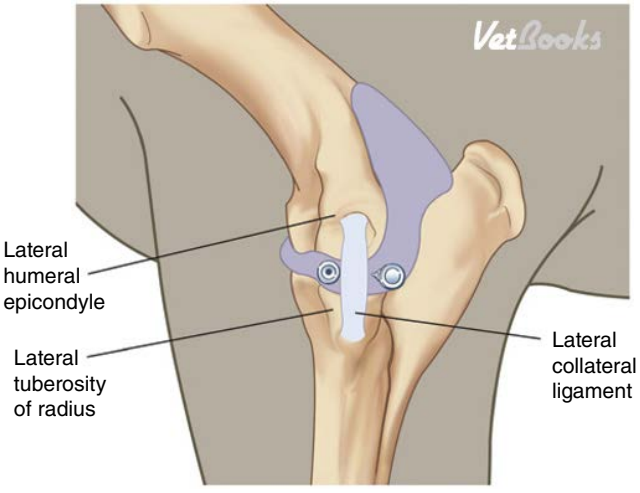


Figure 2.180. The lateral approach to the elbow joint can be made either cranial or caudal to the collateral ligament.

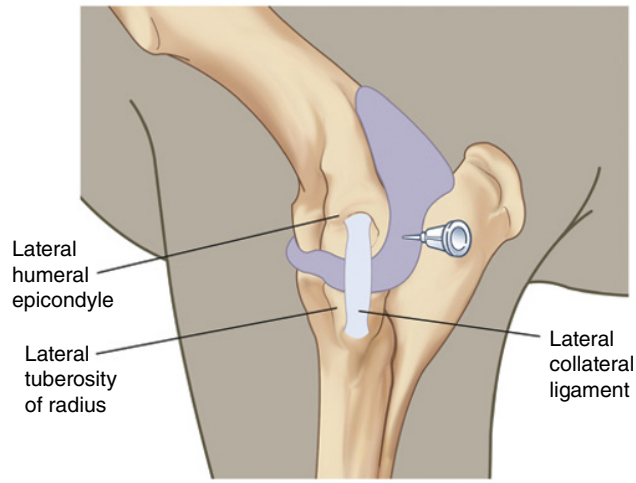


Figure 2.181. Caudolateral approach to the elbow joint just is caudal to the palpable humeral epicondyle in the anconeal notch within the humeroulnar joint.

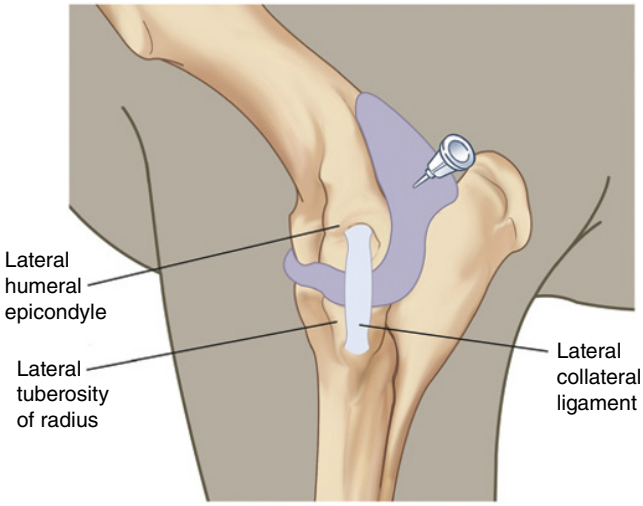


Figure 2.182. The approach to the large caudal outpouching of the elbow joint is 0.5 inches (1 cm) proximal to and one-third of the distance measured caudally from the supracondylar eminence to the point of the olecranon. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is directed distomedially through the triceps musculature at a 45° angle to the long axis of the limb into the olecranon fossa.

Caudal Approach

The large caudal joint pouch of the elbow can be entered from a more proximal location (Figure 2.182). The landmarks are the lateral supracondylar crest of the distal humerus and the most proximal point of the olecranon process.[34,48,58] The injection site is 0.5 inches (1 cm) proximal to and one-third of the distance measured caudally from the supracondylar eminence to the

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point of the olecranon. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is directed distomedially through the triceps musculature at a 45° angle to the long axis of the limb into the olecranon fossa (Figure 2.182). Alternatively, the spinal needle may be directed downward along the lateral shaft of the olecranon process to enter the joint.[75] The injection site is 3 cm distal and 2 cm cranial to the point of the olecranon, and the needle is directed distally and cranially to enter the caudal joint pouch just proximal to the anconeal process.

Scapulohumeral (Shoulder) Joint

Arthrocentesis of the shoulder can be difficult due to the depth of the joint. It is a large joint, and 20–40 mL of anesthetic is usually used for diagnostic purposes. It is always performed with the horse standing, and many clinicians now utilize ultrasound to guide the injection. Ultrasound-guided injections of the scapulohumeral joint and bicipital and infraspinatus bursae have been reported to be highly reliable and more accurate than conventional blind techniques.[59] The shoulder joint may communicate with the bicipital bursa in a small percentage of horses, and temporary anesthesia of the suprascapular nerve and paralysis of the infraspinatus and supraspinatus muscles may occur with periarticular injection of anesthetic.[48,75]

Craniolateral Approach

The site for the craniolateral approach to the shoulder joint is located in the notch formed between the cranial and caudal prominences of the lateral tuberosity of the humerus. The caudal prominence (point of the shoulder) is easiest to palpate, and by exerting deep finger pressure, the depression for needle insertion can be palpated 3.5–4 cm cranial to the caudal prominence. This notch is not as readily palpable in heavily muscled horses. A 3.5-inch (8.8-cm), 18- to 20-gauge spinal needle is inserted into this notch and directed parallel to the ground in a caudomedial direction toward the opposite elbow.[48,75] The depth of penetration depends on the size of the horse, but the joint capsule is usually entered at a depth of 2–3 inches (5–7 cm). Synovial fluid can usually be aspirated and is the only definitive method to document correct needle placement. Alternatively, the spinal needle may be inserted slightly more proximal on the limb in a distinct depression located 1–1.5 cm cranial to the infraspinatus tendon and slightly proximal and cranial to the point of the shoulder. The needle is placed parallel to the ground or slightly downward and directed caudomedially at a 45° angle until bone is contacted (Figure 2.183).

Lateral Approach

The landmarks for the lateral approach to the shoulder are the lateral humeral tuberosity and the infraspinatus tendon. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is inserted 1–2 cm caudal and distal to the infraspinatus tendon in line with the lateral humeral tuberosity (Figure 2.183).[3,75] The needle is directed slightly caudally and upward toward the lateral aspect of the humeral head. In general, this approach is more difficult than the craniolateral approach.

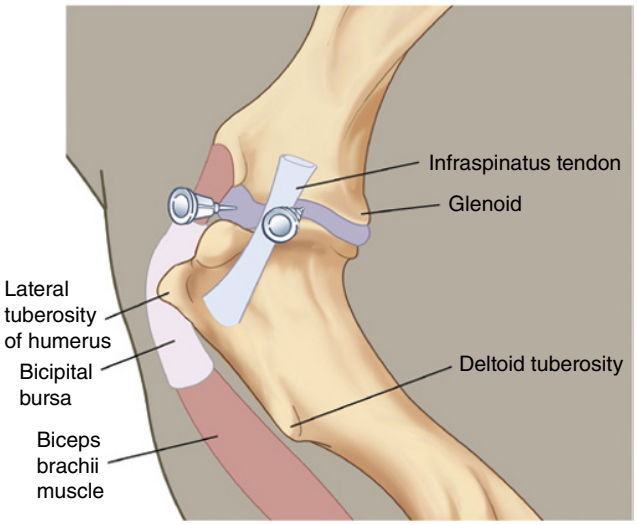


Figure 2.183. Lateral view of the shoulder demonstrating the injection sites for the craniolateral (cranial to the infraspinatus tendon) and lateral (caudal to the infraspinatus tendon) approaches. Ultrasound guidance of the needles can be advantageous with these approaches.

Bicipital Bursa

The bicipital bursa lies cranial to the shoulder and humerus under the biceps brachii muscle. It is a relatively large synovial structure (20–30 mL of anesthetic) but can be difficult to enter because of its depth and the landmarks for injection are not easily palpable. In a recent study that compared two different injection techniques (distal and proximal) to enter the bicipital bursa, the accuracy of injecting the bursa was only 28% and 39%, respectively.[67] The authors concluded that clinicians without previous experience of injecting the bicipital bursa were unlikely to be successful with either approach. This study confirmed the difficulty of injecting the bicipital bursa and is why ultrasound guidance is recommended to improve the accuracy of injections.[59] Alternatively, radiographic examination after injecting radiopaque contrast medium may be used to assess the success of centesis if synovial fluid is not obtained. The proximal and distal approaches to the bursa are usually performed with the limb weight-bearing, but the proximal approach may be done with the limb held.

Distal Approach

The cranial prominence of the lateral tuberosity of the humerus is used as the landmark, as was done for the shoulder joint. The site of injection is 2.5 inches (5–6 cm) distal and 3 inches (7–8 cm) caudal to this prominence. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is directed proximomedially toward the intertubercular groove until it contacts the humerus (Figure 2.184). The depth of the needle depends on the size of the horse, but a 3.5-inch (8.9-cm) spinal needle is usually inserted to the hub in most mature horses.[48] Alternatively, the deltoid tuberosity of the humerus can be palpated and used as a landmark. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is inserted 1.5 inches (3–4 cm) proximal to the distal aspect of the deltoid

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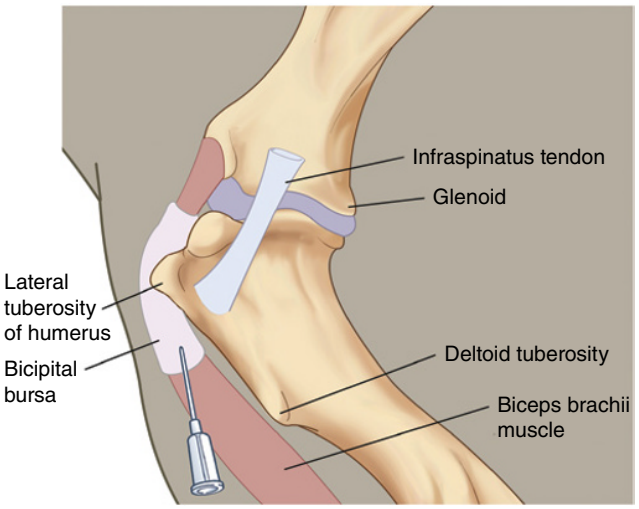


Figure 2.184. Site for the distal approach to the bicipital bursa is located is 2.5 inches (5–6 cm) distal to lateral tuberosity of the humerus.

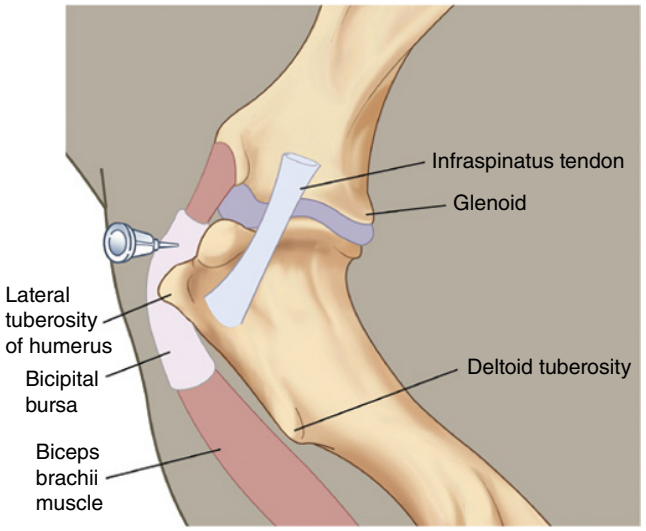


Figure 2.185. Site for the proximal approach to the bicipital bursa is located in the intertubercular groove, which can be palpated medial to the edge of the cranial prominence of the lateral tuberosity of the humerus.

tuberosity and directed proximomedially (toward to opposite ear) to a depth of 2–3 inches (5–7 cm).[48,75]

Proximal Approach

The proximal approach is performed in the intertubercular groove, which can be palpated medial to the edge of the cranial prominence of the lateral tuberosity of the humerus.[48,67] A 1.5-inch (3.8-cm), 20-gauge needle is inserted into the intertubercular groove in a plane parallel to the bearing surface of the foot at about a 45° angle to the sagittal axis of the horse until the needle strikes cartilage (Figure 2.185). The primary advantages of the proximal approach compared to the distal approach are a slightly improved accuracy of entering the bursa and not needing a 3.5-inch (8.9-cm) spinal needle.

Tarsal (Hock) Joints

There are four joint spaces associated with the tarsus: tarsocrural (TC), proximal intertarsal (PIT), distal intertarsal (DIT), and TMT. The TC joint is considered a high-motion joint, whereas the PIT, DIT, and TMT joints are low-motion joints. Numerous studies have been done to determine the consistency of communication between these joints. The TC and PIT joints communicate consistently and are usually considered as one joint as far as intra-articular injection. Reports of communication between the DIT and TMT joints vary from 8% to 38%.[4,20,42] To complicate things further, the PIT and DIT and the PIT and the TMT joints may also communicate occasionally.[42] Because of these findings, the interpretation of intra-articular anesthesia in the tarsus is not always straightforward. In addition, the communication patterns may differ in the diseased tarsus compared with a normal tarsus, and diffusion of medication may occur between the TMT and DIT joints regardless of the communication pattern. Corticosteroids injected into the TMT joint were consistently found within the DIT joint in all horses sampled.[70]

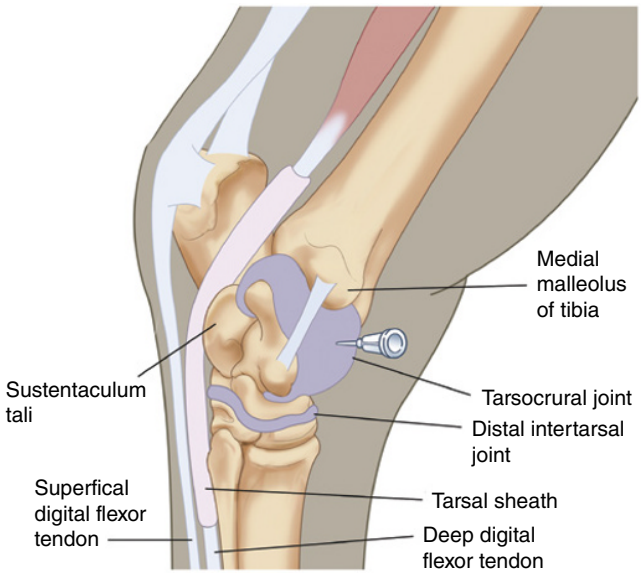


Figure 2.186. Medial view of the injection site for the dorsomedial pouch of the tarsocrural joint.

Tarsocrural Joint

The TC joint is the largest joint pouch in the tarsus and is easy to enter, especially if synovial effusion is present. The joint may be entered dorsally (dorsomedial) or plantarly depending on the clinical situation. For the dorsomedial approach, a 1-inch (2.5-cm), 20-gauge needle is inserted 1–1.5 inches (2–3 cm) distal to the medial malleolus of the tibia, medial, or lateral to the cranial branch of the medial saphenous vein (Figure 2.186; Video 2.34). The needle is advanced in a plantarolateral direction at approximately a 45° until synovial fluid flows from the needle. The dorsomedial approach is usually performed in the weight-bearing limb from the opposite side of the horse, but can be performed from

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the same side of the horse. Fifteen to 20 mL of local anesthetic is recommended.

The medial or lateral plantar outpouchings of the TC joint may be used for arthrocentesis, especially if significant synovial effusion is present (Figures 2.186 and 2.187). The palpable landmarks of the lateral plantar pouch are bordered by the tuber calcis caudally, the caudal aspect of the distal tibia cranially, and the proximal aspect of the lateral trochlear ridge of the talus distally (Figure 2.187; Video 2.35).[34] Confirmation that fluid swellings in this location are part of the TC joint can be determined by applying finger pressure to the swellings and feeling the dorsal pouches of the TC joint distend. A 1-inch (2.5-cm), 20-gauge needle is inserted perpendicular to the skin at the site of the effusion with the limb weight-bearing.

Tarsometatarsal (TMT) Joint

The TMT joint is best approached from the plantarolateral aspect of the tarsus with the limb weight-bearing. It is an easy joint to inject with an accuracy rate of 96% in a recent report.[69] The landmarks for injection are the proximal head of the fourth metatarsal (MT IV) bone and the lateral edge of the SDFT. A 1- to 1.5 inch (2.5- to 3.8-cm), 20- to 22-gauge needle is inserted in the small palpable depression 0.25 inch (0.5–1 cm) proximal to the head of MT IV (Figure 2.188; Video 2.36). The needle is directed toward the dorsomedial aspect of the tarsus in a slightly downward direction to a depth of 0.5–1 inch (1–3 cm).[47,48] Synovial fluid is often observed in the needle hub, and 3–5 mL of anesthetic is used for diagnostic purposes. Injecting the anesthetic under pressure was thought to force anesthetic into the DIT joint, but instead it just causes it to accumulate in the subcutaneous tissues.[48] In addition, the anesthetic may enter the tarsal sheath and extend around the tendons of the tibialis cranialis and fibularis tertius when using this approach.[20] A more difficult alternative approach to the TMT joint is from the distomedial aspect of the tarsus. The site for injection is approximately 0.5 inches (1–2 cm) distal to the site used for the medial approach to the DIT joint.[75]

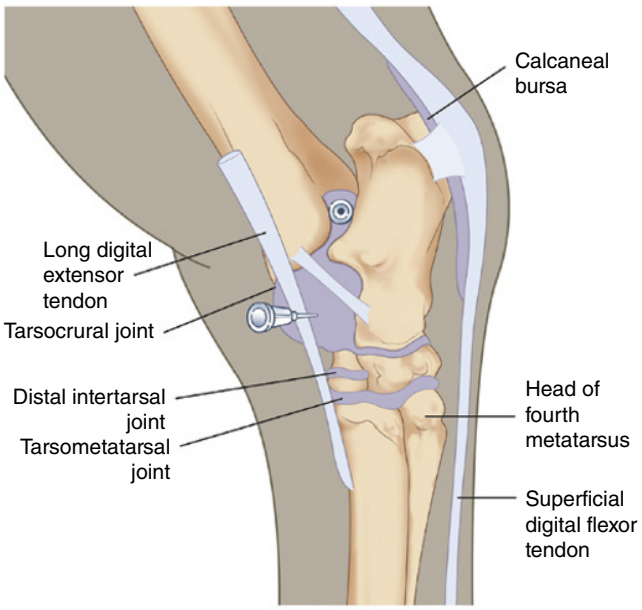


Figure 2.187. Injections sites for the dorsolateral (distal needle) and the lateral plantar (proximal needle) approaches to the tarsocrural joint.

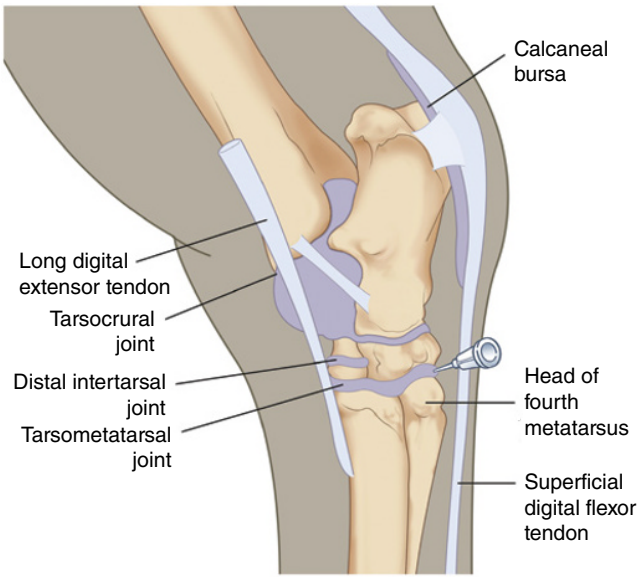


Figure 2.188. Injection site for the TMT joint proximal to the head of the lateral splint bone.

Distal Intertarsal (DIT) or Centrodistal Joint

The site for injection of the DIT joint is on the distomedial surface of the tarsus. The injection is performed from the opposite side of the horse with the limb weight-bearing. It is a difficult joint to inject with an accuracy rate of only 42% in a recent report.[69] One technique is to draw an imaginary line between the palpable distal tubercle of the talus and the space between the second and third metatarsal bones (MT II and III) at their proximal limits.[57,75] A small depression can often be felt with a fingernail just distal to the cunean tendon along this imaginary line. Another approach is to identify the medial eminence of the talus and medial eminence of the central tarsal bone. The site for injection is halfway between these landmarks and 0.5 inch (1 cm) distal to the eminence of the central tarsal bone.[48] A 1-inch (2.5-cm), 22- to 25-gauge needle is directed perpendicular to the long axis of the limb (or slightly caudally) to enter the joint space between the combined first and second tarsal bones, the third, and the central tarsal bones. The needle is advanced to about 0.5 inches (1 cm) and 3–4 mL of local anesthetic is injected (Figure 2.189; Video 2.37). The needle is determined to be within the DIT joint by low resistance to injection without developing a subcutaneous swelling and the ability to aspirate the injected contents of the syringe.[48] The DIT joint is thought to communicate frequently with the cunean bursa.[27]

The DIT or centrodistal joint can also be entered using a dorsolateral approach.[38] The injection site is 2–3 mm lateral to the long digital extensor tendon and

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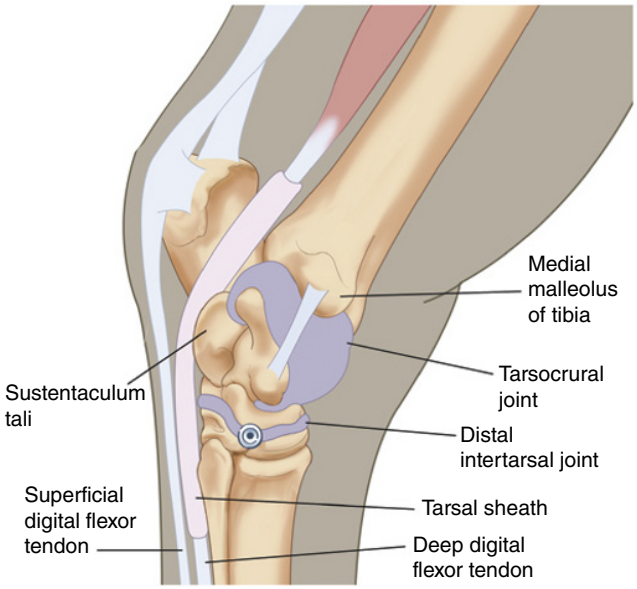


Figure 2.189. Injection site for the medial approach to the DIT joint.

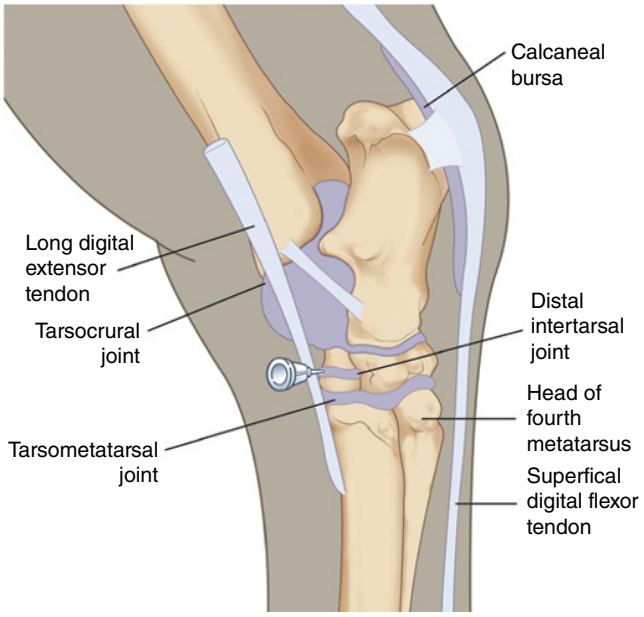


Figure 2.190. The dorsolateral approach to the DIT joint is 2–3 mm lateral to the long digital extensor tendon and approximately 6–8 mm proximal to a line drawn perpendicular to the axis of the third metatarsal bone through the head of the fourth metatarsal bone. This is usually distal to the palpable lateral trochlear ridge of the talus. The needle is directed plantaromedially at an angle of approximately 70° from the sagittal plane until bone is contacted.

6–8 mm proximal to a line drawn perpendicular to the axis of the third metatarsal bone through the head of the fourth metatarsal bone. This is usually distal to the palpable lateral trochlear ridge of the talus. The needle is directed plantaromedially at an angle of approximately 70° from the sagittal plane until bone is contacted (Figure 2.190; Video 2.38). This approach is safer for the clinician because it is performed on the lateral aspect of the tarsus but may be even more technically difficult than the medial approach.

Cunean Bursa

The cunean bursa is located on the medial surface of the distal tarsus between the medial collateral ligament of the tarsus and the medial branch of the tibialis cranialis (cunean) tendon. The bursa is relatively small and is not routinely anesthetized or treated alone because it often communicates with the DIT joint.[27] A 1-inch (2.54-cm), 22-gauge needle is inserted under the distal border of the cunean tendon and directed proximally to enter the bursa. Some clinicians treat the cunean bursa concurrently when medicating the DIT joint in horses with distal tarsal OA.

Calcaneal Bursa

The calcaneal bursa is located between the SDFT and the caudal aspect of the calcaneus. When distended, the bursa has synovial outpouchings medial and lateral to the tendon both proximal and distal to the SDFT retinaculum. These can often be seen as four distinct pockets of fluid surrounding the point of the hock-in horses with bursal distension. Synovial aspiration is best performed using the lateral synovial outpouchings either above or below the SDFT retinaculum with the horse weight-bearing. A 1-inch (2.5-cm), 20-gauge needle is angled proximally within these outpouchings to avoid the SDFT. The sites for needle placement are the same as those described for insertion of the arthroscope into the calcaneal bursa: 1 cm dorsal to the SDFT and 1 cm distal to the medial or lateral aspect of the SDFT retinaculum (Figure 2.191).[36] Approximately 8–12 mL of anesthetic is used to block the bursa.

Stifle Joint

The stifle joint is composed of three synovial compartments: the femoropatellar and the lateral femorotibial (LFT) and medial femorotibial (MFT) joints. Contrast studies have shown that the frequency of communication between the femoropatellar and the MFT joint is approximately 60%–65%.[54,80] The communication, however, is variable and appears to depend on the direction of flow of the injectable agent, the amount of joint inflammation, and anatomic variation. Communication between the femoropatellar and the MFT joint is observed more frequently when the MFT joint is injected than when the femoropatellar joint is injected. Communication between the femoropatellar joint and the LFT joint occurs rarely, and communication between the MFT and LFT joints does not occur under normal situations. Some clinicians feel that each synovial compartment of the stifle should be injected separately to ensure accurate distribution of local anesthetic.[75] However, greater diffusion of local anesthetic between compartments of the stifle probably occurs than what has previously been assumed based on anatomic, latex injection, and contrast arthrography studies.[27,48] The majority of the injection approaches to all compartments of the stifle are performed in the weight-bearing limb, and approximately 20–30 mL of anesthetic is usually recommended in each joint.

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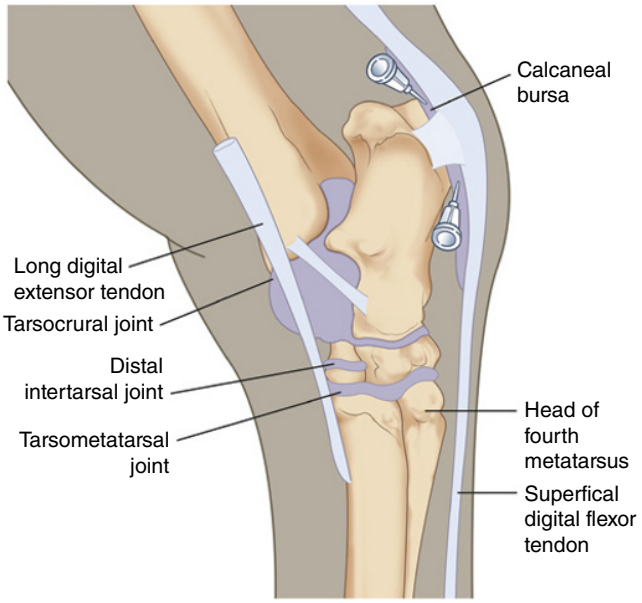


Figure 2.191. Injection sites for the calcaneal bursa are located either above or below the retinaculum of the superficial digital flexor tendon. These injection sites can be difficult to find without effusion.

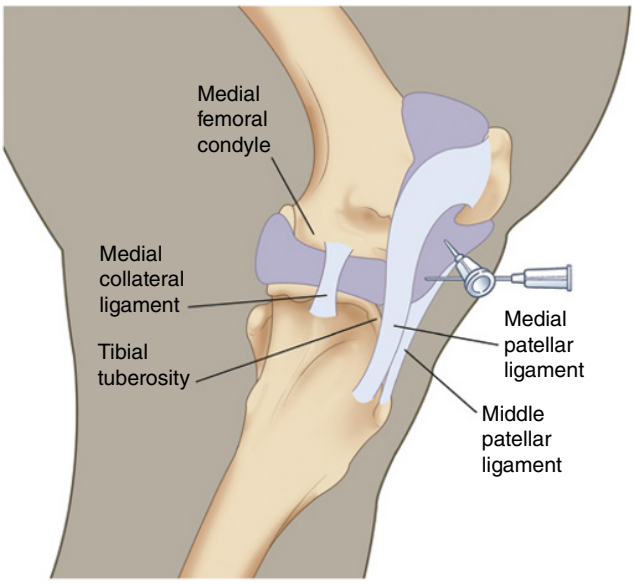


Figure 2.192. Lateral view of the injection sites for the femoropatellar joint. With one approach the needle is inserted perpendicular to the limb, and with the other approach it is angled from distally to proximally under the patella.

Femoropatellar Joint

The femoropatellar joint is the largest of the compartments, and it can be entered from the cranial (on either side of the middle patella ligament) or lateral (caudal to the lateral patella ligament) aspects. In one cranial approach, a 3.5-inch (3.8- or 8.9-cm), 18- to 20-gauge needle is inserted approximately 1–1.5 inches (3–4 cm) proximal to the tibial crest between the middle and medial patella ligaments and is directed proximally under the patella (Figure 2.192). This approach is best performed with the limb in a partial weight-bearing (slightly flexed) position.[2,75] Alternatively, the needle can be directed parallel to the ground with the limb fully weight-bearing.[48] The femoropatellar joint can also be entered just distal to the apex of the patella on either side of the middle patellar ligament with the limb weight-bearing.[79] The joint capsule is superficial at this location, and a 1.5-inch (3.8-cm), 18- to 20-gauge needle is directed at right angles to the skin.

The lateral approach to the femoropatellar joint is performed with the horse weight-bearing (Figure 2.193).[32] The lateral cul-de-sac of the joint is located caudal to the lateral patellar ligament and approximately 2 inches (5–6 cm) proximal to the lateral tibial condyle. A 1.5-inch (3.8-cm), 18- to 20-gauge needle is inserted into the recess perpendicular to the long axis of the femur until the nonarticular portion of the lateral trochlea is contacted. Synovial fluid can be retrieved in most cases, and this approach is usually well tolerated by the horse.

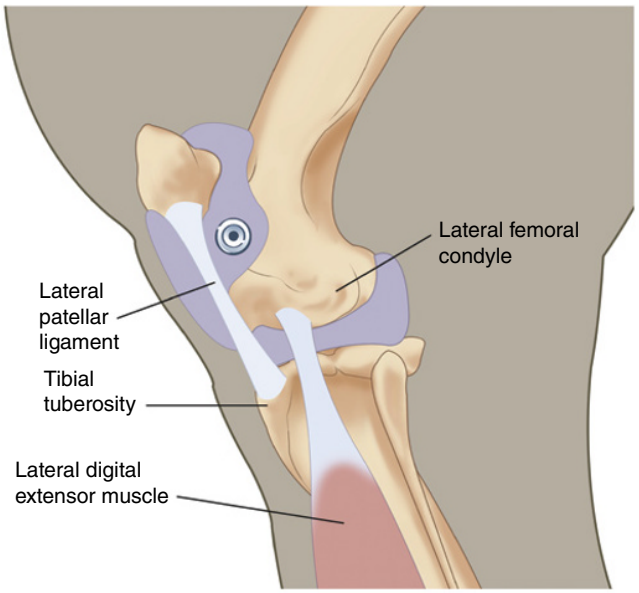


Figure 2.193. Lateral approach to the femoropatellar joint in the lateral cul-de-sac of the joint located caudal to the lateral patellar ligament and approximately 2 inches (5–6 cm) proximal to the lateral tibial condyle.

Medial Femorotibial (MFT) Joint

The site for injection of the MFT joint is located in the space between the medial patellar and medial collateral ligaments just above the palpable proximomedial edge of the tibia in the weight-bearing limb (Figure 2.194).[48] A 1.5-inch (3.8-cm), 18- to 20-gauge needle is inserted just caudal to the medial patellar ligament, 1 cm proximal to the tibia, and directed perpendicular to the long axis of the limb. The needle may need to be repositioned slightly cranially or caudally to help obtain synovial fluid. The medial meniscus will be contacted (penetrated) if the needle is inserted too far caudally or too close to the proximal tibia.[34,79] This approach may be performed from the same side (facing the stifle) or from the opposite side, reaching under the horse's belly.

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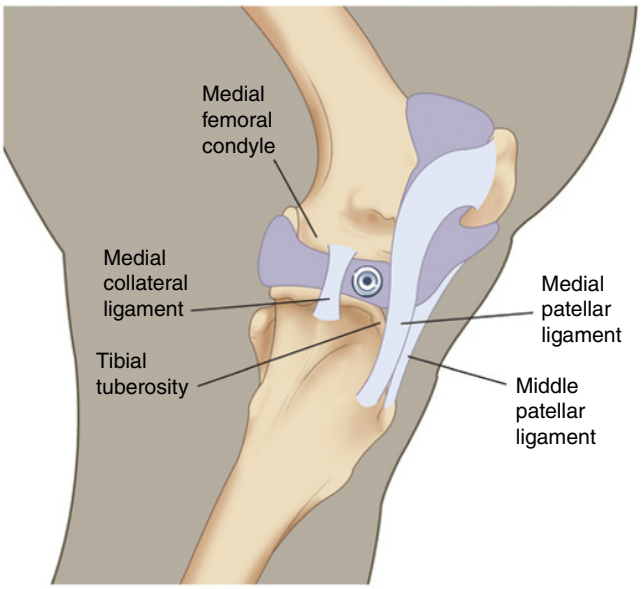


Figure 2.194. Medial approach to the medial femorotibial joint just caudal to the medial patellar ligament and proximal to the tibial crest.

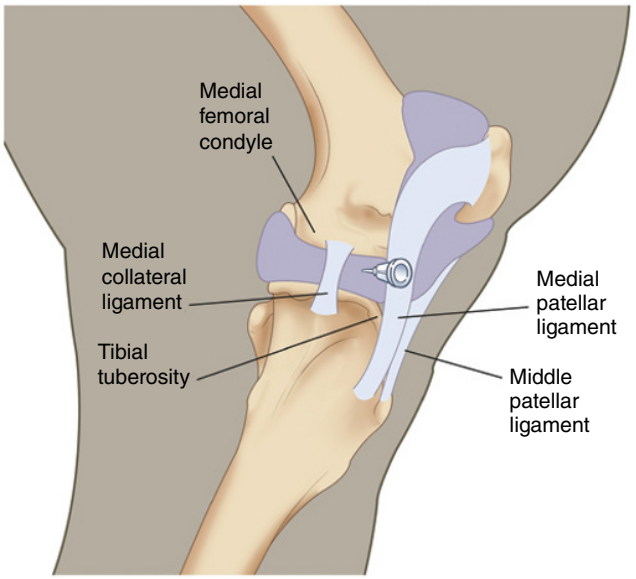


Figure 2.195. With the cranial approach to the MFT joint, the needle is directed in a cranial-to-caudal direction in the depression between the medial patella ligament and the tendon of insertion of the sartorius muscle to enter the proximal outpouching of the joint.

Another approach to the MFT joint is located 0.5–1 inch (1–2 cm) proximal to the medial tibial plateau in the depression between the medial patella ligament and the tendon of insertion of the sartorius muscle.[77] A 1.5-inch (3.8-cm), 20-gauge needle is directed in a cranial-to-caudal direction parallel to the ground and parallel to a plane that bisects the limb (Figure 2.195). The needle enters a medial outpouching of the MFT joint and avoids inadvertent penetration of the medial meniscus and the medial femoral condyle. In addition, synovial fluid is more easily obtained with this approach compared with the medial approach to the MFT joint.

Lateral Femorotibial (LFT) Joint

The site for injection of the LFT joint is slightly caudal to the palpable edge of the lateral patellar ligament just above the proximolateral edge of the tibia with the limb weight-bearing (Figure 2.196; cranial needle). A 1.5-inch (3.8-cm), 18- to 20-gauge needle is inserted at right angles to the long axis of the femur and directed from lateral to medial to a depth of 1 inch (2–3 cm). An alternative approach is to insert the needle just proximal to the tibia in the space between the lateral collateral ligament of the LFT joint and the tendon of origin of the long digital extensor tendon (Figure 2.196; caudal needle).[34,48] The palpable head of the fibula helps to identify these structures. The needle is inserted slowly to a depth of approximately 1 inch (2–3 cm) until the joint capsule is entered.[48]

Single Approach for All Three Joints

All three joints in the stifle can be injected from one cranial site 1.5 cm proximal to the tibia between the lateral and middle patellar ligaments with the stifle slightly flexed.[75] Local skin and subcutaneous anesthesia at the injection site is helpful for this technique. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is inserted and directed caudomedially parallel to the tibial crest toward the MFT joint. The needle is then withdrawn to the subcutaneous tissue and redirected caudolaterally parallel to the tibial crest toward the LFT joint and anesthetic is deposited. The needle is again withdrawn to the subcutaneous tissue and redirected proximally under the patella into the femoropatellar joint.

Trochanteric Bursa

The trochanteric bursa is located beneath the tendon of insertion of the middle gluteal muscle on the cranial

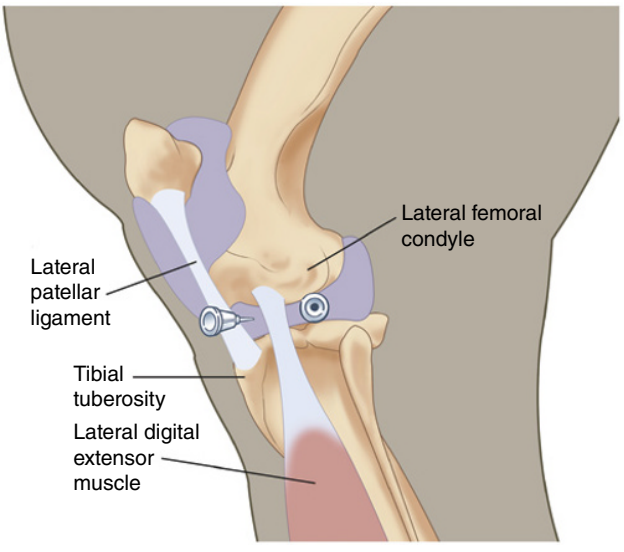


Figure 2.196. Injection sites for the lateral femorotibial joint. One is just proximal to the tibia in the space between the lateral patellar ligament and the tendon of origin of the long digital extensor tendon, and the alternative is caudal to the long digital extensor tendon and cranial to the lateral collateral ligament.

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aspect of the greater trochanter of the femur (Figure 1.48 in Chapter 1). The site for injection is between the tendon and the lateral surface of the greater trochanter at the most cranial aspect of the palpable greater trochanter. A 1.5-inch (3.8-cm), 18-gauge needle is usually all that is needed, although larger Warmblood horses may require a longer needle in some cases.[28] The needle is inserted and directed horizontally at right angles to the sagittal plane until bone is encountered. Seven to 10 mL of anesthetic is injected. An alternate method is to direct the needle medially through the middle gluteal muscle directly over the bursa toward the trochanter.[75] A recent study concluded that positioning the limb caudally with the foot non-weight-bearing and on a Hickman block facilitated centesis of the trochanteric bursa.[78] The author has also recommended using ultrasound guidance to improve the success of the injection.[78]



Figure 2.197. Craniolateral view of the injection site for the coxofemoral joint. The needle is inserted in the trochanteric notch and directed at a 45° angle with the long axis until the joint is entered. Ultrasound guidance is recommended to enter the coxofemoral joint regardless of the approach used.

Coxofemoral (Hip) Joint

The coxofemoral joint is one of the most difficult joints to enter.[34,48] This is particularly true in mature, heavily muscled horses. The joint is well away from the proximal end of the femur, and the landmarks for injection are often difficult to palpate. For these reasons, ultrasound guidance of the needle is recommended.[15,82] The procedure is best performed with the horse standing squarely and restrained within stocks. Mild sedation is often advised because movement during the injection procedure may cause the needle to bend or break.

Both craniodorsal and cranioventral approaches have been described. The cranioventral approach requires ultrasound guidance,[82] while the craniodorsal approach may be performed blindly or more optimally with ultrasound guidance.[15] The most important landmarks for the craniodorsal approach are the paired summits of the greater trochanter of the femur. The trochanter is located about two-thirds of the distance between the tuber coxae and the tuber ischii.[48] The greater trochanter is approximately 4 inches (10 cm) wide with a notch between the cranial and caudal protuberances that can be difficult to palpate. The site for injection is about 0.5 inches (1–2 cm) above the middle of the proximal summit of the trochanter (Figure 2.197).[48] A small bleb of local anesthetic is injected subcutaneously over the injection site, and a small stab incision may aid needle insertion.[2,3] A 6- to 8-inch (15- to 20-cm), 16- to 18-gauge spinal needle is directed in a horizontal plane perpendicular to the vertebral column (Figure 2.197). The needle should be directed slightly downward to stay close to the femoral neck so that it is approximately 0.5 inches (1–2 cm) lower than the insertion site after it has been advanced 3–4 inches (8–10 cm). Firm fibrous tissue is often felt just before the needle penetrates the joint capsule at approximately 4–6 inches (10–15 cm). Synovial fluid is often aspirated and 30–60 mL of anesthetic is recommended.[34,48]

Sacroiliac (SI) Joint

The SI joint is not a true synovial joint, but treatment of this region is common in horses with sacroiliac problems. There are several different approaches that can be used to access the sacroiliac region with most resulting in periarticular deposition of medication (not intra-articular). A cadaver study of two different ultrasound-guided injection techniques (cranial parasagittal and caudomedial) concluded that accuracy was poor for intra-articular injection.[74] The described technique by Engeli and Haussler is used by the author and is described here.[22] The horse is usually sedated, restrained in stocks, and the injection site is anesthetized with local anesthesia. The landmark for injection is the cranial aspect of the tuber sacrale. A 10-inch (25-cm), 15- to 16-gauge spinal needle is bent to an angle of about 40° in the direction of the needle's bevel. The needle is inserted through a stab incision in the skin 1 inch (2 cm) cranial to the contralateral tuber sacrale and directed at a 60° angle to the vertical plane. The needle is advanced across midline aiming for a point midway between the ipsilateral tuber coxae and the greater trochanter of the femur until it contacts the medial aspect of the tuber sacrale (Figure 2.198). The needle hub is lifted, and the needle is advanced at a steeper angle along the medial aspect of the ilial wing until it contacts the dorsal surface of the sacrum at a depth of approximately 6–8 inches (15–20 cm).[48] An alternative technique is to insert a 6-inch (15-cm), 18-gauge needle near the cranial aspect of the tuber sacrale. The needle is directed ventrocaudolateral toward the SI joint of the opposite side at a 20°–40° angle to the vertical plane.[19] Ultrasound guidance of SI injections is commonly performed, and both cranial and caudal approaches have been described.[16,74] The reader is referred to Chapter 6 for additional information on SI injection techniques.

Cervical Facets

Centesis of a cervical facet joint is usually performed to administer medication into the joint of a horse that has clinical signs suggestive of disease within the cervical articulation. Lesions of the cervical facets tend to occur most commonly in vertebrae C5 to C6 and C6 to C7.[44] The procedure is performed in the standing sedated horse with the help of ultrasound and is usually performed bilaterally unless a specific side of the lesion has been documented. The approximate location of the

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Figure 2.198. Needle location and angulation of the spinal needle used to inject the contralateral sacroiliac joint from the cranial aspect of the tuber sacrale. A 10-inch (25-cm), 15- to 16-gauge spinal needle is bent to an angle of about 40° in the direction of the needle's bevel. The needle is inserted through a stab incision in the skin 1 inch (2 cm) cranial to the contralateral tuber sacrale and directed at a 60° angle to the vertical plane.

injection can be made by using one width of the hand to represent the length of one cervical vertebra.[48] The facet joint is imaged with the ultrasound probe, and a 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is advanced toward the joint at an angle so the tip of the needle can be seen. The cervical articular processes and joint form an easily identifiable sonographic landmark, a steplike echogenic surface described as having the appearance of a "chair."[44] Ultrasound is used to direct the needle into the joint and the solution is deposited.

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CHAPTER

Diagnostic Imaging

RADIOGRAPHY

Myra Barrett and Elizabeth Acutt

Radiography is the most frequently used imaging modality in the equine patient, and even with the growth of advanced imaging technologies, radiography remains a first-line screening tool for equine musculoskeletal disease.

Advances in technology have made digital radiography mainstream and accessible to most practitioners. While the many developments in technology have resulted in more accessible and easily shared, high-quality images, the fundamental principles of radiology remain the same as the days of screen-film radiography. The veterinarian must be familiar with the basic instrumentation of an X-ray machine and the radiation safety techniques commonly practiced in radiography. An understanding of the normal equine radiographic anatomy, basic radiographic interpretation principles, examination techniques, and post-capture image processing is essential to perform diagnostic radiographic studies. Too often, knowledge of these basic concepts is overlooked, resulting in inferior radiographic examinations. Poor-quality radiographs may result in erroneous diagnoses and conclusions.

This section is not intended to comprehensively cover the physics behind the different radiographic systems; however, a review of the various technologies available in veterinary medicine including types of digital imaging systems and image display and storage is included. More detailed information is available elsewhere.[22,29,31,49,50,56,63] The normal equine radiographic anatomy with standard projections used in the diagnosis of lameness in addition to the common radiographic signs of bone and soft tissue response to different pathologic processes are also discussed.

EQUIPMENT

Knowledge of radiography equipment including X-ray machines, detector systems, film processing equipment, image viewing devices, and accessory equipment is necessary for obtaining good-quality diagnostic radiographs safely. The reader should also be aware of the advantages and disadvantages of the different equipment options that are available for veterinary practice.

X-ray Machines

The basic control settings of an X-ray machine, the milliamperage (mA), exposure time, and kilovoltage potential (kVp) are located on the control panel and may be changed to vary the exposure (Figure 3.1). Milliamperage is the tube current and refers to the quantity of electrons flowing per second in the X-ray tube. Ultimately, it determines the quantity of X-rays emitted from the X-ray tube.

Exposure time is an important variable in equine radiology. Because of problems related to patient and detector movement, exposure time should be 0.1 second or less, if possible, for equine limb examinations. The use of electronic timers is recommended for accurate timing when the exposure is less than 0.1 second. For equine radiology, an electronic timer with two-step exposure button is desirable.[48] The first step warms the X-ray tube filament; the exposure is made in the second step. The two-step exposure button prolongs the X-ray tube life. Because exposure time (in seconds) multiplied by the milliamperage equals milliamperage-seconds (mAs), radiation exposure is directly related to milliamperage-seconds.

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Companion website: www.wiley.com/go/baxter/lameness

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Kilovoltage determines the energy of X-rays being produced. Kilovoltage potential should range from 70 to 90 for equine limb radiographs on mature horses when a portable X-ray machine is used. A 10% increase or decrease in kilovoltage potential effectively doubles or halves the radiation exposure.

The focal spot-film distance (FFD) is the distance from the X-ray tube focal spot to the detector. The focal spot location is usually marked on the outside tube housing; if it is not, the approximate center of the tube housing can be used for its location. A constant FFD is necessary to minimize improper exposures because the intensity of X-rays that expose the film is inversely proportional to the square of the FFD. Therefore, a small difference in the FFD can dramatically change the exposure on the X-ray film.

Some method of measuring the FFD before each exposure should be employed. Measuring devices with converging light beams at the correctly set FFD are easy to use and less cumbersome than a measuring tape or string. The FFD for equine limb radiography should be between 36 and 40 inches (85 and 100 cm) and no less than 24 inches (60 cm). When the FFD is less than 24 inches, the object is magnified, and spatial resolution is reduced. FFDs greater than 40 inches can be used if exposure times are not excessively long. When making radiographs of larger body parts such as necks, it is important to remember that the distance needs to be set from the X-ray tube to the detector, not to the skin surface, a concept that is more easily forgotten when the plate is not well seen by the person holding the X-ray generator.

Collimation is the process of limiting or restricting the primary X-ray beam to the appropriate size to cover the anatomical region of interest within the X-ray detector being used. Collimating the primary beam is a safety practice that must be monitored for each exposure and must be reset if the cassette size changes. Limiting the size of the primary X-ray beam is a major factor in reducing scatter radiation, which keeps radiation exposure to personnel as low as possible. Collimation also improves image quality, particularly in large body parts such as the caudal cervical spine. Fixed cylinders or cones and adjustable light-beam collimators are available (Figure 3.2). The disadvantage of fixed primary beam restrictors is that they do not conform to different cassette sizes. Adjustable light-beam collimation is recommended for equine radiography and is standard to many X-ray machines. They come with an internal light source, preferably 40 W or greater,[48] so the limits of the primary X-ray beam are projected on the X-ray cassette/detector as visible light.

The type of X-ray machines best suited for equine practice depends on the type of practice: out of hospital versus in hospital. The features of the X-ray machines must be matched to fit different practice situations. Some compromises or trade-offs must be made, e.g. less milliamperage or kilovoltage potential for more portability. An ideal X-ray machine for equine radiography has the following features:

1. Easily and quietly movable, with a tube head that can extend to the floor or ground surface
2. Adjustable milliamperage and kilovoltage potential setting
3. Electronic, two-step timer capable of accurate exposure times of 0.1 second or faster
4. Line voltage compensator and compensation meter



Figure 3.1. Control panel for a 30-mA portable X-ray machine.



Figure 3.2. Adjustable light-beam collimator. The primary beam is limited by adjustable leaves in the collimator. A light source within the collimator defines the limits of the primary beam (white arrows).

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5. Primary X-ray beam restrictor (the preferred system is an adjustable light-beam collimator)
6. Absence of radiation or electrical hazards

Portable, mobile, and fixed (ceiling-suspended) X-ray machines are available for equine radiography. These differ in size and capacity, from small, portable 15-mA machines to large, fixed 800-mA machines. Portable X-ray machines are best suited for out-of-hospital locations. They are lightweight (30–55 lbs) and can be easily stored for transport. Options for portable X-ray machines include both corded and battery-operated wireless and range in size and weight. Most manufacturers also provide a variety of types of stands on which the portable X-ray machine can be suspended. This helps reduce dose to personnel and minimize motion artifact and fatigue.

High-frequency transformers that reduce the exposure time are available for portable X-ray machines. They are equivalent to a 3-phase, 12-pulse X-ray generator and provide a 40%–50% increase in exposure compared with a full-wave rectified machine. The practical application is to reduce the exposure time and still obtain an adequate exposure for a diagnostic radiograph. A line voltage compensator is also desirable for portable X-ray machines. It is especially important in field situations, where the line voltage may fluctuate with the simultaneous use of the electrical equipment, and it can compensate for a line voltage drop when a long electrical extension cord is used.

Fixed X-ray machines are usually suspended from the ceiling. They are limited to use within a single room. These machines can be coupled with large transformer systems that are capable of providing high milliamperage (800–1,000 mA). Ceiling-suspended tubes are easily movable and can be locked into position by magnetic locks while the X-ray exposure is made. The tube should be capable of extending to the floor. X-ray machines with high milliamperage capability have fast exposure times, eliminating most motion artifacts when radiographing equine limbs.

Types of Digital Radiography

Digital radiography can be divided into computed radiography (CR) or direct digital radiography (DDR).

CR uses a detector panel that stores the image and then uses a separate image readout unit. In DDR, the detector panel converts the X-rays into electrical charges by means of a direct readout process. DDR systems can be further divided into direct or indirect conversion technologies depending on the type of X-ray conversion used. Both CR and DDR systems convert the electronic signal to digital data and send it to an acquisition device. At this point, the digital data is considered "raw," and it can be further processed.

The term picture archiving and communication system (PACS) is used to describe a system that allows for image retrieval, review, processing, interpretation, and storage. The term "image processing" incorporates multiple steps in the acquisition of a digital image before the study is completed. During the image processing, the raw image can be manipulated with the purpose of having the best image possibly available for interpretation at the viewing station. Key factors that can be manipulated include the lookup table (LUT), which allows the operator to change the grayscale of the image by altering the actual pixel values, region of interest, and contrast, as well as other options. Once the study is completed, the images are sent to the viewing station where Digital Imaging and Communication in Medicine (DICOM) viewing software is used for image display on a computer monitor. A complete discussion of image processing is beyond the scope of this chapter. More in-depth information is available in textbooks of diagnostic radiology physics and radiology journal articles.[9,29,31,41]

DIGITAL RADIOGRAPHY SYSTEMS

Computed Radiography

CR systems are similar to screen-film systems, in which a cassette stores the latent image until processing. In CR, the cassette contains an imaging plate possessing photostimulable phosphors (PSP). After the imaging plate is exposed to X-ray photons, the PSP change to a higher energy state and store the latent image until processing.

When the cassette is placed in the reader unit, the imaging plate is automatically removed from the cassette and scanned by a laser beam. The laser light stimulates the energy trapped in the PSP, and visible light is released from the plate. The released light strikes a photomultiplier tube and is converted into an electronic signal. The magnitude of the electronic signal represents the degree of X-ray attenuation of the structure that was imaged and is also assigned a corresponding shade of gray after conversion from analog-to-digital format.[62] The data is then stored temporarily in a local hard disk. Subsequently, the imaging plate is exposed to bright white light to erase any residual trapped energy before it is returned to the cassette for reuse.[9] The whole readout process for a 14 × 17-in. image plate takes approximately 30–40 seconds.[29] The resultant image is sent to an acquisition computer, where post-processing manipulations can be performed.

The separation of the readout unit in CR systems means that in a multi-veterinarian practice, cassettes can be divided between practitioners, both in the field and in the clinic to allow for simultaneous performance of multiple radiographic examinations. It also enables a degree of wireless imaging not always afforded by digital systems since the cassettes themselves do not require wires. The initial costs of a CR system are less than those of a DDR system, which may also make the system a more attractive prospect for purchase.[2] CR units can also be appealing in mixed animal practice because of the flexibility in the use of plates. However, the resolution achieved by these systems is often inferior to DR systems, which have several other advantages, outlined below.

Direct Digital Radiography

DDR systems have a direct readout, meaning that the detector sends the signal straight to the computer instead of using a reading unit such as the CR system. The time

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lapse between exposure to image display takes less than 10 seconds, which increases the patient throughput.[29] The ability to rapidly take multiple sequential views can facilitate greatly the workflow in a busy practice and is more likely to result in the acquisition of high-quality, diagnostic images, since immediate assessment of patient positioning and generator settings is possible. Two different DDR conversion systems exist: direct and indirect conversion.

Direct conversion systems feature a flat panel detector containing photoconductors such as selenium (most commonly used), lead iodide, lead oxide, thallium bromide, or gadolinium compounds.[29] The photoconductors are the first layer in contact with the X-ray photons that exit the patient and are responsible for converting them into electrical charges. The electrical charges are stored in capacitors and then read row by row, and the information is sent to the computer after analog-to-digital conversion. More detailed information is available in radiology journal articles.[29,62]

Indirect conversion can be done with two technologies: charged coupled device (CCD) or indirect flat panel detector. In a CCD, the X-ray photons from the patient are converted to light after striking a scintillator layer. The light is then usually minified with a lens to fit in the small CCD where it is recorded and converted into electrical charges. In the process of light minification, some of the light photons are lost and do not reach the CCD unit. This results in increased image noise compared with flat panel detectors in which minification is not applied.[62] The noise, edge distortion, and artifacts that are frequently seen in CCD systems mean that this technology has largely fallen out of favor. As a result, servicing and repairing such systems has become complex and costly.

An indirect flat panel detector also contains a scintillator layer, or intensifying screen, made of cesium iodide crystals. Other materials used in the scintillator layer include gadolinium-based crystals. The advantage of cesium iodide scintillators is that the crystals can be structured into 5–10-μm-wide parallel needles, which reduce the spreading of light within the scintillator. Hence, the spatial resolution is increased. The high resolution of these systems means that they are the gold standard of medical imaging.

While initial DDR systems required wired connections between the plate and the reader, many fully wireless systems are now on the market. Comparison charts are available for the commercially available DDR systems and are a good reference source when selecting a system for purchase. Other considerations besides cost when selecting a system include the logistics of maintenance, service and repair, warranty duration, and the availability of technical help in the case of software/hardware malfunction.[2]

Digital Image Display

A computer used in a digital image viewing station consists of two basic parts: one or more monitors (hardware) and a DICOM viewer (software). A monitor used for digital radiography should have excellent resolution, high brightness (luminance), and wide dynamic range. The resolution of a monitor is dictated by the pixel and matrix size. As the pixel size decreases and the matrix size increases, the monitor's resolution improves. The dynamic range of a monitor is the luminance ratio between the darkest (black) shade and the brightest (white) shade that the monitor can display. The dynamic range of a monitor is usually correlated to the luminance; thus, the higher the monitor brightness, the higher the dynamic range.

A wide variety of Windows- or Apple-based DICOM software programs are available and include free Internet-downloadable versions to more sophisticated software that requires purchase or leasing contracts. Free versions typically are less robust and may limit the size of study that can be downloaded. The different DICOM viewing software provides the user with the ability to further manipulate and improve the image displayed on the monitor. Features such as modifying contrast (window and level), image sharpening, edge enhancement, zoom, measurement tools, multiplanar reconstruction, etc. are included in most DICOM viewers but differ among vendors depending on the quality of the software (Figure 3.3).

Digital Image Storage and Transmission

The technology for digital imaging storage and transmission is rapidly evolving, making storage and sharing relatively simple. Images can be stored on an on-site server, and many vendors offer cloud-based storage as well. While convenient, reliance solely on cloud-based storage requires a fast and reliable Internet connection. The growth of the practice of teleradiology—the electronic transmission of images for interpretation and consultation—has been enhanced via downloadable DICOM images or web-based viewing of images. These options for image transmission are preferable to emailing jpeg images, which have lower resolution and have to be converted back to DICOM images in order to be manipulated.

Accessory X-ray Equipment

Accessory X-ray equipment for equine musculoskeletal examinations has special requirements. Good-quality, well-maintained accessory equipment is necessary to ensure that quality radiographic examinations are performed safely. Accessory equipment consists of grids, markers, and film-marking systems, detector holders, padded tables for radiographic projections under general anesthesia, and positioning aids. These pieces of equipment are used with conventional or digital radiography. Most accessory equipment is available commercially, but because of the unique requirement of equine radiology, some equipment, such as cassette holders, padded tables, and positioning aids, may have to be custom built locally.

Grids

Radiographic grids are used to decrease the amount of scattered radiation that exposes the X-ray film. Because scattered radiation exposes film from several directions, it has the effect of decreasing image detail and contrast. Radiographs of thicker body parts, such as

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Figure 3.3. Foot radiographs displayed on Horos, an example of free-downloadable image viewing software developed for Apple computer products. *Source*: Courtesy of Dr. Kathryn Wulster.

the caudocranial view of the stifle or cervical spine, are more affected by scatter radiation and can most benefit from the use of a grid. Phosphor is very sensitive to scatter radiation, and grids remain of importance even when using CR and DDR systems.

The advantages of using a grid are increased film detail and contrast, which improve the diagnostic quality of the film. The main disadvantage of a grid is increased radiation exposure to personnel. Increased radiation exposure is particularly important in equine radiography, for which low-output X-ray machines are extensively used.

Current recommendations are to consult with the system vendor regarding the requirement for and specifications of a grid.[22]

Film Markers and Marking Systems

Adequate, legible film marking is necessary but often overlooked in equine radiography. Proper identification procedures are necessary for follow-up comparative examinations and for accurately documenting when, where, what, and by whom the examination was performed. Such documentation on the film in a proper permanent fashion may also be needed for medical-legal purposes. Although most digital systems provide electronic markers, they don't always transfer with the metadata, and if the plate is held incorrectly, the laterality marker may not be on the correct side. Therefore, it is optimal to use a lead marker that indicates the limb being imaged and laterality in conjunction with the digital marker. The lead marker should be placed on the lateral aspect of the limb.

Positioning Aids

Positioning blocks are necessary for radiation safety purposes and to facilitate consistent views or projections of an examined part. Detector holders and blocks enable personnel who hold the cassette to position their hands away from the primary X-ray beam, even though they are wearing lead-impregnated gloves. For equine limb examinations, a detector holder (tunnel) for weight-bearing foot studies and a positioning block are necessary.

A plain wooden or Perspex block can be used to elevate the foot and enable acquisition of the entire foot and enable proper alignment of the X-ray beam for lateromedial and dorsopalmar/plantar views. The foot should be positioned on the block in such a way as to enable its close proximity to the cassette or plate and minimize magnification. Blocks should be used bilaterally to allow even weight bearing. A detector holder (tunnel) is necessary for the proximopalmaroproximodistal oblique (skyline) view of the navicular bone and can also be used for the dorsal 60° palmar oblique view. The detector holder protects the detector from damage caused by direct weight bearing by the horse. An alternative technique for acquiring the dorsal 60° palmar oblique view is to place the foot resting on its toe on a Redden navicular block using a horizontal beam alignment (Figure 3.4). These positioning aids allow routine examinations of the limb to be performed in a safe, consistent manner.

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Figure 3.4. Acquisition of 60° dorsoproximal to palmarodistal oblique using a Redden navicular block.

RADIATION SAFETY

The general principles of radiation safety are as follows:

1. Keeping personnel as far away from the radiation source (X-ray tube) as possible
2. Using protective barriers
3. Reducing X-ray exposure factors
4. Using a radiation-monitoring system

Keeping personnel as far away from the X-ray source as possible can be accomplished by having nonessential personnel leave the immediate area, using cassette holders and positioning blocks, providing tranquilization or general anesthesia when needed,[1] using an X-ray tube stand or support, and having a 1- to 2-meter-long cord connected to the exposure button. No part of the body should be exposed to the primary X-ray beam.

The personnel subject to the greatest exposure when performing equine examinations are those holding the horse's limb, the detector holder, and those holding the halter.[1] Therefore, if personnel must be near the animal when the X-ray examination is performed, they should wear lead personal protective equipment (PPE).

A radiation-monitoring system should be used by all radiology personnel. These systems not only provide safety guidance but also protect against possible legal implications. Film-badge monitoring systems and service can be purchased from commercial sources.

Equipment necessary for safely operating an X-ray machine includes detector holders, lead PPE, aluminum filters, and an adjustable light-beam collimator. Detector holders eliminate the need to handhold detectors, increasing the distance between hands and the X-ray beam.

Lead aprons, thyroid shields, and gloves should be worn by everyone assisting with the X-ray examination. Protective gear should have at least 0.5-mm lead equivalent. Lead PPE provides adequate protection from secondary and scattered radiation but not from primary radiation. The life of lead PPE can be prolonged by hanging them when they are not in use. This prevents cracks or holes forming in the lead-impregnated lining as a result of improper care. All lead PPE should be radiographed periodically to check for damage (Figure 3.5).

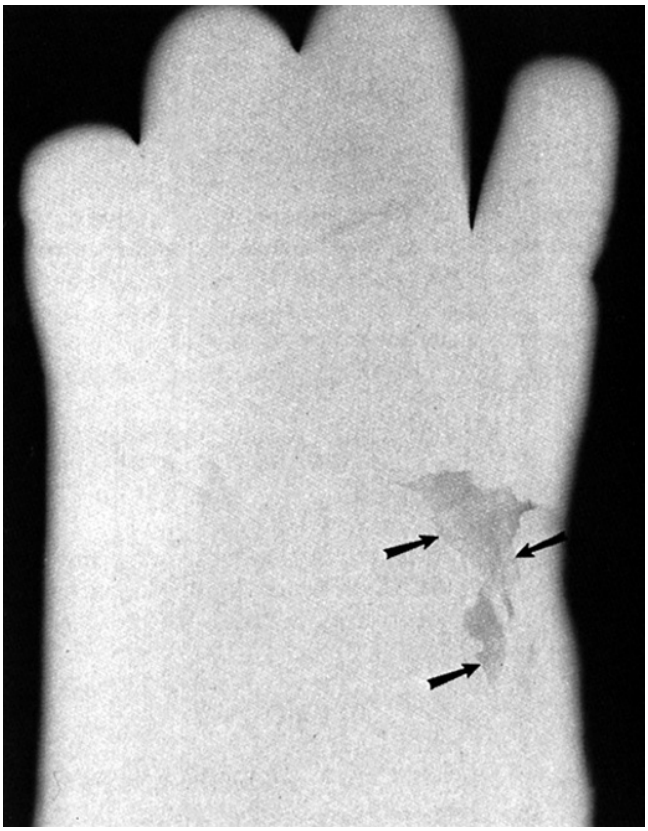


Figure 3.5. Radiograph of a lead glove, showing a lucent area (arrows) that represents a defect or hole in the lead lining.

An X-ray beam-limiting device or collimator (Figure 3.2) is an important, yet overlooked, piece of radiation safety equipment. The size of the primary X-ray beam is a major factor in determining radiation dose to the hands. Adjustable light-beam collimators have the advantage of limiting the primary X-ray beam to the exact cassette size, regardless of the FFD. The light also assists alignment of the primary X-ray beam with the cassette. Every X-ray machine should have an adjustable beam-limiting device.

In summary, the following safety measures should be observed:

- Never handhold the X-ray detector during an exposure. Detector holders or handles should be used.
- All personnel not needed for assistance with the X-ray examination should leave the immediate area.
- Lead gloves, thyroid shields, and aprons should be worn by all individuals assisting with the X-ray examination.
- A primary X-ray beam-restricting device should be used, preferably an adjustable light-beam collimator.
- If possible, rotate personnel responsible for holding the detector; avoid routinely using the same person for this job.
- Do not allow anyone under 18 years of age to assist with an X-ray examination.

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- Pregnant personnel should have limited role in X-ray examination and use additional monitoring (waist badge under lead gown).
- Chemical restraint and positioning devices should be used to minimize motion and personnel exposure and limit retake examinations
- Use a radiation-monitoring system

The veterinarian in charge is responsible for the radiation safety practices used by his/her employees. Providing necessary radiation safety equipment and following these rules should keep exposure levels below the limits recommended by the National Council on Radiation Protection and Measurements.[40]

CONTRAST EXAMINATIONS

A contrast radiographic examination consists of using a radiocontrast agent to better define suspicious lesions detected clinically or radiographically but not distinctly seen on survey radiographs. Nonionic, iodinated contrast material is most useful for contrast examinations in lame horses. While previously cost prohibitive, nonionic formulas are now more affordable and readily available and are safer than ionic contrast material. Positive-contrast agents are commercially available in an injectable form and include iohexol (Omnipaque [Nycomed Inc, 90 Park Avenue, New York, NY]) and iopamidol (Isovue [Bracco Diagnostics, Princeton, NJ]). The use of negative-contrast agents (gas) has been reported but has not found widespread, routine acceptance.[3] Procedures most commonly performed are injection of a draining tract (sinography or fistulography) and myelography. Other contrast examinations such as arthrography and tendonography have become less popular and in the majority of cases have been replaced by ultrasound, computed tomography, magnetic resonance imaging, or arthroscopy.

Fistulography or Sinography

Fistulograms provide valuable diagnostic information when chronic draining tracts or recent traumatic puncture wounds are present. Survey radiographs of the area should be made first. If the source or cause of the draining tract or puncture wound is not clearly identified on the survey radiographs, a fistulogram can be performed to obtain additional diagnostic data.[13,15,33,36]

The technique consists of injecting undiluted water-soluble triiodinated contrast material into the draining tract as aseptically as possible. Water-soluble contrast material is used because it is less viscous and penetrates chronic draining tracts more easily than oil-based contrast material. To avoid contrast material draining from the tract after injection, an inflatable, cuffed (Foley) catheter or a small polyethylene tube inserted some distance into the tract can be used before injecting the material. Filling is best accomplished if the contrast material is injected under pressure; thus, some form of occlusion of the tract opening is necessary.

When the distal extremity is being examined, it is important to flex and extend the region slowly. This allows a tract that may be closed while the horse is standing to open up, permitting the contrast to enter the tract. The volume needed for injection is not consistent, and when fistulograms are performed, the injection of an insufficient volume is a common error.[32] Contrast material should be injected until back pressure is felt on the syringe plunger or external leakage is observed. A single radiograph can also be made to determine if an adequate volume of contrast material was injected. After contrast material has been injected to delineate the entire tract, orthogonal radiographic views should be made for complete evaluation. Any contrast material that leaks onto the skin surface should be removed before radiographs are made.

A fistulogram may demonstrate (1) the extent and direction of the tract to aid in surgical exploration, (2) communication with underlying soft tissue structures (e.g. tendon sheaths or synovial joints) (Figure 3.6a), (3) osseous involvement (e.g. sequestra or osteomyelitis associated with surgical implants), and (4) filling defects (which appear radiolucent because of displacement of contrast material) (Figure 3.6b).[15] Filling defects may be caused by fibrous reaction within the tract or by foreign bodies. Fibrous tissues generally have irregular borders, whereas foreign bodies such as wood splinters have sharp, straight borders. Small foreign bodies may not be identifiable on a fistulogram because of the overlying opacity of the contrast material. In such cases, ultrasound imaging may provide additional diagnostic information.[13]

Myelography

Myelography in the horse is used to substantiate cervical spinal cord compression suspected from a neurologic and/or radiographic examination. It also serves to identify the location, extent, and type of compressive lesion present, which is necessary for determining the prognosis and indication for surgical intervention.

Prior to myelography, survey radiographic examination of the entire cervical spine is necessary. Survey radiographs should include neutral lateral images as well as right 45–55° dorsal-left ventral and left 45–55° dorsal-right ventral oblique images. The radiographs should be assessed for malalignment, cervical vertebral malformation and stenosis, articular facet pathology, fractures and osteochondrosis, disk space or endplate changes, and any other potentially relevant lesions (Figure 3.7). The accuracy of the subjective evaluation of survey radiographs for predicting a compressive lesion has been reported to be 70% at C3–C4 and only 40% in all other levels of the cervical spine.[37]

There are different methods for quantitatively assessing cervical spine survey radiographs to try to predict a possible compressive spinal cord lesion. Moore et al., in 1994, suggested the intravertebral sagittal ratio that represents the ratio of the minimum sagittal diameter of the vertebral canal to the maximum sagittal diameter of the corresponding vertebral body, obtained at the cranial aspect of the vertebra and perpendicular to the vertebral canal.[38] A sagittal ratio of less than or equal to 50% from C4 to C6 or less than or equal to 52% at C7 is a strong predictor (26.1–41.5 likelihood ratio) of vertebral canal narrowing. The sensitivity and specificity of this method for detecting cervical stenotic myelopathy is greater than or equal to 89% at each vertebral site from C4 through C7.[38] Mayhew and Green, in 2000, reported

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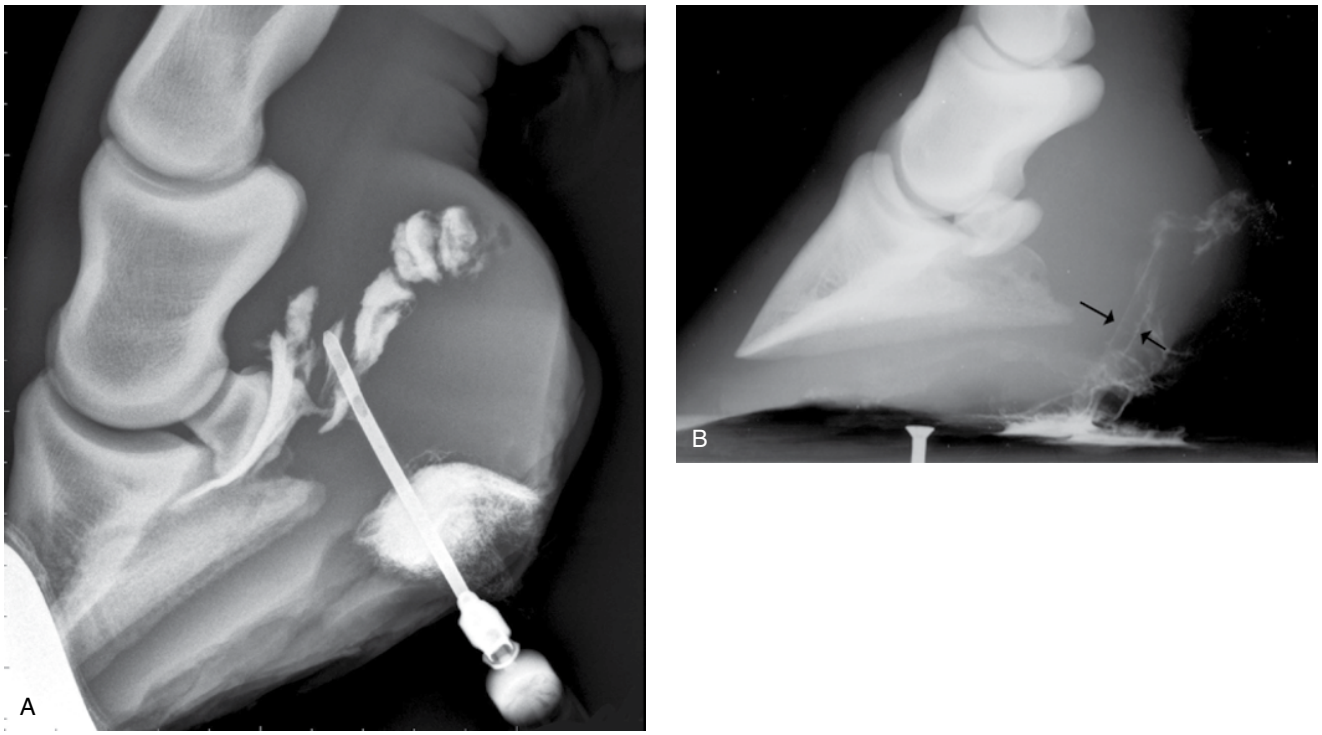


Figure 3.6. (A and B) Lateromedial (LM) projections of the distal limb after injection of iodinated contrast material into an externally draining tract. Image (A) shows filling of the navicular bursa with contrast material consistent with a communication between the draining tract and the bursa. Image (B) shows a well-defined filling defect outlined by contrast material representing a wood foreign body (black arrows).

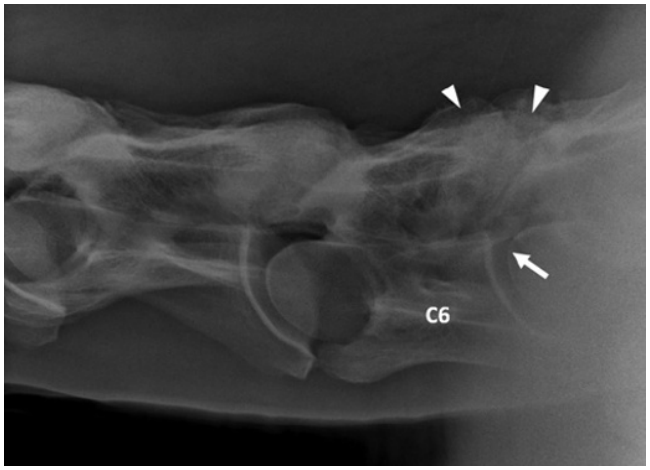


Figure 3.7. Lateral survey radiograph of the caudal cervical spine, showing moderate enlargement and irregular bony proliferation (arrow heads) around the articular facets of C6–C7, with narrowing of the intervertebral foramen (arrow). There is also mild enlargement of the articular facets at C5–C6.

the intervertebral sagittal ratio as a way to evaluate survey radiographs for cervical vertebral malformation. This technique represents the ratio of the minimal distance taken from the most cranial aspect of the vertebral body to the most caudal aspect of the vertebral arch of the vertebra located cranially and the maximal sagittal diameter of the vertebral body.[37] A study by Hahn et al. in 2008 compared the utility of the intra- and intervertebral sagittal ratio methods for diagnosing cervical vertebral malformations. It was concluded that a sagittal diameter ratio of less than or equal to 48% at any intra- or intervertebral site from C2 to C7 represents cervical vertebral malformation.[21] Hughes et al. examined intra- and inter-observer agreement of cervical vertebral sagittal diameter ratios and found that intra-observer repeatability was moderate and that significant inter-observer variation was seen, as such acceptable agreement was rarely achieved for any observer pair.[26] Considering the above discussion, the practitioner should be aware of the inevitable false-positive results because some horses may have a narrow vertebral site and not necessarily represent a spinal cord compressive lesion, as well as being cognizant of the subjective nature of such measurements and their interpretation.

Myelography with nonionic, water-soluble contrast material has proven to be an acceptable and safe diagnostic procedure in the horse.[8,33,36,44] Iohexol and iopamidol are nonionic contrast agents that have relatively few side effects.[33,37,61] With the horse under general anesthesia and in the lateral recumbent position, its head is elevated, and approximately 40–50 mL iopamidol 300, or 370 mg iodine/mL or iohexol 300, or 350 mg iodine/mL is injected into the subarachnoid space at the cisterna magna.[8,36] Iohexol may produce slightly less of an inflammatory reaction than iopamidol, but both contrast agents are relatively safe for myelography in the horse.[8] The higher concentration contrast material provides better contrast in large horses.

Lateral cervical radiographs centered over the cranial, mid-cervical, and caudal cervical regions are made with the spine in a neutral position. A flexed lateral

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projection is made in the mid-cervical region, and an extended lateral one is made in the caudal cervical region. Additional flexed and extended projections at different sites may be necessary. The contrast material can be visualized via ventrodorsal radiographs over the cranial and mid-cervical regions, but because of the thickness of the body area, it is usually not possible to visualize the contrast material in the caudal cervical region of the adult horse.

On a normal myelogram study, several variations need to be noted and not confused with false-positive lesions. On neutral lateral radiographs, there are areas of some degree of elevation of the ventral contrast column at each intervertebral disk. On flexed lateral radiographs, there is also narrowing of the dorsal subarachnoid space, most frequently at C3–C4 and C4–C5, and the ventral column at these locations appears as a thin line. On extended lateral views, the dorsal column does not narrow, and the ventral column is increased in width.

Most lesions detected via cervical myelograms in the horse are compressive lesions from cervical stenosis, either bony or ligamentous, or the result of vertebral instability (Figure 3.8). These lesions may cause substantial narrowing, obliteration, or displacement of the contrast column. The most common sites of compression, in order of decreasing frequency, are C3–C4, C6–C7, C5–C6, and C4–C5.[47] Suggested methods for assessment of a spinal cord compressive lesions include the reduction of 50% or more of the dorsal contrast column compared with the thickness of the subarachnoid space cranial to the narrowing,[44] narrowing of the dorsal and ventral contrast columns by more than 50% in



Figure 3.8. (A) Normal flexed lateral myelogram of the cranial cervical region, showing contrast material in the subarachnoid space and narrowing of the ventral column at C3–C4 (arrow). No narrowing of the dorsal column or significant spinal cord compression is present. (B) Flexed lateral myelogram of the cranial cervical region, showing ventral and dorsal (arrows) contrast columns narrowed by more than 50% and a narrowed spinal cord, suggesting dynamic cervical stenosis. (C) Extended lateral myelogram of the mid-cervical region at the level of C4–C5, showing stenosis of the spinal canal on the cranial aspect of C5 with narrowing of the dorsal contrast column by at least 50% (arrow).

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diametrically opposed sites,[47] and 40% reduction of the entire dural diameter.[58]

The results obtained from these methods should be interpreted with caution due to the risk of false-positive results. It has been suggested that a reduction of 70% of the dorsal contrast column is needed to avoid false-positive diagnosis.[6] A study by van Biervliet et al. in 2004 showed a high sensitivity and specificity of the reduction of 20% of the entire dural diameter at C6–C7 in neutral or flexed myelographic projections for diagnosing cervical stenotic myelopathy. Using this parameter at any other site, the test had only low sensitivity and high specificity.[5] One should be aware that besides obtaining false-positive results, misinterpretation of myelograms can occur with suboptimal technique or when the lesion is lateralized, and only lateral radiographic views are made. Increasing use and availability of CT myelography of the cervical spine has also revealed lateralized compressive lesions from enlarged articular process joints that may be occult on standard radiographic myelograms.[30]

Arthrography

For diagnosis of articular cartilage and subchondral bone injury, arthrography in most cases has been replaced by more advanced imaging modalities and arthroscopy. However, positive and double contrast examinations may provide diagnostic information when evaluating cartilage and subchondral bone damage in joints that are not accessible for advanced imaging such as the shoulder.[42] Arthrography continues to prove useful in assessment of traumatic joint injuries for joint capsular disruption or synovial defects (fistulae or hernias). In cases of trauma, care must be exercised to not enter the joint through damaged or contaminated tissues to minimize the risk of joint infection or inflammatory reaction.

Tendonography

Although the use of ultrasound has rendered contrast studies of tendon sheaths almost obsolete, there remain cases in which contrast studies can be useful and can be more readily interpreted than an ultrasound evaluation. In particular, the use of contrast studies when trying determining the presence of synovial herniation or intrasynovial communication is generally rewarding.[14] For example, distension of an extensor tendon sheath on the dorsal aspect of the carpus is easily evaluated with a contrast study and can determine if communication with the carpal joint exists. In addition, the use of intrathecal contrast radiography alongside intrathecal analgesia for detection of digital flexor tendon sheath pathology has been shown to provide high sensitivity (96%) and specificity (80%) for detection of manica flexoria tears.[16] Reported detection of these tears on ultrasonographic evaluation alone is as low as 52%.[53]

Other modalities such as computed tomography and magnetic resonance imaging have proved to add substantial diagnostic information in the diagnosis of soft tissue and bone injuries and will be discussed further in this chapter.

PRINCIPLES OF RADIOGRAPHIC INTERPRETATION

There are three basic steps to radiographic interpretation: (1) evaluating the quality of the examination, (2) listing radiographic findings, and (3) synthesizing a radiographic impression, diagnosis, and/or prognosis. Checking film exposure, labeling, collimation, and positioning are performed to evaluate image quality. This is an important step because poor-quality radiographs result in missed or improperly diagnosed conditions. A properly exposed radiograph should allow observation of bone and soft tissue outlines, and the film detail should be sufficient to demonstrate bone trabeculae.

Positioning should be evaluated by inspecting joint space and bone alignment. Poorly positioned studies may result from the horse not standing squarely and/or improper alignment of the X-ray tube and detector.

Complete radiographic studies with orthogonal and oblique images are required for thorough evaluation of the region of interest. Limiting studies for the sake of saving time or money risks lesions going undetected. In addition to standard studies, special projections are sometimes needed to better define and demonstrate suspected lesions.

When reading the radiograph, a systematic thorough inspection of the entire image should be done so that nothing is missed. Identifying radiographic abnormalities requires knowledge of both normal radiographic anatomy and radiographic signs of disease. Without knowledge of either, a correct radiographic interpretation is usually not made and risks both false-negative and false-positive diagnoses. One of the most common errors in assessment is termed "satisfaction of search" and refers to the tendency to incompletely examine the study for other abnormalities once one lesion is identified. This is particularly common when other abnormalities are on the periphery of the image.

The third step is formulating a radiographic impression, diagnosis, or differential diagnosis. Knowledge of disease pathophysiology and its relationship to radiographic signs is necessary for this step. The radiographic diagnosis cannot be made in a vacuum, and the radiographic findings and their significance must be correlated with other diagnostic information, such as history, signalment, physical examinations, and perineural anesthesia results, to arrive at a final diagnosis.

Radiology of Soft Tissue Structures

Soft tissue changes may be primary pathologic changes, secondary to more serious bone changes, or incidental findings of no clinical significance. Fascial planes, tendons, ligaments, and some portion of joint capsules may be seen because of adipose tissue (fat) within and around these structures. Fat is less opaque and appears slightly darker than muscle, skin, tendons, or ligaments on a radiograph (Figure 3.9). Soft tissue structures should be evaluated for thickening, mineralization, and free gas (radiolucencies).

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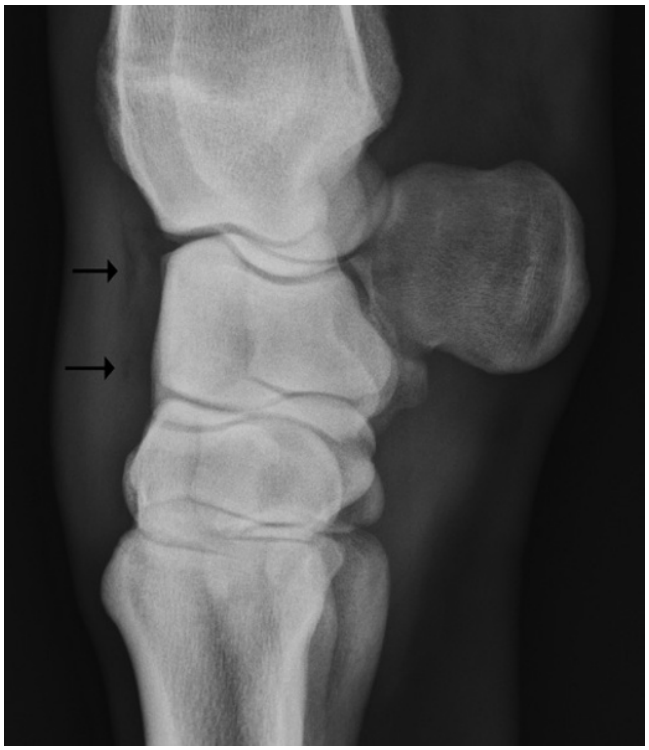


Figure 3.9. Lateromedial (LM) projection of a normal carpus showing the fat pads (adipose tissue bodies) as lucent structures within the dorsal soft tissues (arrows).



Figure 3.10. Lateromedial (LM) projection of carpal effusion. Note the soft tissue thickening on the dorsal aspect of the carpus obliterating the normal fat pads (see Figure 3.9). There are multiple, small, and irregular osseous bodies on the palmar and dorsal aspects of the carpus consistent with osteochondral fragments.

Soft Tissue Thickening

Soft tissue thickening in the equine extremity can be localized or diffuse. Diffuse swelling is often associated with edema or cellulites. Localized thickening may be identified radiographically within or around joints, tendons, or muscles (Figure 3.10). Differentials include synovial effusion, direct soft tissue injury such as tendinous lesions, and masses such as granulomas and abscesses, among other differentials. Though neoplasia of the equine limb is rare, it should still be considered as a differential when warranted. Radiographic signs of soft tissue thickening include an increased soft tissue prominence, displacement of fat bodies (adipose tissue) around the joint capsule or tendon sheaths, and mottling or obliteration of adipose tissue in fascial planes around muscles, joint capsules, or tendons.

Mineralization

Dystrophic mineralization of soft tissue in equine limbs is most frequently secondary to trauma or degenerative injury. Mineralization within ligamentous and tendinous structures usually indicates chronic or more advanced degenerative disease and is most commonly seen in the suspensory ligament branches and distal deep digital flexor tendon (DDFT; Figure 3.11).[45] Synovial mineralization and articular chondral fragment mineralization indicate chronic joint disease. Hematomas, granulomas, and areas of necrosis can mineralize as can certain types of neoplasia such as mast cell tumor.[34] Calcinosis circumscripta is a form of dystrophic mineralization most frequently seen periarticular in the horse. A round



Figure 3.11. Lateromedial (LM) projection of the distal extremity. Linear soft tissue mineralization is present just palmar to the flexor cortex of the navicular bone consistent with dystrophic mineralization of the deep digital flexor tendon in this region.

"cauliflower-shaped" appearance is usually present with calcinosis circumscripta.

Gas

Gas may be present in the soft tissue structures of equine limbs as a result of traumatic lacerations, puncture

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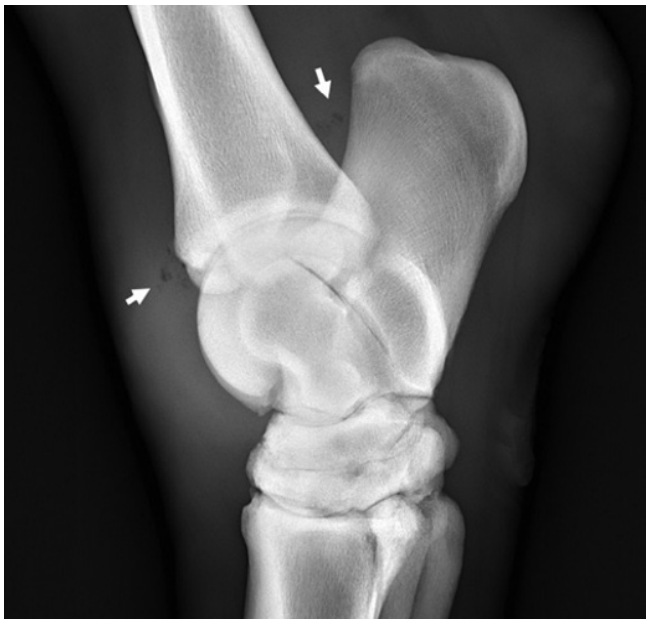


Figure 3.12. Lateromedial projection of the tarsus. Multiple, small gas opacities are visible within the proximal aspect of the tarsocrural joint (arrows). This horse was subsequently diagnosed with septic arthritis of the joint, and the presence of intra-articular gas was likely a result of anaerobic bacterial infection. Degenerative changes within the distal tarsal joints were an incidental finding.

wounds, needle centesis, or gas-producing bacterial organisms. Radiographic signs of soft tissue emphysema include radiolucent regions within soft tissue structures (the radiolucencies should be differentiated from fat) and a focal accumulation of gas with an air–fluid level that occurs with abscesses.

Localization of intra-articular gas is important diagnostically and prognostically. When gas is identified in a joint (Figure 3.12), there are three primary causes: (1) iatrogenic, incidental gas due to synovial centesis, (2) septic arthritis, and (3) "vacuum phenomenon" (nonclinically significant) gas that occurs when a joint is flexed.[20,54] In rare cases, idiopathic gas is found in markedly osteoarthritic joints; in the authors' experience, this primarily occurs in the carpus and is of uncertain origin. Because of the high clinical importance of diagnosing septic arthritis, it is preferable to obtain radiographs of a joint suspected of being septic prior to synovial centesis to reduce confounding factors for interpreting sources of intra-articular gas.

Entheses

An enthesis is a point in the bone at which a soft tissue structure attaches. The soft tissues involved can include tendons, ligaments, or joint capsule. A pathologic change at these sites is known as enthesopathy and can be secondary to many disorders, but most commonly includes trauma or degenerative or inflammatory conditions and may be intra- or extra-articular (Figure 3.13). In cases of an acute traumatic event, an avulsion fracture at the enthesis may occur. Radiographic changes associated with enthesopathy include bone erosion, bone proliferation or hyperostosis (thickening of cortical bone), sclerosis, fragmentation, and adjacent soft tissue mineralization. Enthesopathy can provide indirect evidence of other pathologic processes such as joint disease or ligamentous or tendinous injury.

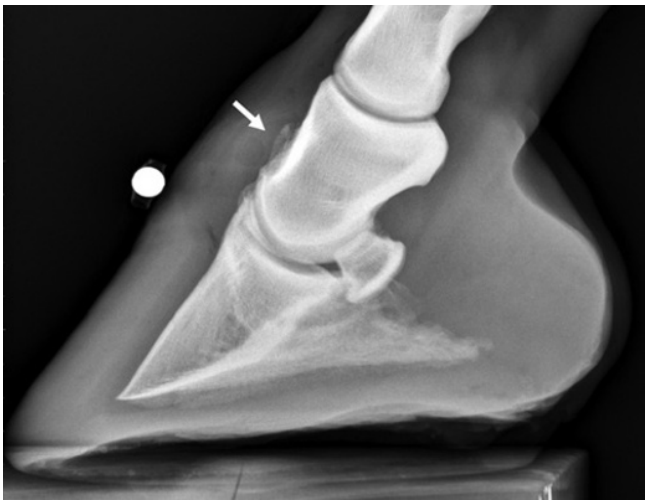


Figure 3.13. Lateromedial projection of the front foot with irregular osseous proliferation along the dorsal distal aspect of the middle phalanx (arrow), consistent with enthesopathy of the distal interphalangeal joint capsule. On magnetic resonance imaging of this region, marked diffuse articular cartilage damage was visualized within the joint.

Radiology of Bone

Fundamental Patterns of Bone Response

The response of bone to different pathologic processes is limited and consists in one or a combination of the following processes: new bone production/formation, bone resorption/destruction, or shape alteration. Bone remodeling and bone modeling are two terms that are often used interchangeably when referring to musculoskeletal radiology. Physiologically, osteoclastic and osteoblastic activities occur in both processes.

During the bone remodeling process, the osteoblasts and osteoclasts are coupled and act together; therefore, bone resorption and formation occur at the same time and at the same site on a bone surface.[18] On the other hand, during the bone modeling process, the bone alterations result from independent actions from osteoclasts and osteoblasts. This means that the bone resorption and formation may occur on different sites. In addition, modeling may cause large changes in bone structure, whereas remodeling will replace bone, maintaining the current amount of bone structure.[17] Therefore, the correct term for describing visible bone structural changes is usually modeling rather than remodeling.

Radiographically, new bone production is frequently manifested as a periosteal or endosteal reaction (adjacent to the cortex), new bone at periarticular margins or entheses, or increased bone opacity (sclerosis). Areas of bone destruction are seen as bone lysis (aggressive bone lesions or osteoarthritis [OA]) or decreased bone opacity (osteopenia). Pathologic shape alteration is commonly seen radiographically in, but not limited to, skeletally immature horses, usually secondary to physeal

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problems or abnormal weight bearing. To formulate a correct diagnosis, the clinician should note fundamental bone response patterns and distribution within bones and any associated soft tissue changes on equine limb radiographs. The clinician also should be able to differentiate whether these changes are a response to pathologic processes or secondary to normal bone modeling as adaptation to a particular athletic activity.

Periosteal Reactions

The periosteum is stimulated when elevated by hemorrhage, purulent material, edema, or infiltrating neoplastic cells. In the horse, direct trauma, extension of soft tissue infections, and avulsion of ligaments, tendons, and/or joint capsules are most frequently associated with periosteal new bone production. Periosteal bone production may be acute or chronic (Figure 3.14). Acute periosteal bone production has an irregular, indistinct border and may be continuous or interrupted, laminated, or speculated. Acute periosteal reaction is usually active. Chronic periosteal bone production has a smooth, well-defined border, is solid, and often blends with the adjacent cortex. This type of periosteal reaction is usually inactive and often indicates a healed process such as a healed fracture or previous active periosteal bone production that has changed to a chronic, probably inactive stage (Figure 3.14).

Cortical Changes

Cortical changes that can be identified radiographically consist of defects, erosions, lysis, and changes in thickness. Cortical defects seen most frequently in equine extremities are caused by fractures. Fractures must be differentiated from nutrient foramina (Figure 3.15), physeal lines, and edge enhancement shadows caused by superimposed bones. Long bone cortical stress fractures may not be evident in all cases as a distinct fracture line (Figure 3.16); a periosteal and/or endosteal reaction may be the only visible radiographic change (Figure 3.17). When a long bone fracture is suspected but not visible radiographically, it is essential to continue to treat the patient conservatively as though there were a fracture and utilize further imaging including nuclear scintigraphy and/or repeat radiographs in approximately 10 days.

Cortical lysis is usually caused by infection and typically has a permeative and/or moth-eaten pattern. A sequestrum also may be associated with a focal area of cortical lysis (Figure 3.18). In such cases, a dense sequestered piece of bone can be identified surrounded by a lytic zone (cloaca), which in turn is surrounded by bone sclerosis, producing an involucrum within the parent bone.

Cortical erosion changes can extend from either the endosteal or the periosteal surface. In the horse, they are most frequently encountered adjacent to the periosteal surface. Erosive changes with an irregular border usually result from infiltration into the bone and are most often caused by infectious processes. Cortical erosive areas with a smooth border are the result of pressure



Figure 3.14. Dorsolateral to palmaromedial oblique (DLPMO) projection of the metacarpus. Note the smooth periosteal reaction and cortical thickening of the mid-distal diaphysis of MCII, consistent with a chronic exostosis.



Figure 3.15. Lateromedial (LM) projection of the pastern. A radiolucent line is seen through the dorsal cortex of the mid portion of the first phalanx (arrow). This is a nutrient foramen that should not be mistaken for a fracture.

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Figure 3.16. Craniolateral to caudomedial oblique projection of the tibia. A stress fracture of the tibia is present on the laterocaudal cortex of the mid-diaphysis. Note the smooth, thickened periosteal reaction with a faint radiolucent cortical fracture line (arrow). A subtle endosteal reaction is also present at that level. *Source:* Courtesy of New Bolton Center, University of Pennsylvania.

erosion (Figure 3.19), such as that seen with proliferative synovitis in the metacarpophalangeal or metatarsophalangeal joints.

Cortical erosions seen on the flexor surface of the navicular bone are common and significant sites of disease.[66] Flexor cortical erosive lesions most frequently begin by affecting the palmar fibrocartilage and subsequently create bone erosion.[7] However, it is also possible to have flexor cortical erosions as a result of pressure resorption from dilated synovial invaginations, possibly associated with distal interphalangeal synovitis, which affect the endosteal surface and then extend palmarly.[46] The palmoroproximal to palmorodistal oblique (skyline) of the navicular bone is an essential radiographic view to assess these types of lesions, should be standard in the foot protocol, and may require several varying angles to adequately assess the flexor surface (Figure 3.20).[27]

Cortical bone change is also associated with enthesopathy and can be proliferative, resorptive, or both. Joint capsule enthesopathies and the palmar ligaments of the pastern often are proliferative. Resorptive lesions are more frequently found at the insertion of the collateral ligaments on the distal phalanx (Figure 3.21) and the insertion of the impar ligament on the flexor surface of the distal phalanx.[10,65] The origin of the proximal suspensory ligament on the third metacarpal/metatarsal (MCIII/MTIII) bones and the insertion of the suspensory ligament branches on the sesamoid bones often have mixed resorptive and proliferative patterns.[19,55,57,60]

Cortical thickening is usually produced by increased weight bearing. Such changes in cortical width are frequently present with valgus or varus limb abnormalities or as an adaptive response to exercise.

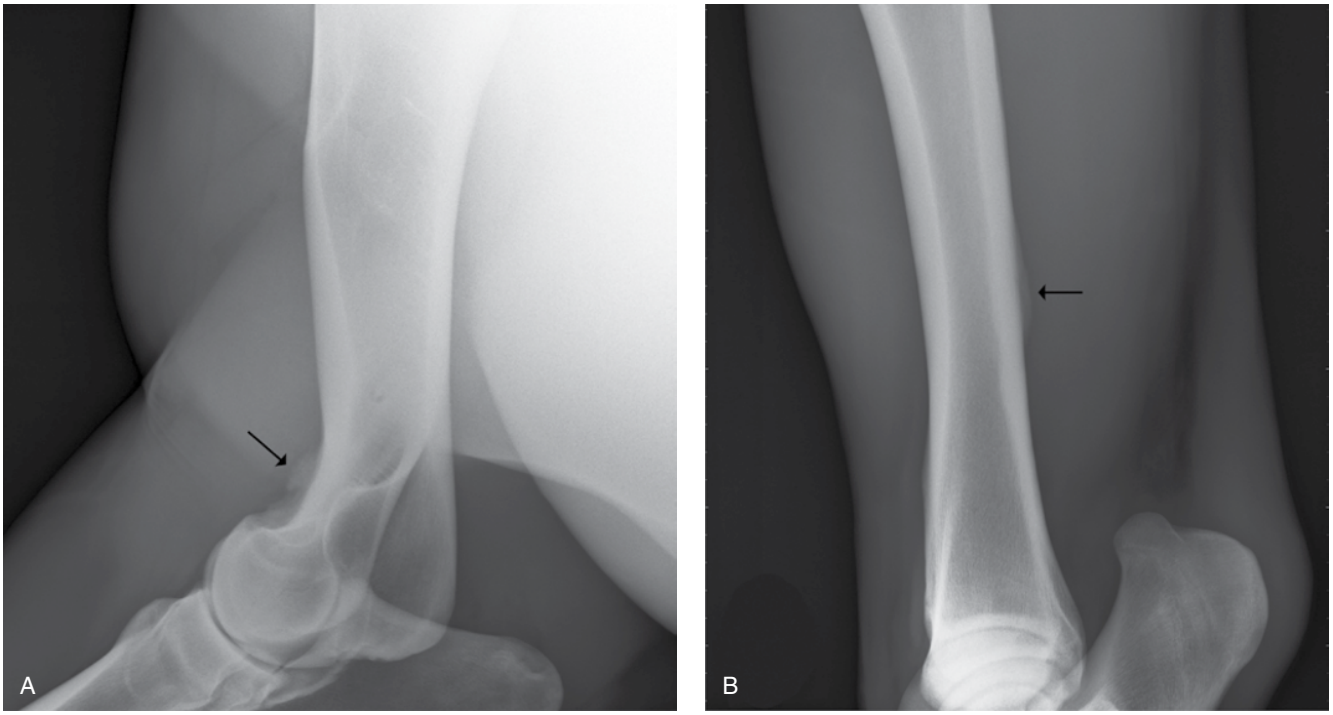


Figure 3.17. Examples of humeral (A) and tibial (B) stress fractures with only radiographic evidence of a periosteal reaction (arrows). *Source:* Courtesy of New Bolton Center, University of Pennsylvania.

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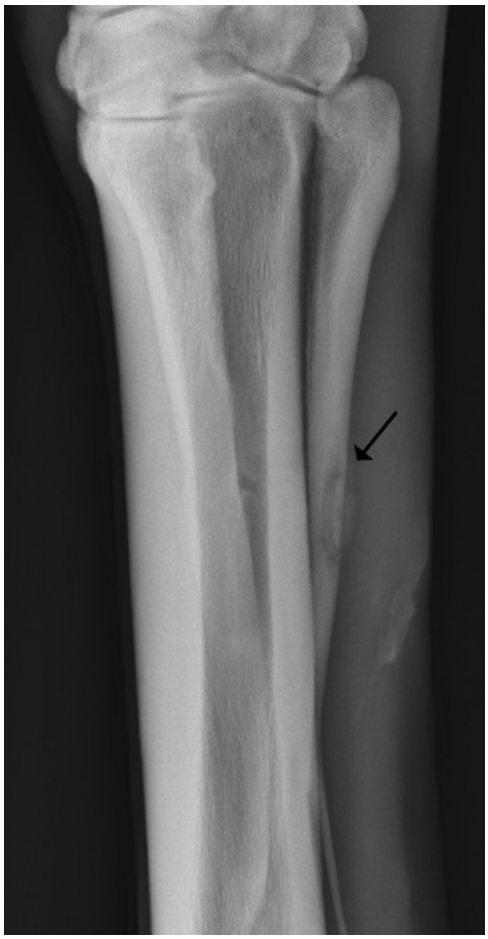


Figure 3.18. Dorsolateral to palmaromedial oblique (DLPMO) projection of the metacarpus showing a well-defined osseous body (sequestrum) surrounded by a radiolucent rim (cloaca) on the mid-diaphysis of MCIV. There is mild surrounding sclerosis and a small periosteal reaction just proximal to the cloaca, representing the involucrum (arrow).



Figure 3.19. Lateromedial (LM) projection of the metacarpophalangeal joint with marked supracondylar bone loss on the palmar cortex of distal MCIII (short arrow), secondary to chronic inflammation and effusion within the joint.



Figure 3.20. Palmaroproximal to palmarodistal oblique (Pa45Pr-PaDiO, skyline) projection of the navicular bone. On the flexor aspect of the navicular bone, note the poor corticomedullary definition, irregular medullary sclerosis, and cortical erosions consistent with advanced degenerative changes.

Trabecular Bone

Generalized bone opacity may be decreased or increased. Decreased bone density is seen secondary to chronic moderate to severe lameness resulting in decreased load bearing. The osteopenia that develops in these limbs can be recognized radiographically as a coarse trabecular pattern with or without thin cortices (Figure 3.22).

Increased bone opacity, often referred to as sclerosis, is identified radiographically with loss of trabecular pattern secondary to bone deposition within the medullary cavity (Figure 3.23). Sclerosis is a common adaptive response to load bearing and is frequently found in the third carpal bone and central and third tarsal bones (Figure 3.24). Increased bone density is also routinely seen at proximal MCIII or MTIII at the origin of the suspensory ligament.[23,56]

Radiographic Signs (Bone Response Patterns) with Osteomyelitis

Osteomyelitis in equine limbs may be hematogenous in origin or result from penetrating wounds or open fractures. The region affected depends on the source and route of infection. Both acute and chronic osteomyelitis can be identified radiographically (Figures 3.25 and 3.26). It generally takes 7–10 days after clinical signs of acute osteomyelitis are observed before the earliest detectable radiographic bone changes occur because at least 50% of mineral content must be depleted from the bone to be radiographically visible.

Because osteomyelitis can affect any bone in an equine limb and must be differentiated from other focal bone

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Figure 3.21. Dorsoproximal to palmarodistal oblique view of the distal phalanx. Osteolysis and irregular margins of the collateral ligament fossa (arrow) indicate resorptive enthesopathy. This finding was confirmed with magnetic resonance imaging.

lesions, it will be used to illustrate the use of radiographic signs or bone response patterns to arrive at a radiographic diagnosis. Identifying radiographic signs requires close inspection of the radiograph and is an important step in accurately establishing a specific or differential diagnosis.

The following are radiographic signs manifested by acute osteomyelitis:

- Soft tissue thickening adjacent to the bone is observed as increased opacity, mottling, and obliteration of adipose tissue in fascial planes.
- Periosteal new bone production. This new bone has an irregular, indistinct border and parallels the bone cortices. Subtle areas of subperiosteal bone lysis may be seen in association with the acute periosteal bone reaction. These changes are not usually seen until 7–10 days after clinical signs of the disease have been observed. As the disease progresses, the periosteal bone production parallels the cortex and spreads proximal and distal from the original infection site.
- Permeative lysis is observed as small round lucent areas, 2–3 mm in size, within cortical bone and may extend into the medullary cavity. A moth-eaten pattern of lysis with larger lucent areas can also be seen in acute and active aggressive lesions. These changes are usually seen in association with acute periosteal bone production.
- Areas of bone lysis within the physis, metaphysis, or epiphysis secondary to septic osteomyelitis in young animals are very aggressive and mostly destructive, giving little or no opportunity to the body to produce bone in the attempt to ward off the infection.





Figure 3.22. (A) Lateromedial (LM) and (B) dorsopalmar (DP) projections of a distal limb with rotating laminitis. Note the coarse trabecular pattern (more noticeable on distal P1 and proximal P2) secondary to disuse osteopenia from chronic non-weight-bearing.

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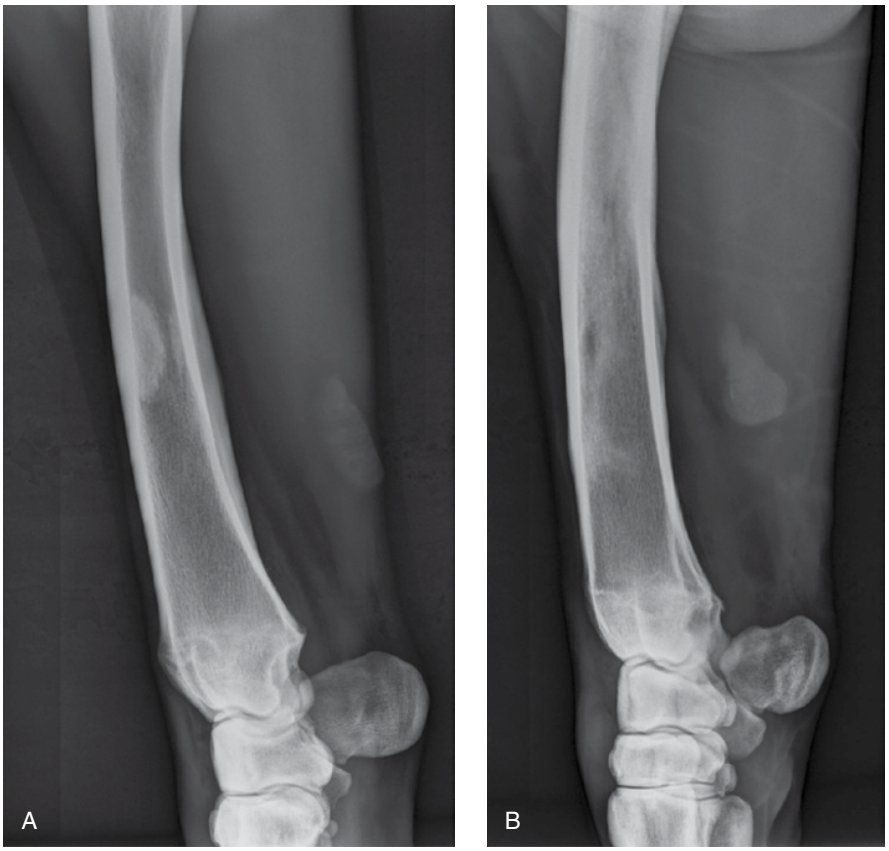


Figure 3.23. Lateromedial (LM) projections of the radius. Image (A) shows a well-defined, oval medullary sclerosis adjacent to the endosteal surface of the cranial mid-diaphysis and a similar elongated opacity adjacent to the caudal endosteal surface at the same level. Image (B) shows a diffused, irregular medullary sclerosis affecting mostly the proximal and mid thirds of the diaphysis. Nuclear medicine exams showed increased radiopharmaceutical uptake in both cases, which is consistent with enostosis-like lesions. *Source*: Courtesy of Dr. Jeremy D. Hubert.

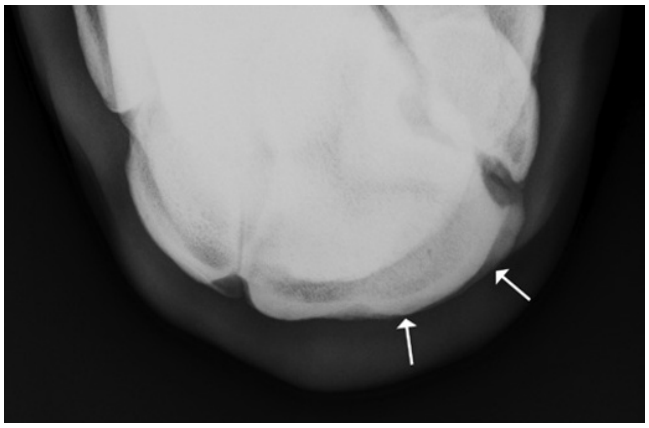


Figure 3.24. Flexed dorsoproximal to dorsodistal oblique (flexed D30Pr-DDiO) projection of the distal row of carpal bones. Note the sclerosis of the radial facet of the articular surface of the third carpal bone with partial loss of the corticomedullary definition (arrows).

Chronic osteomyelitis may have the following radiographic changes:

- Large cortical defects, some of which may be as large as 1 cm in size and can also involve the medullary cavity. Although rare, bone abscesses may appear as a geographic pattern of lysis represented by a single well-defined medullary lytic area with surrounding sclerosis.[24,64]



Figure 3.25. Dorsolateral to plantaromedial oblique (D35L-PlMO) projection of the tarsus, showing focal widening of the medial aspect of distal tibial physis with adjacent subchondral bone lysis and marked soft tissue thickening consistent with septic physitis. *Source*: Courtesy of Dr. Jeremy D. Hubert.

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Figure 3.26. Lateromedial (LM) projection of the distal radius with radiographic changes, consistent with osteomyelitis. Note the irregular and discontinued periosteal reaction with lysis of the craniodistal cortex of the distal radius and adjacent ill-defined increased medullary opacity and soft tissue thickening. On the most proximal aspect of the lesion, the cranial cortex is wider and presents a circular mineral opacity with a surrounding radiolucent rim and sclerosis, suggesting the presence of a sequestrum (arrow). *Source*: Courtesy of Dr. Gary Baxter.

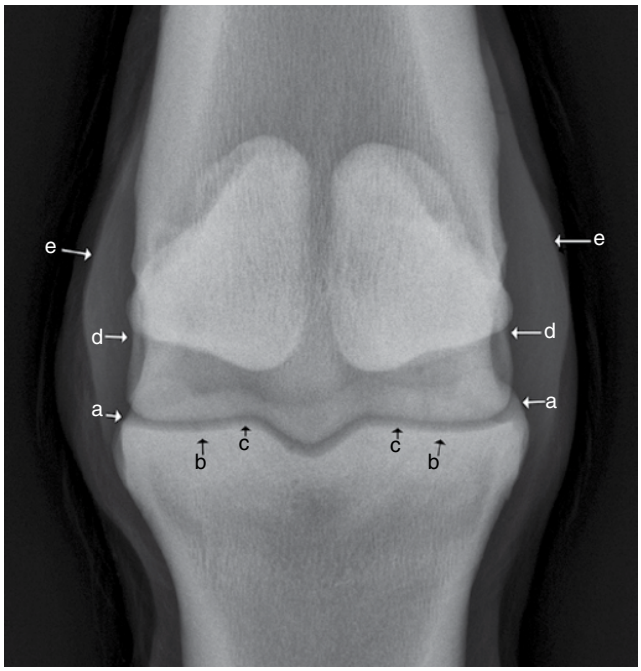


Figure 3.27. Dorsoproximal to distopalmar oblique (D30Pr-DiPaO) projection of a normal metacarpophalangeal joint, showing an example of the structures that should be evaluated around a joint. (a) Joint margins. (b) Subchondral bone. (c) "Joint space" (articular cartilage). (d) Areas for collateral ligament attachment. (e) Joint capsule and general joint alignment.

- Localized increased bone densities (sclerosis), which are produced within the host bone, e.g. thick cortices, and in which a sequestrum sometimes may be identified within the sclerotic and lytic bone patterns.
- Periosteal bone production, which is usually abundant with a well-defined irregular or smooth border.
- It is often impossible to determine radiographically whether chronic osteomyelitis is active or inactive. This diagnosis is best determined by physical examination, clinical signs, or other imaging techniques, such as nuclear scintigraphy.

Radiology of Synovial Joints

Radiographic evaluation of joints in the equine limb is an important part of the diagnostic workup for lameness and encompasses evaluation of multiple components, including soft tissue structures (both intracapsular and extracapsular); joint margins; subchondral bone; joint space and alignment; and ligament, tendon, and joint capsule attachment areas (Figure 3.27).

Normal Joint Structures

The joint capsule and periarticular soft tissue structures should not be distended. Fat bodies and adipose tissue within muscle fascial planes can be identified around some normal joints. Visibility and location of adipose tissue may change with inflammation or joint capsule distension or thickening. The normal location and the presence of fat bodies vary, depending on the joint and animal being examined.

Joint margins are bony regions at the edge of articular cartilage that also coincide with the edge of the subchondral bone. Articular cartilage, the periosteum, and the joint capsule meet in this region. In the normal joint, the margins are smooth and blend with the surrounding bone structures. Subchondral bone is a dense, compact bony zone 1–3 mm in width and adjacent to the articular cartilage. The subchondral bone surface adjacent to the articular cartilage is smooth and even.

The area referred to as radiographic "joint space" is not an actual space but is composed of articular cartilage with a thin layer of synovial fluid between the opposing cartilaginous surfaces. It appears radiolucent on a radiograph, compared with the adjacent radiopaque subchondral bone. The joint space should be of even thickness throughout a specific joint, but thickness differs from joint to joint; e.g. the distal interphalangeal joint space is wider than the proximal interphalangeal joint space, which is wider than the metacarpophalangeal joint space.

Ligaments, tendons, and the joint capsule, which attach periarticularly, add stability to the joint. The attachment areas vary with the joint and may be located at different distances both proximal and distal to the joint margins. It is important to know regions of insertion for ligaments and tendons around specific joints.

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The normal subchondral bone surfaces should align evenly. Positional changes of the horse or of the X-ray tube when the radiograph is made may make a normal joint appear slightly malaligned.

Radiographic Changes Associated with Joint Disease

Radiographs are a first-line screening tool for assessing joint disease. The radiographic manifestations of joint disease occur in the soft tissue and bone structures and may develop before or after clinical signs of the disease develop, depending on the type and severity of disease. Bone changes follow clinical signs in septic arthritis, whereas bony changes may either precede or follow clinical evidence of disease with OA.

Soft tissue changes associated with joint disease may be radiographically evident prior to bone change, although this is somewhat joint dependent. Common soft tissue abnormalities include periarticular soft tissue thickening, joint distension due to effusion and/or synovitis, and mineralization. The location of fat pads and adipose tissue in fascial planes can be used to evaluate periarticular swelling and joint capsule distension. Joint distension is more readily identified in some joints than others; for example, effusion of the fetlock joint is typically obvious, whereas distension of the medial or lateral femoral tibial joints is challenging to detect radiographically. Periarticular mineralization may be associated with numerous causes but in the horse is predominantly dystrophic or secondary to blunt soft tissue trauma.

Periarticular osteophyte formation is the most common change found on the joint margin in the OA process (Figure 3.28). Not all osteophytes are clinically significant, particularly when mild and/or when seen on low-motion joints. Marginal bone lysis can be found on joint margins in joints affected with more advanced OA and is also a hallmark finding for septic arthritis (Figure 3.29). In early stages, marginal changes may be subtle, but with advanced or more severe disease, the changes are easily identified.

Subchondral bone changes include sclerosis, lysis, and fragmentation (Figure 3.30). Subchondral bone sclerosis is often found as an adaptive response to exercise; however, with increasing sclerosis, the sclerosis changes from adaptive to maladaptive and can be associated with degenerative disease and increased risk of subchondral necrosis and fracture.[12,28,39,43] Subchondral bone lysis can have a local or general distribution within the joint and may be seen in association with subchondral bone fragments. Subchondral bone lysis may be present with septic arthritis, OA, and osteochondrosis, as well as secondary to trauma. While lysis is a defining characteristic of septic arthritis, subchondral bone lysis is also seen with OA, particularly in low-motion joints. OA of low-motion joints, particularly the distal tarsus and carpometacarpal joints, can present with extensive subchondral lysis, and it is important to differentiate this from a septic process. Localized, well-defined lytic



Figure 3.28. Lateromedial projection of the carpus. Moderate periarticular osteophytosis is present at the middle carpal and the carpometacarpal joints with mild sclerosis of the cuboidal bones involved.



Figure 3.29. Lateromedial (LM) projection of the distal limb showing radiographic changes consistent with advanced, severe septic arthritis. Note the severe irregular subchondral bone lysis on the adjacent articular surfaces of the distal interphalangeal (DIP) joint with marked articular cartilage loss, circumferential irregular periosteal proliferation, and the severe soft tissue thickening with irregular skin surface centered over the dorsal aspect of the DIP joint. In addition, there is a coarse trabecular pattern on P1 consistent with disuse osteopenia.

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Figure 3.30. Lateromedial (LM) projection of the distal limb. Severe chronic degenerative changes are present on the proximal and to a lesser degree the distal interphalangeal joints consistent with osteoarthritis (high and low "ringbone"). Note the marked undulating bone proliferation (osteophytes and enthesophytes) on the dorsal aspect of P1 and P2 and extensor process of P3; there is severe narrowing of the proximal interphalangeal joint with adjacent subchondral bone sclerosis. Enthesophytes are also seen on the palmar aspect of distal P1 and proximal P2. *Source*: Courtesy of Dr. Gary Baxter.

lesions are seen with osteochondrosis or repetitive trauma, which may develop into subchondral cyst-like lesions. Focal subchondral lytic areas associated with bone fragments are seen with osteochondritis dissecans and traumatic arthritis lesions from chronic microfractures in the subchondral bone.

The joint space width may be increased or decreased. Increased joint space associated with marked subchondral bone lysis can be seen with extensive septic arthritis (Figure 3.29). A decreased joint space, either general or localized within the joint, is associated with cartilaginous lesions and degeneration and occurs predominately with OA. Decreased joint space is usually accompanied by subchondral bone sclerosis (Figure 3.30). It is important to be sure that the patient is standing squarely to avoid positional joint space narrowing.[11]

Periarticular enthesophytes are usually associated with joint capsule, ligament, or tendon damage or avulsion from their bony attachments (Figure 3.13). The location, larger area of bone change, and typically more undulating margins of enthesophytes typically help distinguish them from osteophytes; however, in some locations such as the dorsoproximal third metatarsal bone, it can be difficult to differentiate enthesopathy from osteophytosis.

Alignment abnormalities may consist of subluxation or luxation of a joint or may simply result in an abnormal degree of flexion or extension of a joint in a resting position. Abnormal joint alignment may be associated with ligament laxity and/or injury (Figure 3.31); tendon

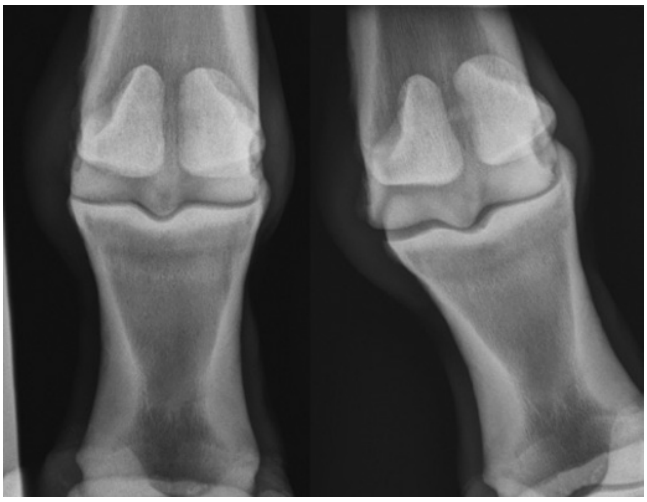


Figure 3.31. Medial (left) and lateral (right) stressed dorsopalmar views of the metacarpophalangeal joint. There is soft tissue swelling along the lateral aspect of the joint. On the lateral stress view, there is asymmetric widening of the lateral aspect of the joint. This is not seen on the medial aspect of the joint during medial stress. These findings are indicative of severe damage or rupture of the lateral collateral ligaments of the fetlock joint.

injury or contracture; abnormal bone growth, i.e. angular limb deformities in foals; and healed, malaligned fractures. Chronic alignment abnormalities predispose the joint to OA from abnormal weight bearing and stress distribution through the joint.

Radiographic Changes with Specific Joint Conditions

As discussed above, there are cardinal radiographic changes associated with joint disease. Some of the changes are seen across multiple types of joint disease, whereas other findings are predominant for specific types of pathologic processes. Below is a summary of typical radiographic findings of specific joint conditions.

Osteoarthritis

OA is a common pathologic process in horses. The changes seen radiographically are secondary to inflammation and degenerative processes within the joint, predominantly those affecting the articular cartilage. The degree of secondary change not only is somewhat dependent on the severity and chronicity of the disease process but also can vary by joint, as some joints are more prone to secondary osteoarthritic changes than others (Figures 3.28, 3.30, and 3.32). The following are radiographic changes seen in OA of the horse:

- Periarticular osteophytes.
- Subchondral bone sclerosis.
- Narrowed joint space, which may involve all or only part of the joint; distinct, smooth borders remain on the subchondral bone adjacent to the articular cartilage.
- Focal or multifocal, well-defined subchondral bone lysis, most frequently in low-motion joints.
- Degenerative subchondral cyst-like lesions secondary to chronic subchondral bone and articular cartilage injury.

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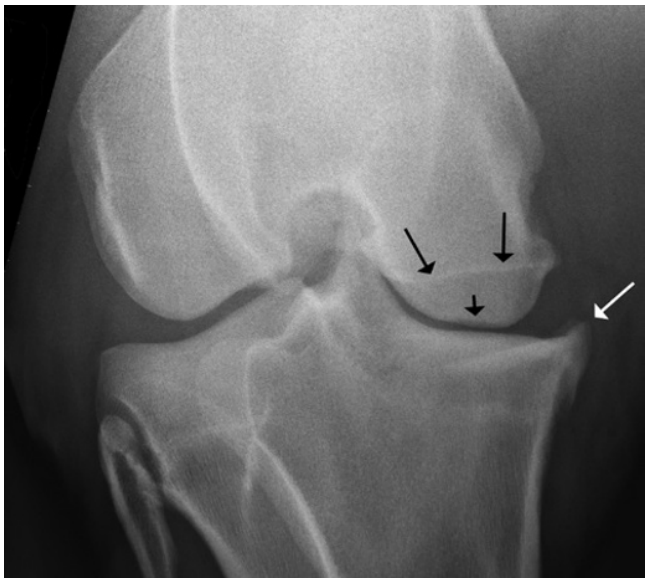


Figure 3.32. Caudocranial (Ca-Cr) projection of the stifle. A rounded periarticular osteophyte is present on the medial periarticular margin of the medial tibial condyle, consistent with osteoarthritis (white arrow). The articular surface of the medial femoral condyle has a slight concave defect (short black arrow). The horizontal radiopaque line on the cranial margin of the medial femoral condyle (long black arrows) indicates enthesophytosis at the insertion of the medial femorotibial and medial aspect of the femoropatellar joint capsules.

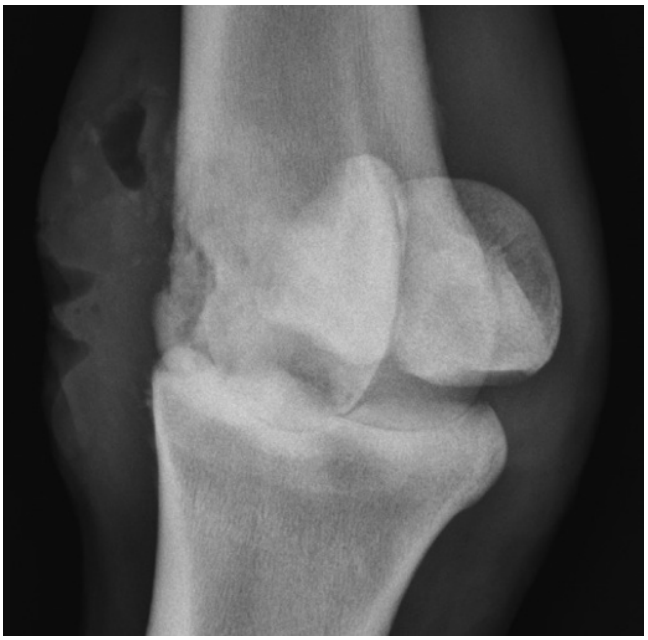


Figure 3.33. Dorsomedial to palmarolateral oblique (DMPLO) projection of the metacarpophalangeal joint. An area of permeative lysis is present on the dorsal aspect of the lateral condyle of MCIII. Marked narrowing of the joint space, mild irregular periosteal reaction on the dorsolateral aspect of proximal P1, and associated soft tissue thickening with subcutaneous gas are also present. These findings are consistent with septic arthritis.

Septic Arthritis

In foals, septic arthritis is most commonly hematogenous in origin. Septic arthritis in adult horses typically occurs from penetrating wounds, as an iatrogenic consequence of needle centesis or, less frequently, an extension of adjacent osteomyelitis or cellulitis. (Figures 3.29 and 3.33). The following are radiographic signs of septic arthritis:

- Periarticular soft tissue thickening and joint distension.
- Marginal bone lysis, which may occur early in the disease.
- Subchondral bone destruction, which may be an extension from or occur without the marginal lysis.
- Periosteal reactions, which may be adjacent to the joint but are generally distributed around the joint; when septic arthritis has occurred from extension of an adjacent osteomyelitis or cellulitis, the periosteal reaction may precede the intra-articular changes.

Osteochondrosis

Osteochondrosis is associated with regions of high predilection in specific joints, which should be observed when diagnosing the condition. In the horse, the stifle, tarsus, and fetlock are most commonly affected by osteochondrosis, although lesions can be found in all joints. It is caused by defective osteochondral development, which usually involves subchondral bone (Figures 3.34–3.37). The following are radiographic changes present with osteochondrosis:



Figure 3.34. Dorsolateral to palmaromedial (DLPMO) projection of the tarsus. There is a well-defined, irregular lucent line through the distal intermediate ridge of the tibia with an associated osseous fragment (arrow). There is mild sclerosis surrounding the lesion. This is a common site of osteochondritis dissecans (OCDs).

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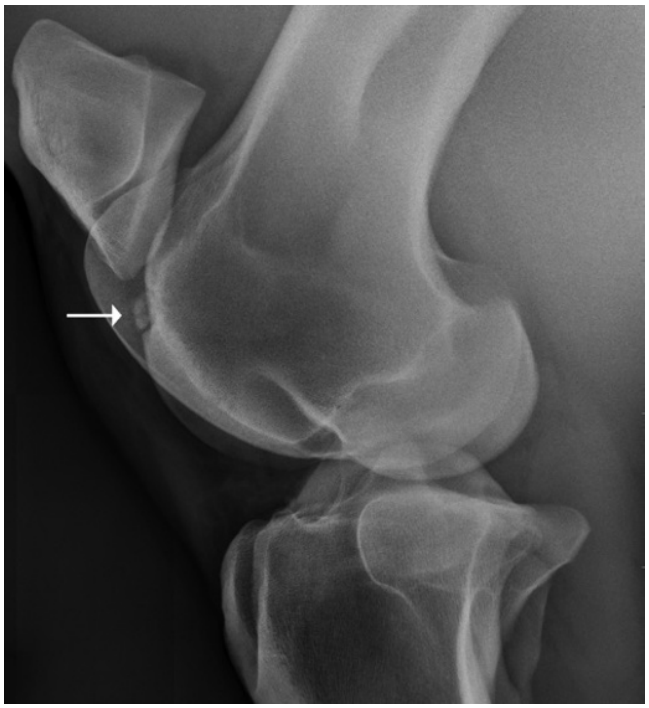


Figure 3.35. Lateromedial (LM) projection of the stifle. Note the well-defined, oval, osseous body associated with a concave subchondral bone defect on the lateral trochlea ridge (arrow), consistent with osteochondritis dissecans.

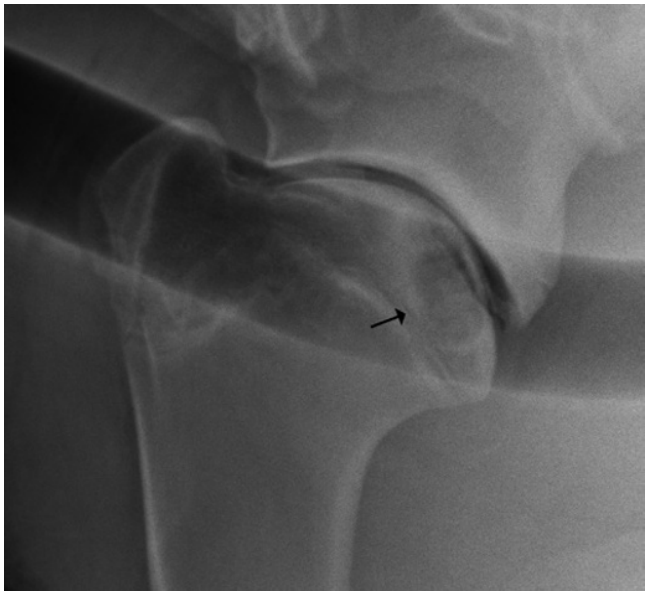


Figure 3.36. Mediolateral (ML) projection of the shoulder. A large radiolucent subchondral bone defect (cyst) surrounded by a rim of sclerosis is seen on the caudal aspect of the humeral head (arrow), consistent with osteochondrosis.

- Flattening of the subchondral bone surface with adjacent subchondral bone sclerosis.
- Localized subchondral bone defect, typically well defined and generally smooth.
- Osteochondral bone fragments, which are seen radiographically as osseous bodies representing osteochondritis dessecans.

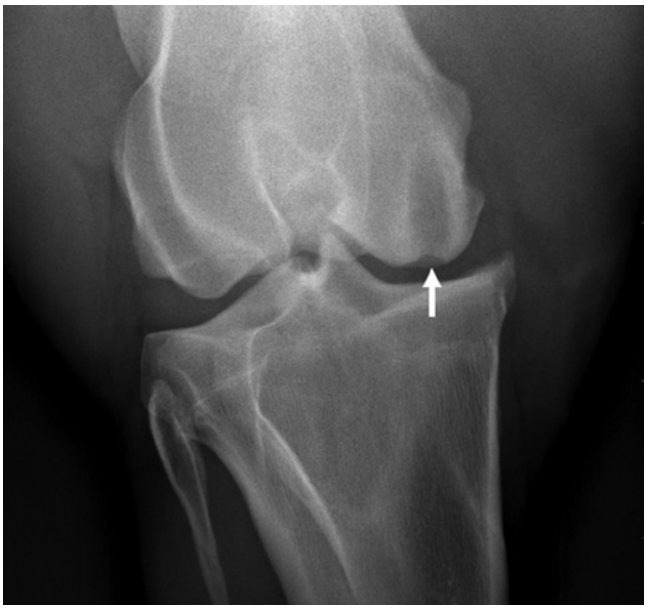


Figure 3.37. Caudocranial (CdCr) projection of the stifle. There is a large, well-defined ovoid radiolucent subchondral bone defect (cyst) surrounded by a rim of sclerosis in the medial femoral condyle (arrow). Subchondral cystic lesions of the medial femoral condyle can be a result of osteochondrosis or trauma.

- Secondary osteoarthritic changes that may also be present.
- Subchondral cyst-like lesions that may develop secondary to osteochondrosis.
- Joint distension may be present, particularly noticeable in the tarsocrural joint.

Traumatic Joint Disease

Traumatic joint disease is caused by repetitive trauma that causes microfracture and bone necrosis and can lead to subchondral lysis, cystic lesions, and fragmentation.[28] Areas of predilection for traumatic joint disease are the metacarpophalangeal joints (Figure 3.38) and carpus (Figure 3.39). Traumatic joint disease may also develop after hyperextension injury in joints such as the carpus, producing microfractures, subchondral bone lysis, and small subchondral bone fragments. The following are radiographic signs of traumatic joint disease:

- Focal subchondral bone lysis with indistinct borders
- Subchondral bone sclerosis
- Bone fragments, which may be adjacent to the subchondral bone lysis
- Incomplete articular fractures or subchondral fissures (stress fractures)
- Changes in contour of the bones, such as flattening of the articular surface

LIMITATIONS OF RADIOGRAPHY

Radiography is the most common imaging modality and is widely used for the diagnosis of musculoskeletal injuries. While an excellent screening tool, radiography has well-known limitations. Radiography allows identification of soft tissue thickening and indirect evidence of soft

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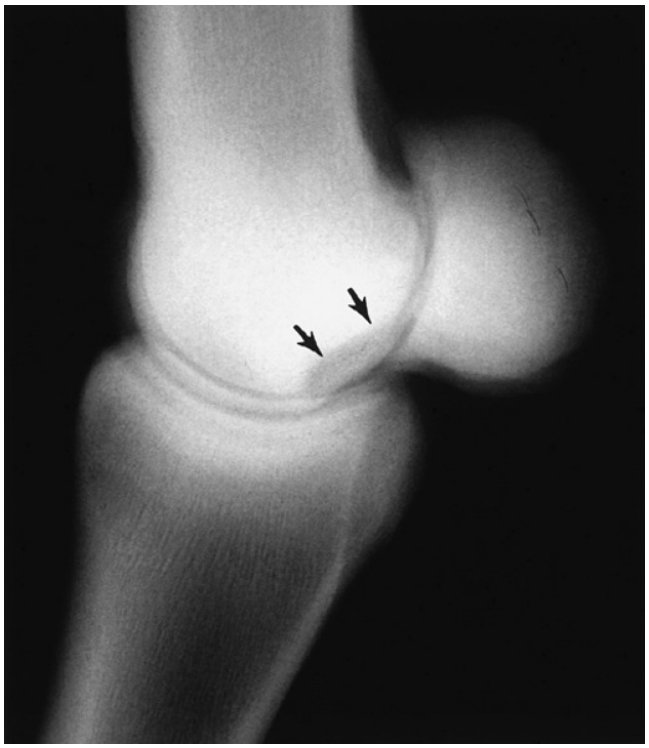


Figure 3.38. Lateromedial (LM) projection of the metacarpophalangeal joint showing palmar osteochondral disease of MCIII (arrows).

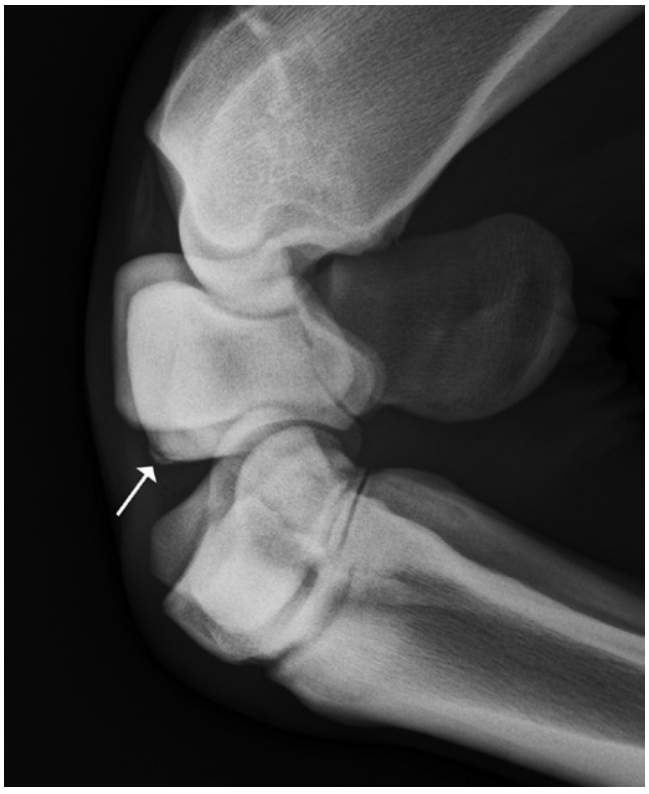


Figure 3.39. Flexed lateromedial (flexed LM) projection of the carpus. An area of lysis with a well-defined associated osseous body (chip fracture) is present on the dorsodistal periarticular margin of the radial carpal bone secondary to traumatic hyperextension injury (arrow). *Source*: Courtesy of New Bolton Center, University of Pennsylvania.

tissue injuries due to secondary bone change; however, there are marked limitations in the ability for radiography to evaluate soft tissue injures. In order to have the most accurate diagnosis and subsequently be able to provide the best care to the patient, visualization of architectural changes within the soft tissues is essential. Ideally, in these cases, other imaging modalities (i.e. ultrasound or MRI) should be considered to complement the radiographic findings.

Skeletal abnormalities are identified radiographically based on changes in bone density/opacity (reduced or increased) and shape. Because skeletal lesions may only be detected with radiographs after approximately 50% change in bone mineralization is present, some conditions such as acute osteomyelitis, early synovitis, early cartilage loss/erosive arthritis, and minimally or nondisplaced fractures may not be recognized on the initial radiographic exam. In cases of acute trauma, it may take up to 2 weeks before the bone changes are detected with plain radiographs. It is also possible to have relatively extensive articular cartilage damage with minimal radiographic changes. Therefore, when clinical signs and radiographic findings are incompatible, or radiographic findings are insufficient, further imaging with other modalities is often required.

In cases of comminuted fractures, it can be difficult to completely characterize the extent and orientation of the fracture radiographically. In these cases, computed tomography with multiplanar reconstructions should be considered for surgical planning.

In summary, the practitioner should be aware of the abovementioned limitations of radiography and that the combined results of different imaging modalities may be necessary to obtain a diagnosis. More important, it should always be remembered that radiographic changes do not necessarily represent lameness; therefore, a complete history and thorough physical and lameness examinations are imperative in every clinical situation.

NORMAL RADIOGRAPHIC ANATOMY

Recognition of normal radiographic anatomy and variations of normal in the mature and immature horse is essential in equine radiology to avoid erroneous diagnoses. The normal radiographic anatomy of horse extremities is presented for reference in the following pages (Figures 3.40–3.88). A diagram accompanies each of the radiographic projections, demonstrating the position of the X-ray machine and detector in relation to the anatomical site of interest, as well as the angle orientation of the X-ray beam. In addition, radiographic projections not commonly included in a standard examination are described. A brief explanation of the advantage of obtaining these projections is also included.

The nomenclature system used in this chapter is that proposed by the Nomenclature Committee of the American College of Veterinary Radiology,[40] which uses proper veterinary anatomic directional terms[26] and describes the direction in which the central X-ray beam penetrates the body part of interest, from the point of entrance to the point of exit (Figure 3.40). The standard abbreviation for the view is given in parentheses in the figure legends.

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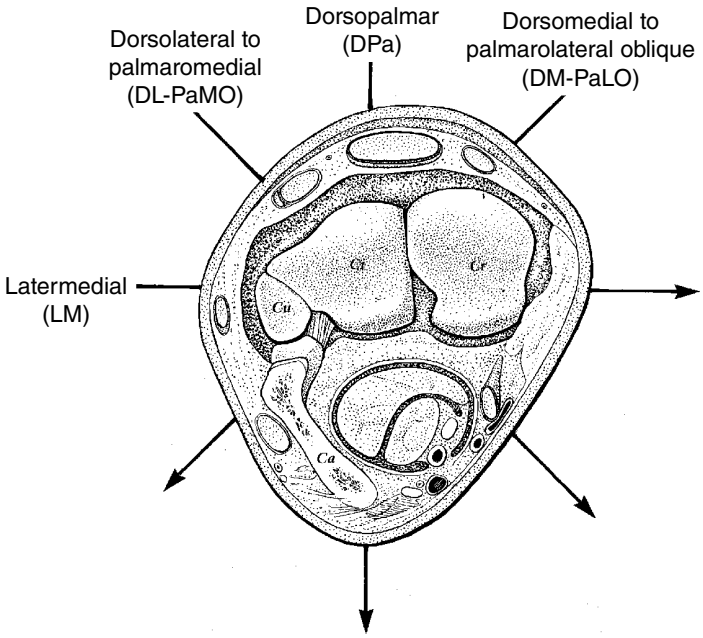
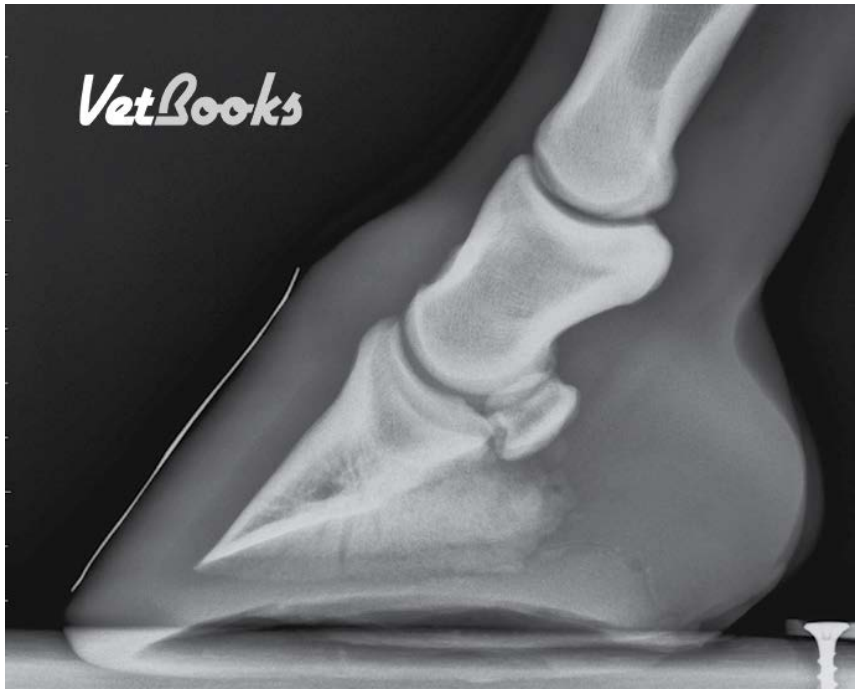


Figure 3.40. Cross-sectional diagram at the level of the proximal row of carpal bones demonstrating the nomenclature and method for labeling radiographic projections. Cr = radial carpal bone, Ci = intermediate carpal bone, Cu = ulnar carpal bone, and Ca = accessory carpal bone.



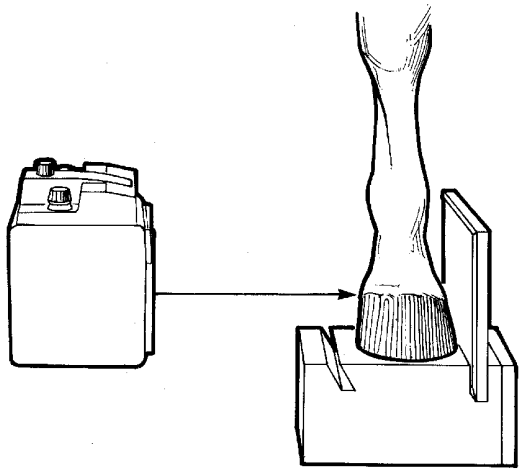


Figure 3.41.

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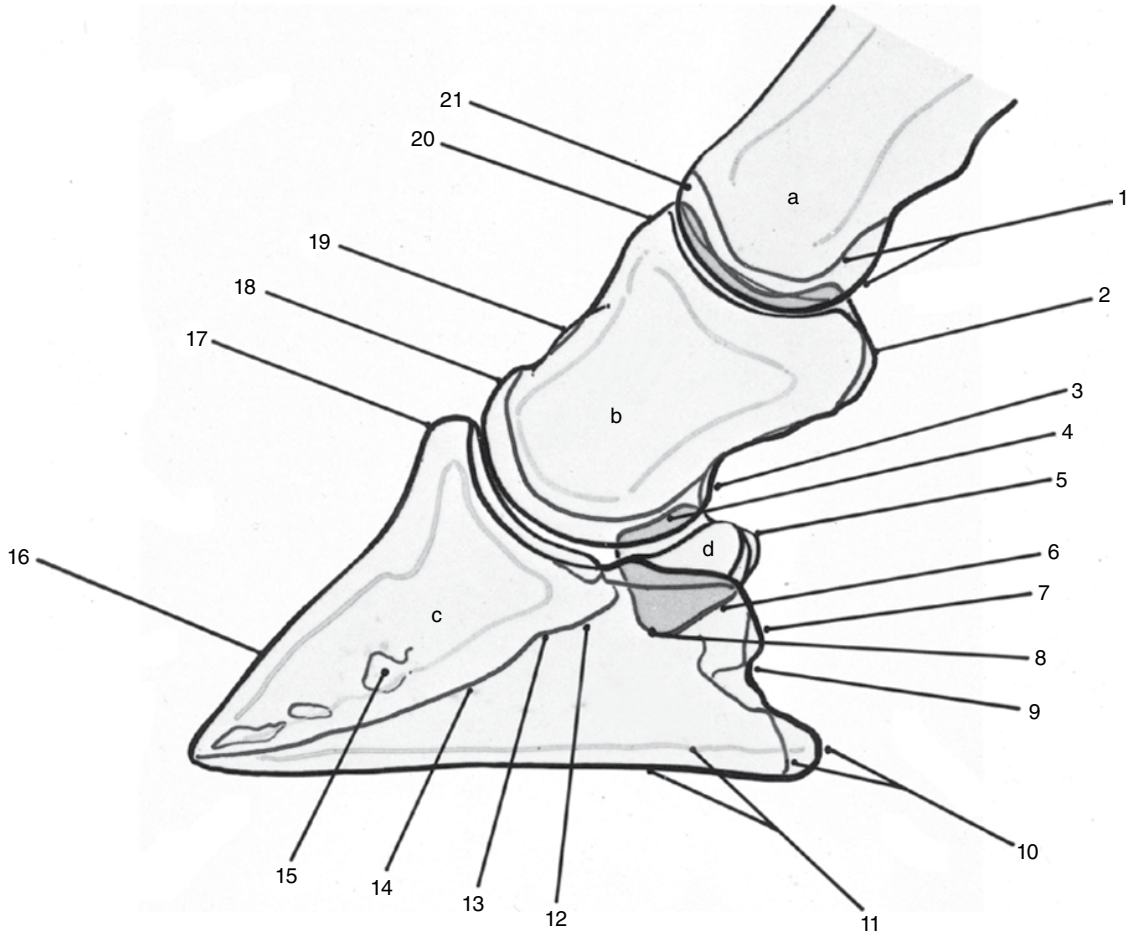


Figure 3.41. (*Continued*) Lateromedial (LM) projection of the distal phalanges and navicular bone. (a) Proximal phalanx, (b) middle phalanx (c) distal phalanx, and (d) navicular bone.

1. Palmar aspect of the medial and lateral condyles on the distal extremity of the proximal phalanx.
2. Transverse bony prominence on the proximopalmar aspect of the middle phalanx.
3. Superimposed medial and lateral condyles on the distopalmar aspect of the middle phalanx.
4. Articular surface of the navicular bone.
5. Proximal border of the navicular bone.
6. Flexor cortex and surface of the navicular bone; the medullary cavity (spongiosa) is the less opaque area in the center of the navicular bone.
7. Superimposed medial and lateral proximal parts of the palmar process on the distal phalanx. The size of this palmar process varies depending on the mineralization and ossification of the collateral cartilages. The palmar process also superimposes over the navicular bone, sometimes creating confusing opacities.
8. Distal border of the navicular bone, the border of which may appear as a distinct ridge or may blend with the contour of the navicular bone. Slight obliquity on the lateromedial projection alters the navicular bone's apparent shape.
9. Palmar process incisure.
10. Superimposed distal parts of the medial and lateral palmar processes on the distal phalanx.
11. Medial and lateral distal (solar) borders of the distal phalanx. On oblique projections, these borders may be separated farther.
12. Flexor surface of the distal phalanx, where the deep digital flexor attaches.
13. Semilunar line on the solar surface of the distal phalanx.
14. Opaque line representing the bone cortex on the concave solar surface of the distal phalanx.
15. Solar canal of the distal phalanx on end. This canal makes a semicircular loop in the distal phalanx, and its visibility depends on its size and the X-ray beam angle.
16. Dorsal surface of the distal phalanx.
17. Extensor process of the distal phalanx, which may have a single-humped, double-humped, or pointed appearance. The surface should always be smooth.
18. Dorsal extent of the distal articular surface on the middle phalanx. The slight projection on the articular margin should not be mistaken for an osteophyte.
19. Eminences for collateral ligament attachments from the distal interphalangeal joint. They may be prominent or small, but their surface should be smooth; they should not be mistaken for periosteal bone production.
20. Extensor process of the middle phalanx.
21. Distal dorsal articular surface of the proximal phalanx.

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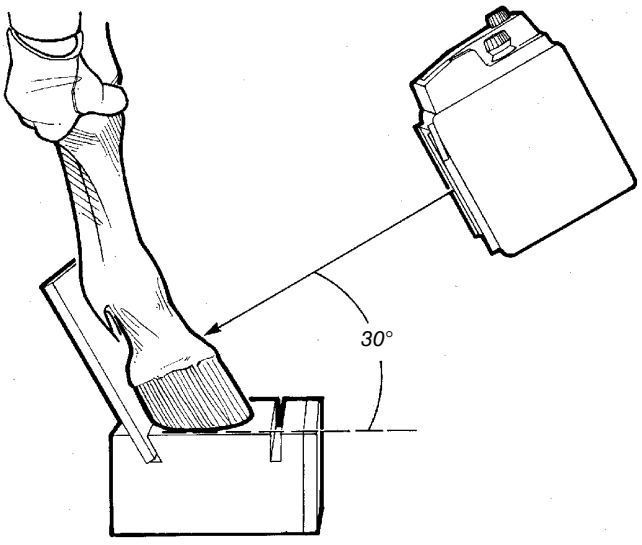


Figure 3.42.

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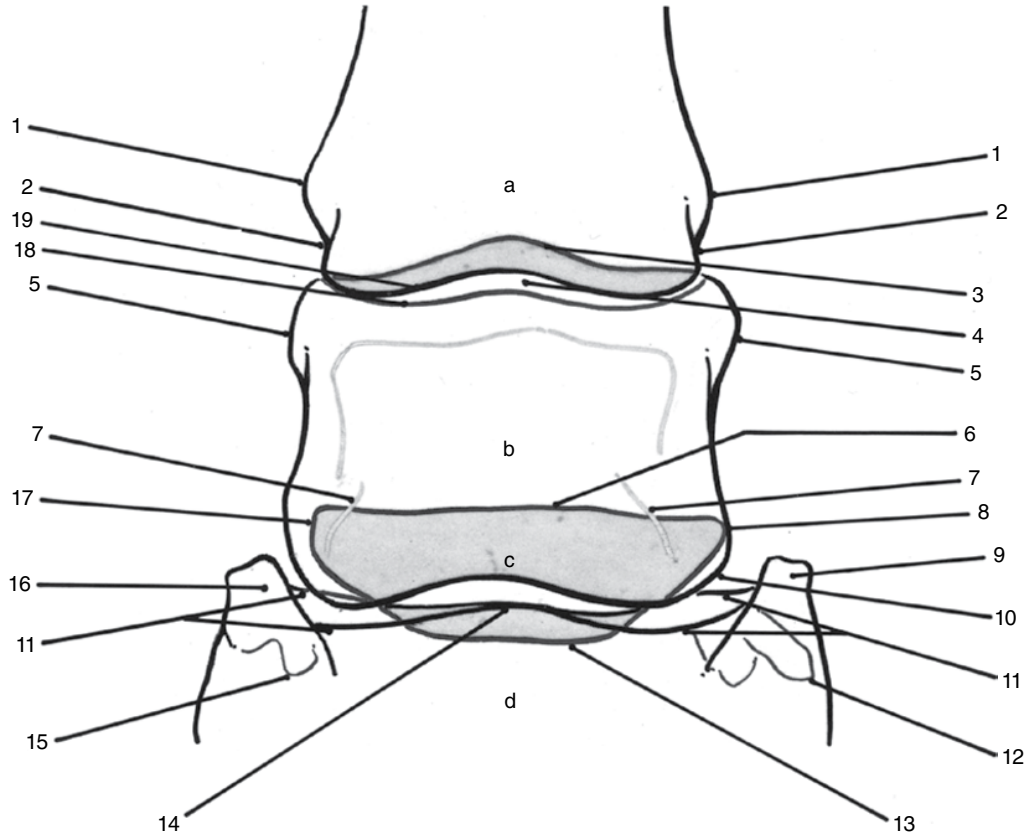


Figure 3.42. (*Continued*) Dorsoproximal to palmarodistal oblique (D30Pr-PaDiO) projection of the distal phalanges and navicular bone (foot). (a) Proximal phalanx, (b) middle phalanx, (c) navicular bone (distal sesamoid bone), (d) distal phalanx.

1. Medial and lateral eminences on the distal extremity of the proximal phalanx for attachment of collateral ligaments.
2. Medial and lateral depressions for attachment of collateral ligaments.
3. Proximal palmar border of the middle phalanx.
4. Articular cartilage thickness in the proximal interphalangeal joint space, which is approximately half the thickness of the articular cartilage in the distal interphalangeal joint.
5. Medial and lateral eminences for ligamentous and tendinous attachments on the proximal palmar aspect of the middle phalanx.
6. Proximal border of the navicular bone, which is seen with the least amount of distortion on this projection. It should be straight, smooth, and distinct.
7. Dorsal eminences on the medial and lateral aspects of the middle phalanx for attachment of the collateral ligaments of the distal interphalangeal joint.
8. Medial extremity of the navicular bone, which is slightly more rounded in appearance than the lateral extremity.
9. Proximal part of the medial palmar process on the distal phalanx.
10. Medial aspect of the distal articular surface on the middle phalanx.
11. Proximal articular surface of the distal phalanx; palmar and dorsal borders.
12. Fossa on the palmar medial surface of the distal phalanx.
13. Distal palmar border of the navicular bone, which cannot be adequately evaluated on this projection because of superimposition over the distal interphalangeal joint.
14. Extensor process for the distal phalanx.
15. Fossa on palmar lateral surface of the distal phalanx.
16. Proximal part of the lateral palmar process, the size of which depends on the extent of ossification in the cartilages of the distal phalanx. Separate ossification centers may occur in this region and should not be mistaken for fracture fragments.
17. Lateral extremity of the navicular bone, which has a sharper-angled appearance than the medial extremity.
18. Proximal articular surface (articular fovea) of the middle phalanx.
19. Distal articular surface of the proximal phalanx.

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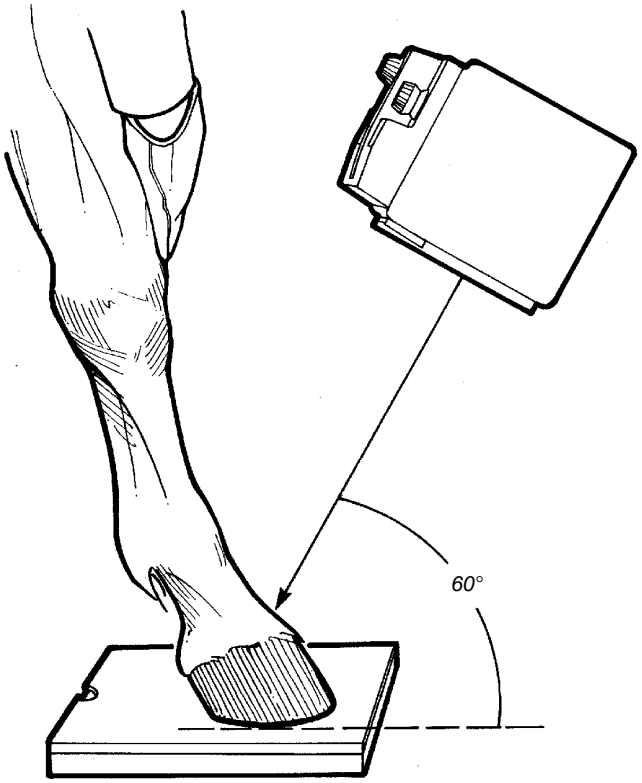


Figure 3.43.

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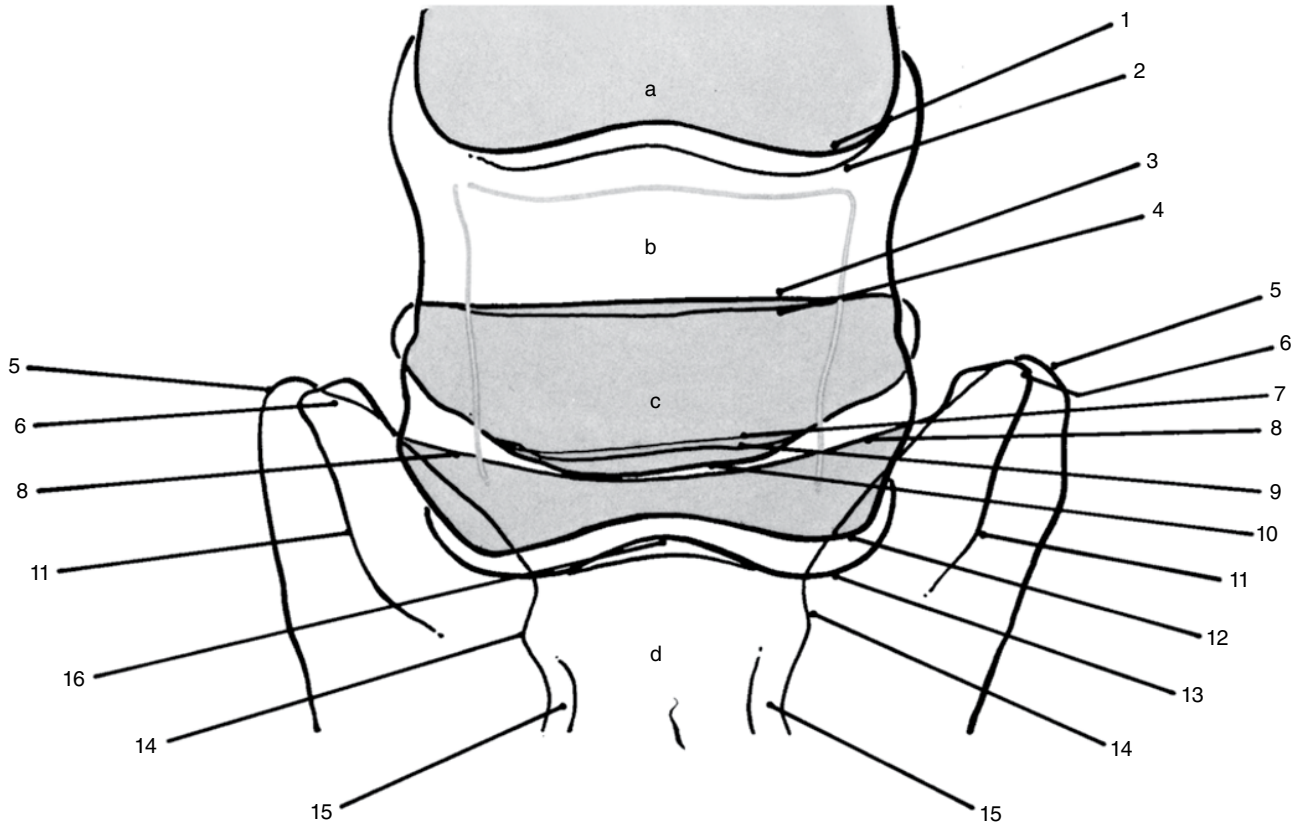


Figure 3.43. (*Continued*) Dorsoproximal to palmarodistal oblique (D60Pr-PaDiO) projection of the distal phalanx and navicular bone (foot). This projection is used extensively for evaluating the navicular bone. Position and exposure are critical for optimal visualization. (a) Proximal phalanx, (b) middle phalanx, (c) navicular bone (distal sesamoid bone), and (d) distal phalanx.

1. Distal articular surface of the proximal phalanx.
2. Proximal articular surface of the middle phalanx.
3. Proximal border of the flexor surface on the navicular bone.
4. Proximal border of the articular surface on the navicular bone, which often looks indistinct and slightly irregular on this projection because of the projection angle of the X-ray beam.
5. Distal part of the medial and lateral palmar processes.
6. Proximal part of the medial and lateral palmar processes.
7. Groove on the distal navicular bone between the flexor and articular margins. Vascular foramina are in this groove.
8. Palmar articular margin of the distal phalanx.
9. Distal margin on the navicular bone.
10. Distal margin of the flexor surface on the navicular bone.
11. Medial and lateral parietal sulci on the distal phalanx.
12. Distal articular surface of the middle phalanx.
13. Articular border of the distal phalanx.
14. Medial and lateral solar grooves on the solar surface of the distal phalanx.
15. Solar canal.
16. Extensor process of the distal phalanx.

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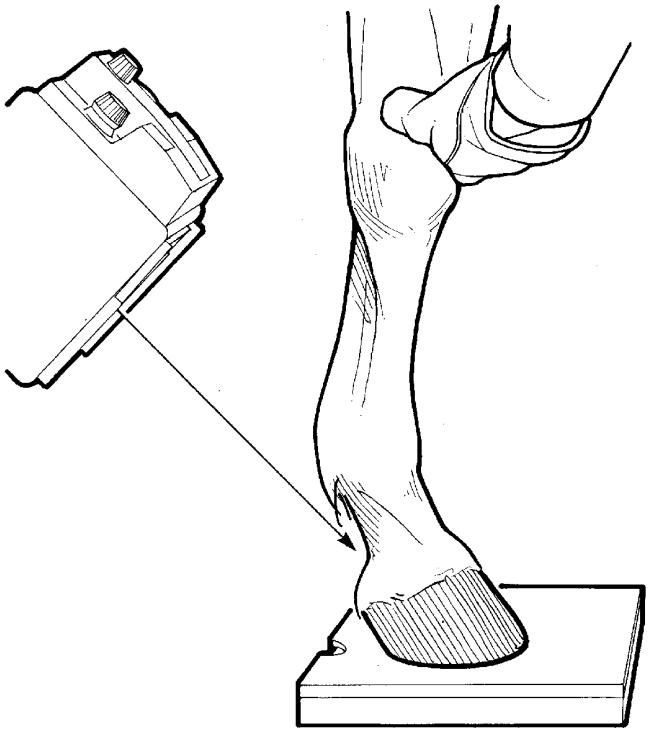


Figure 3.44.

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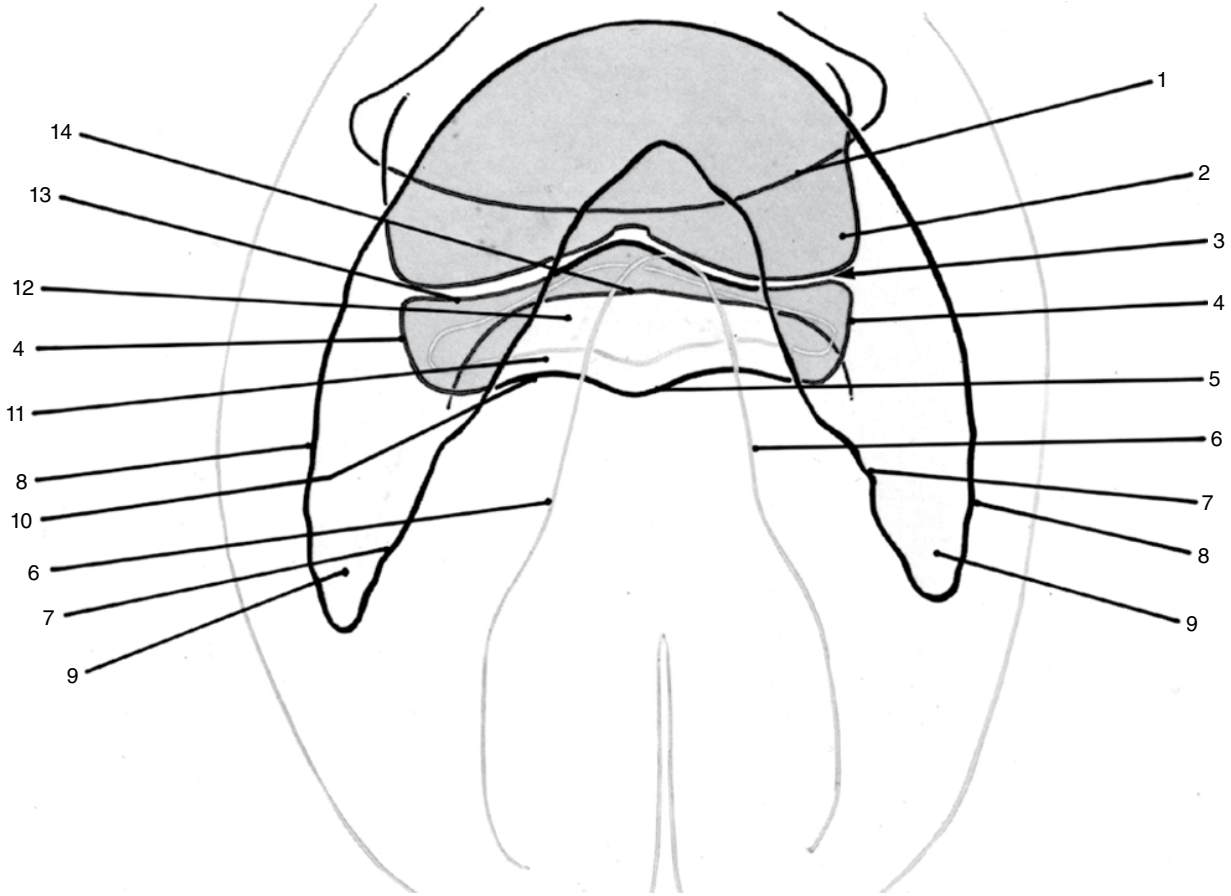


Figure 3.44. (*Continued*) Palmaroproximal to palmarodistal oblique (Pa45Pr-PaDiO) projection of the distal phalanx and navicular bone (foot). This projection shows the navicular bone with minimal superimposition over other bones. The angle of the X-ray beam and exposures are critical for eliminating projection artifacts, e.g. lack of good cortical and medullary cavity definition of superimposition of the distal phalanx over the navicular bone.

1. Palmar border of the middle phalanx.
2. Distal medial condyle of the middle phalanx.
3. Articulation between the navicular bone and the middle phalanx.
4. Medial and lateral extremities of the navicular bone.
5. Central eminence on the flexor surface of the navicular bone.
6. Collateral (paracureal) sulci of the frog.
7. Semilunar line on the solar surface of the distal phalanx.
8. Medial and lateral aspects of the solar border of the distal phalanx.
9. Distal part of the medial and lateral palmar processe
10. Flexor surface of the navicular bone.
11. Flexor cortex of the navicular bone.
12. Medullary (spongiosa) cavity of the navicular bone.
13. Articular surface of the navicular bone.

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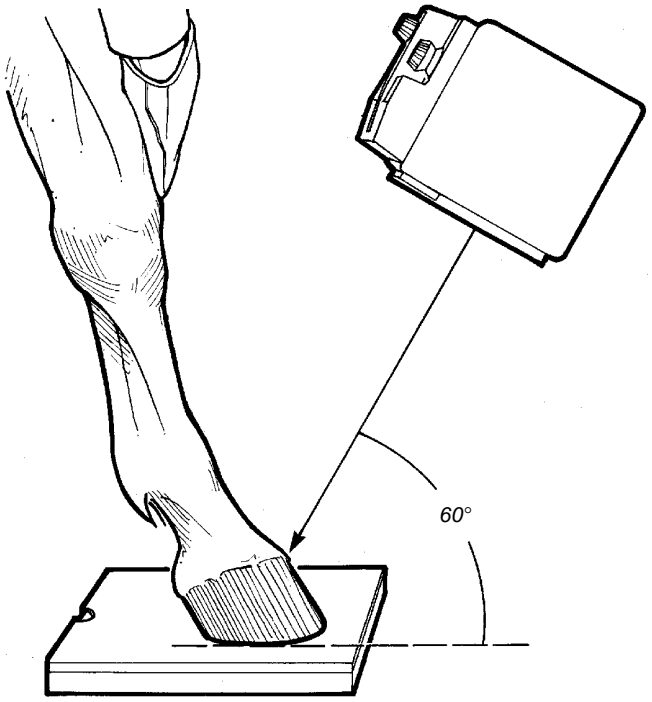


Figure 3.45.

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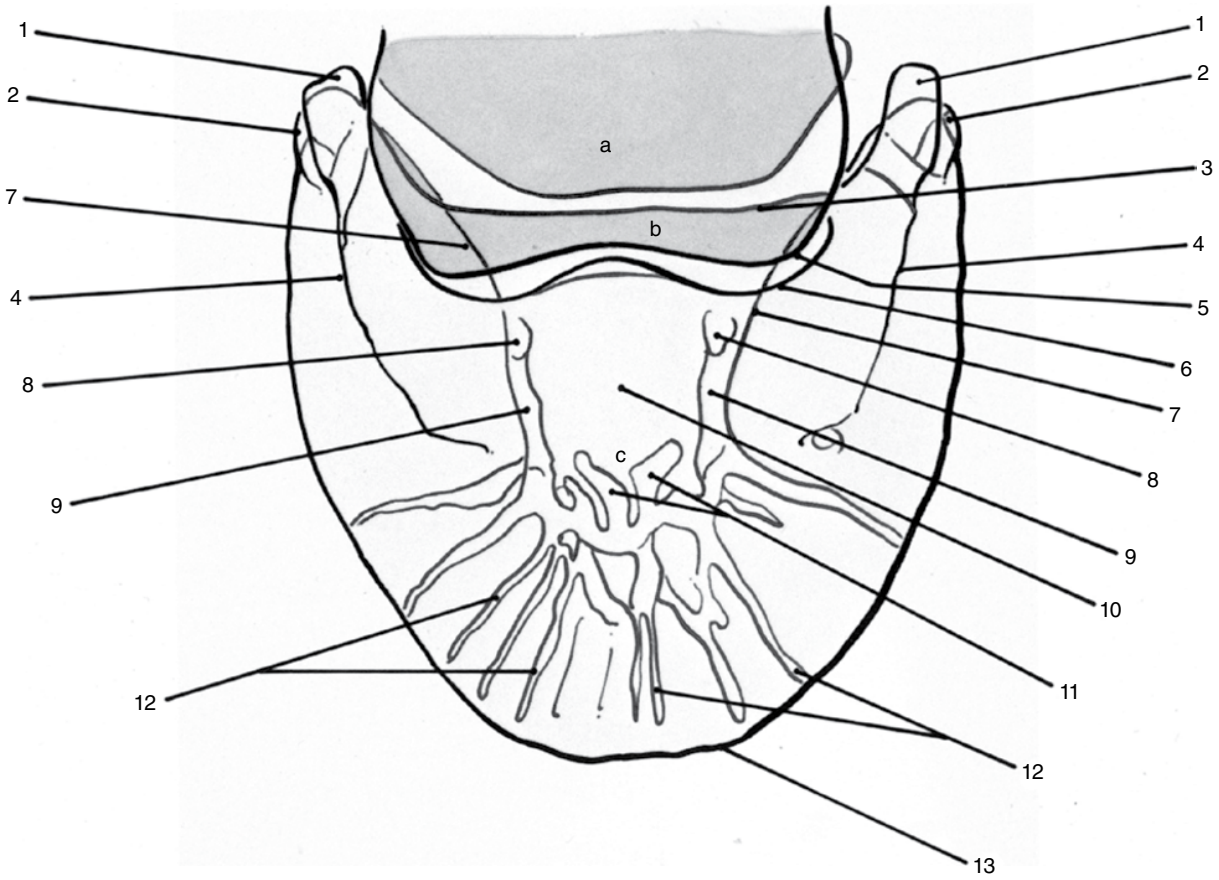


Figure 3.45. (*Continued*) Dorsoproximal to palmarodistal oblique (D60Pr-PaDiO) projection of the distal phalanx (foot). The X-ray beam for this projection is centered at the coronet, and the exposure is half that necessary for visualizing the navicular bone. (a) Navicular bone (distal sesamoidean bone), (b) middle phalanx, and (c) distal phalanx.

1. Proximal part of the medial and lateral palmar processes.
2. Distal part of the medial and lateral palmar processes.
3. Palmar articular margin of the distal phalanx.
4. Medial and lateral parietal sulci on the distal phalanx.
5. Distal articular surface of the middle phalanx.
6. Proximal dorsal margin of the articular surface of the distal phalanx.
7. Borders of the medial and lateral solar grooves.
8. Medial and lateral solar foramina.
9. Solar canal, the width and distinctness of which are variable in normal distal phalanges.
10. Flexor surface of the distal phalanx, where the deep digital flexor tendon attaches.
11. Vascular canals in the region of the solar canal.
12. Peripheral vascular canals. The vascular canals may be of variable width in normal distal phalanges. The peripheral solar border of the distal phalanx should be relatively smooth and symmetrical, although a slight irregular peripheral border may be considered normal in older animals.
13. Distal solar margin of the distal phalanx. The normal distal border of the distal phalanx may be convex or have some degree of concavity. A mid-distal notch, when present, is the crena marginis solaris.

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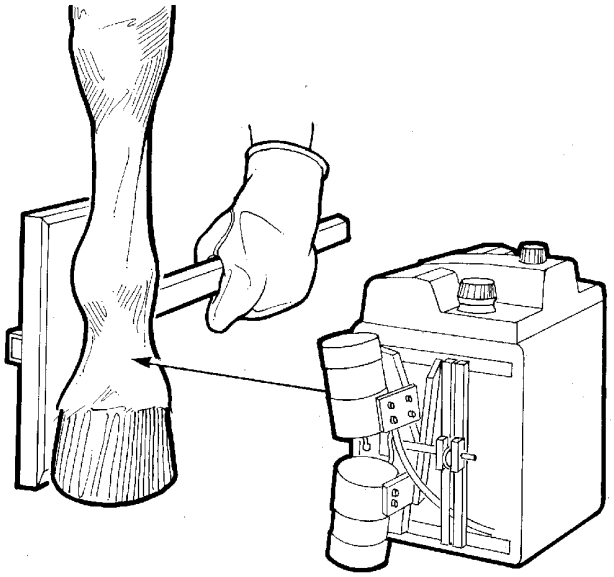


Figure 3.46.

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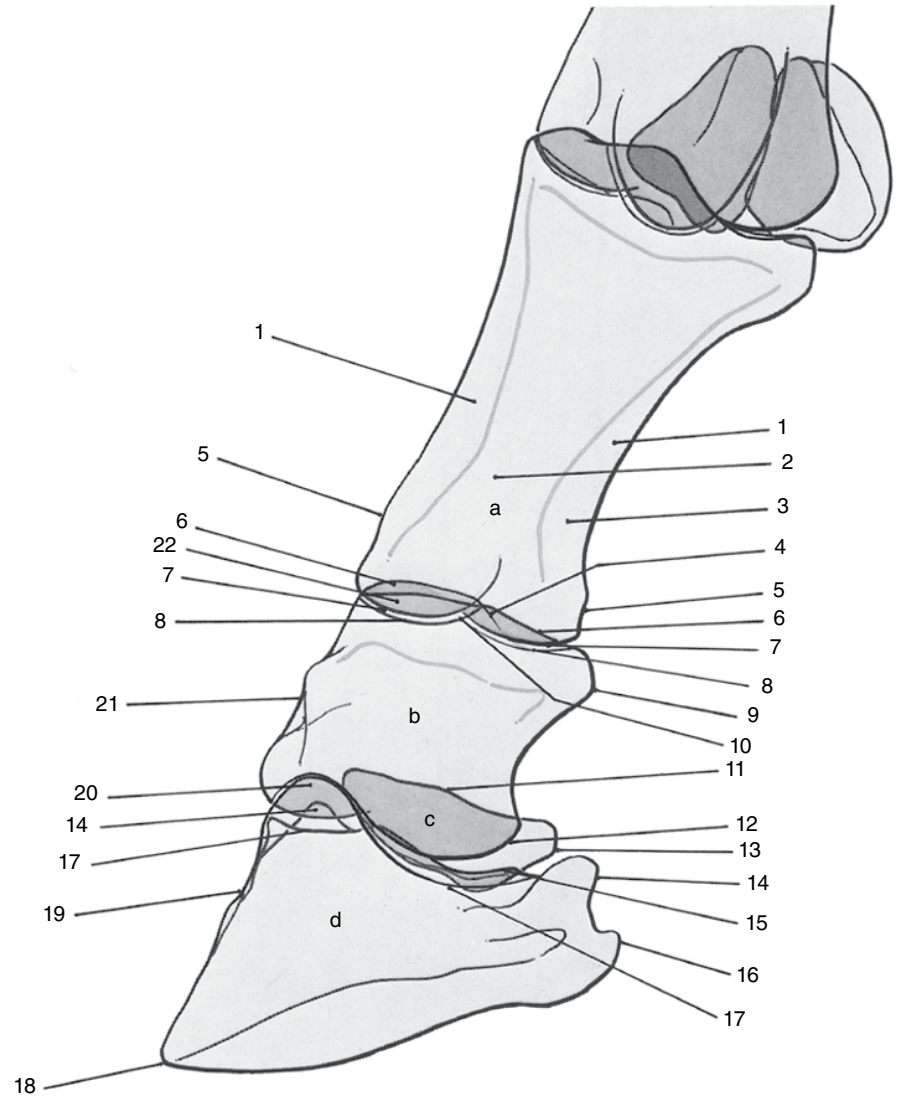


Figure 3.46. (*Continued*) Dorsomedial to palmarolateral oblique (D35M-PaLO) projection of the phalanges. (a) Proximal phalanx, (b) middle phalanx, (c) navicular bone, and (d) distal phalanx.

1. Mediopalmar and dorsolateral cortices of the proximal phalanx.
2. Medullary cavity of the proximal phalanx, which can sometimes be distinctly seen as a 2- to 3-cm lucency in the center of the proximal phalanx.
3. Surface for attachment of the middle (oblique) distal sesamoidean ligament.
4. Sagittal ridge on the proximal articular surface of the middle phalanx.
5. Eminences for attachment of the medial and lateral collateral ligaments of the proximal interphalangeal joint on the distal aspect of the proximal phalanx.
6. Palmar border of the articular fovea on the base of the middle phalanx.
7. Medial and lateral condyles of the distal aspect of the proximal phalanx.
8. Articular fovea on the base of the middle phalanx.
9. Medial proximal eminence for attachment of the medial collateral ligament and the medial branch of the tendon of the superficial digital flexor on the middle phalanx.
10. Sagittal ridge between the fovea on the base of the middle phalanx.
11. Proximal border of the navicular bone.
12. Distal medial condyle of the middle phalanx.
13. Medial extremity of navicular bone.
14. Proximal part of the medial palmar process of the distal phalanx.
15. Palmar part of the medial palmar process of the distal phalanx.
16. Distal part of the medial palmar process of the distal phalanx.
17. Medial and lateral aspects of the coronary border of the articular surface on the distal phalanx.
18. Solar border of the distal phalanx.
19. Depression and bony prominence on the lateral parietal surface of the distal phalanx for lateral collateral ligament attachment. The bony prominence has a smooth surface and should not be mistaken for bone production.
20. Extensor process of the distal phalanx.
21. Eminence on the dorsal surface of the middle phalanx for collateral ligament attachment, which has a smooth surface and should not be mistaken for periosteal bone production.
22. Dorsolateral articular border on the middle phalanx.

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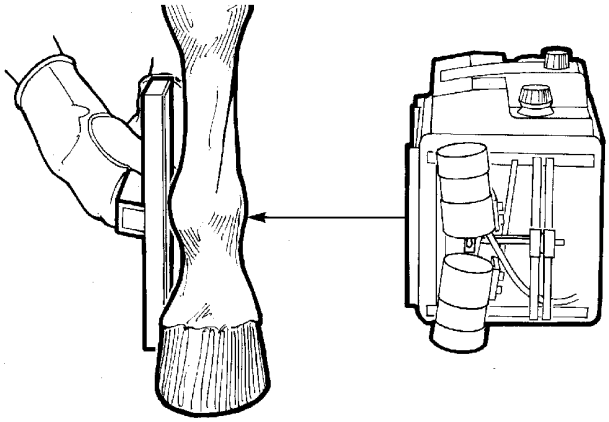


Figure 3.47.

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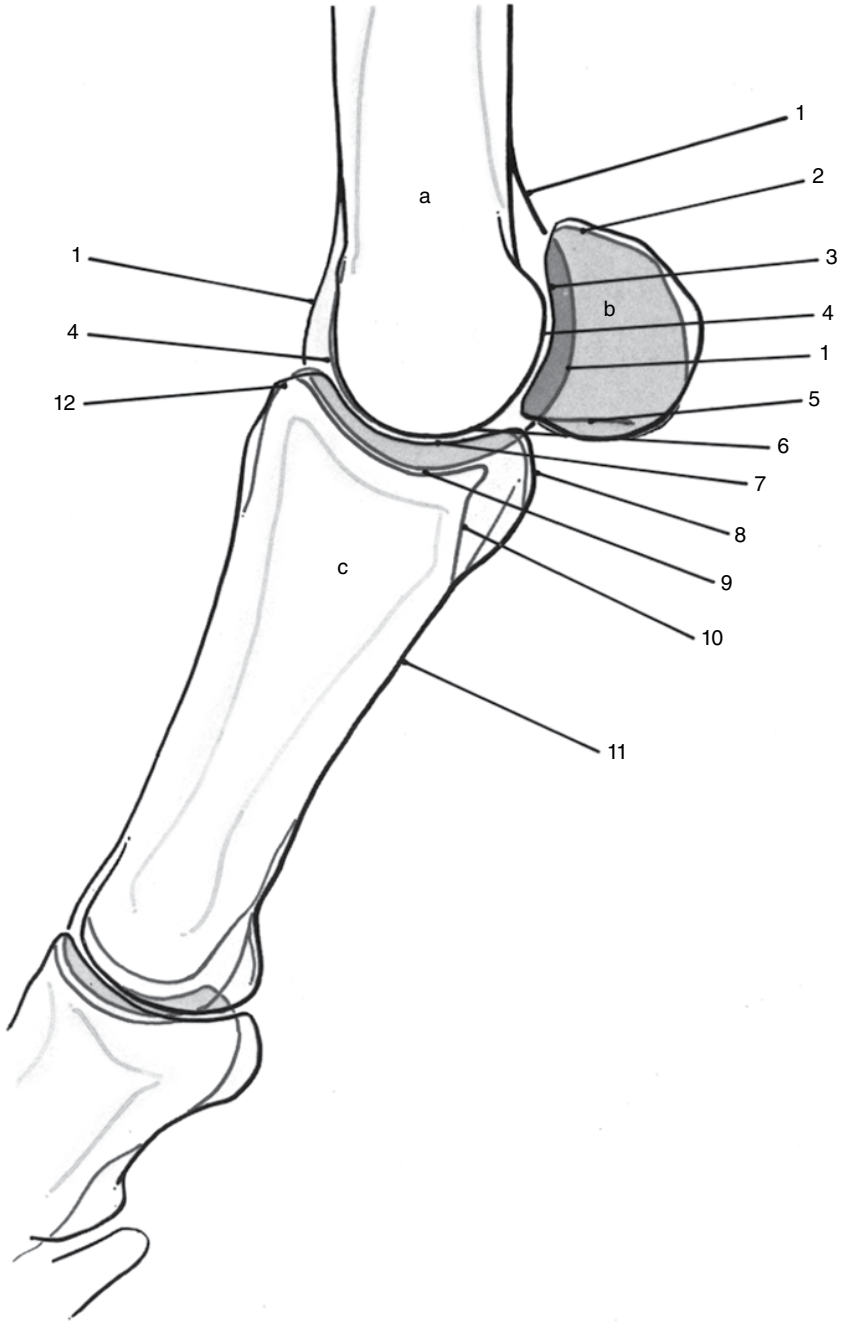


Figure 3.47. (*Continued*) Lateromedial (LM) projection of the metacarpophalangeal joint. (a) Third metacarpal bone, (b) proximal sesamoid bone, and (c) proximal phalanx.

1. Sagittal ridge on the distal extremity of the third metacarpal bone.
2. Apex of the superimposed proximal sesamoid bones.
3. Articular surface of the proximal sesamoid bones.
4. Articular surface of the superimposed medial and lateral condyles of the third metacarpal bone.
5. Superimposed base of the proximal sesamoid bones.
6. Transverse ridge on the distal articular surface of the third metacarpal bone, which divides the distal metacarpal articular surface into dorsal and palmar areas. It varies in size and visibility.
7. Superimposed medial and lateral articular fovea of the proximal phalanx.
8. Superimposed medial and lateral tuberosities on the proximal palmar aspect of the proximal phalanx for ligamentous attachment.
9. Sagittal groove on the proximal articular surface of the proximal phalanx, which articulates with the sagittal ridge of the third metacarpal bone.
10. Midpalmar surface of the proximal phalanx located between the lateral and medial tuberosities.
11. Palmar surface of the proximal phalanx, where the middle (oblique) distal sesamoidean ligament attaches.
12. Superimposed medial and lateral bony eminences for attachment of the extensor tendon on the proximal phalanx.

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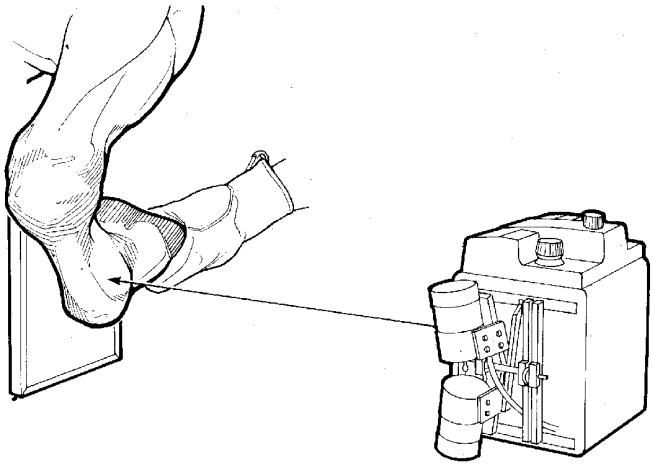


Figure 3.48.

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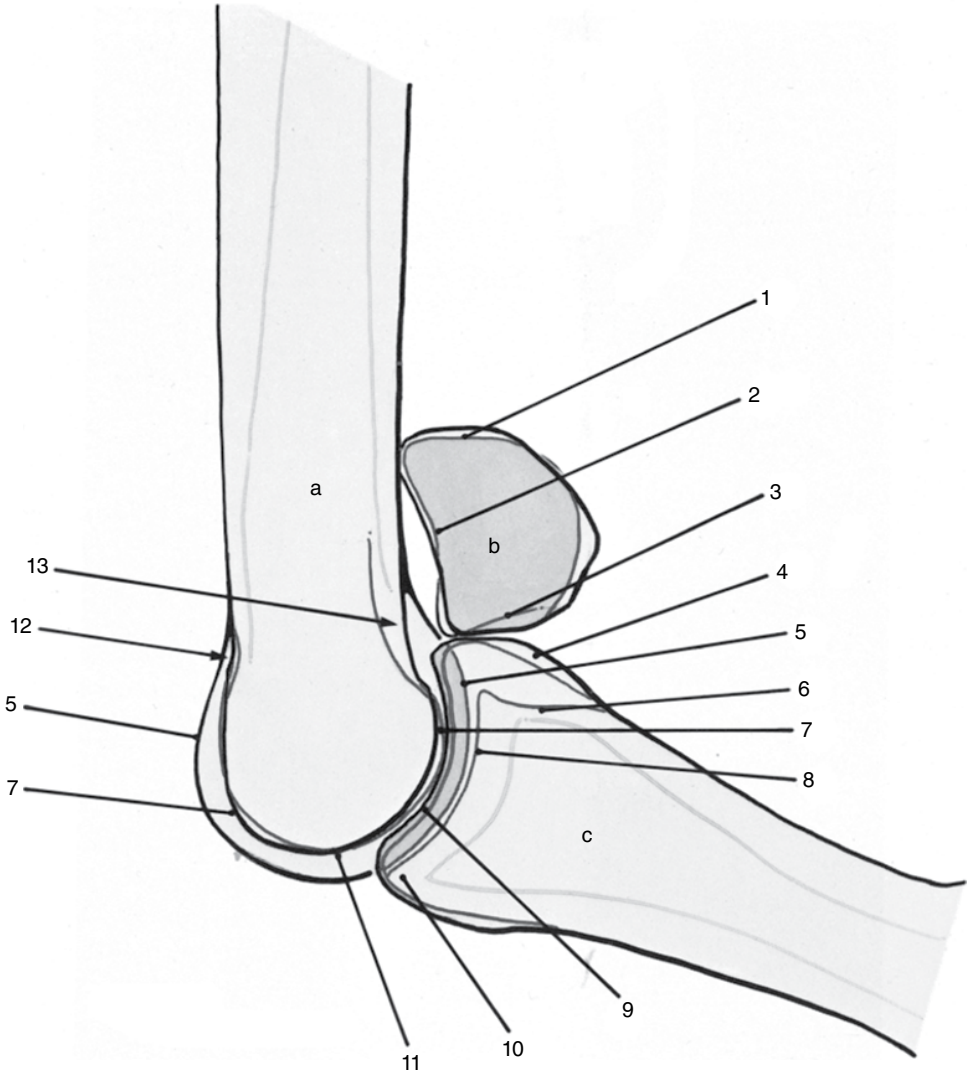
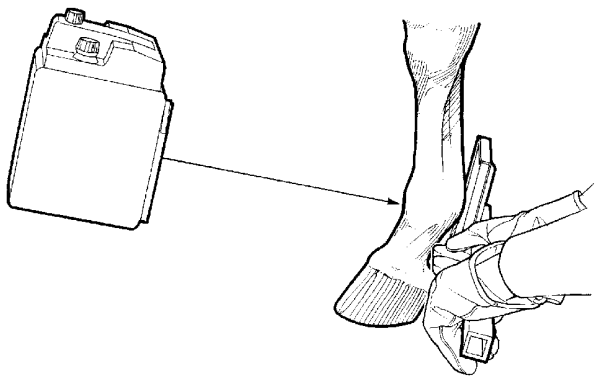


Figure 3.48. (*Continued*) Flexed lateromedial (flexed LM) projection of the metacarpophalangeal joint. This projection allows the most effective evaluation of the articular surface of the sesamoid bones, offers good visualization of small articular basilar sesamoid fractures and changes, and provides a distinct projection of the proximal tuberosities. Furthermore, the dorsal articular surface of the third metacarpal bone can be evaluated without superimposition of the proximal phalanx. (a) Third metacarpal bone, (b) proximal sesamoid bones, and (c) proximal phalanx.

1. Apex of the superimposed proximal sesamoid bones.
2. Superimposed articular surfaces of the proximal sesamoid bones.
3. Bases of the superimposed proximal sesamoid bones.
4. Superimposed medial and lateral tuberosities on the proximal caudal aspect of the proximal phalanx, where ligamentous attachments occur.
5. Sagittal ridge on the distal articular surface of the third metacarpal bone; the dorsal and palmar aspects are labeled.
6. Midpalmar surface of the proximal phalanx, located between the medial and lateral tuberosities.
7. Palmar and dorsal aspects of the superimposed medial and lateral condyles of the third metacarpal bone.
8. Sagittal groove on the proximal articular surface of the proximal phalanx, which articulates with the sagittal ridge on the distal third metacarpal bone.
9. Superimposed fovea (articular surface) of the proximal extremity of the proximal phalanx.
10. Superimposed medial and lateral parts of the extensor eminence of the proximal phalanx.
11. Transverse ridge on the distal articular surface of the third metacarpal bone, which separates the distal articular surface of the third metacarpal bone into dorsal and palmar parts.
12. Bony depression where the proximal dorsal recess from the metacarpophalangeal joint is located.
13. Bony depression where the proximal palmar recess from the metacarpophalangeal joint is located.

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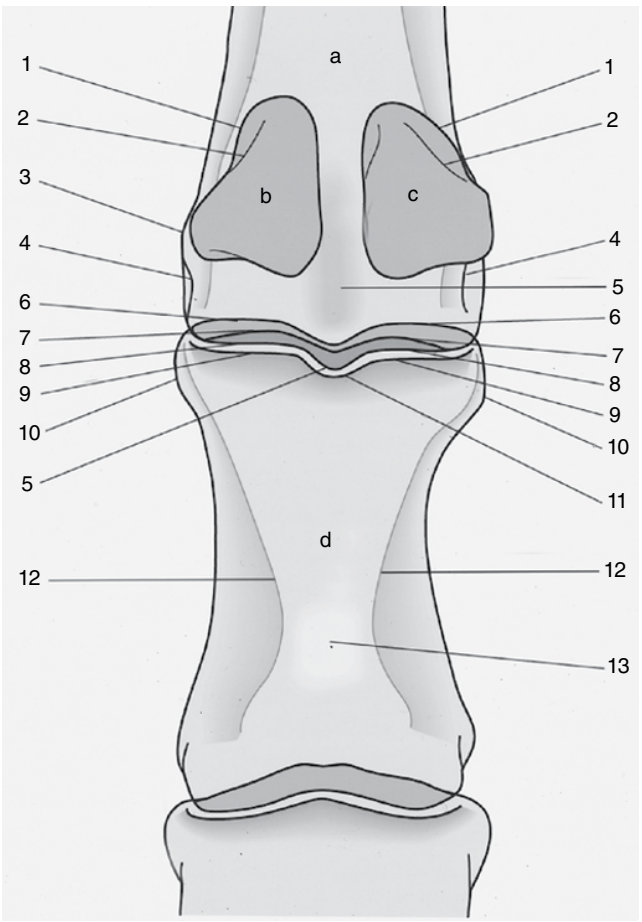


Figure 3.49. Dorsoproximal to palmarodistal (D30Pr-PaDi) projection of the metacarpophalangeal joint. (a) Third metacarpal bone, (b) lateral proximal sesamoid bone, (c) medial proximal sesamoid bone, and (d) proximal phalanx.

1. Peripheral abaxial margins of the proximal sesamoid bones. The peripheral margin of the medial proximal sesamoid bone is more convex than that of the lateral proximal sesamoid bone.
2. Abaxial margin of the surfaces of the medial and lateral proximal sesamoid bone.
3. Eminence for attachment of the lateral collateral ligament.
4. Depression on the medial and lateral aspects of the third metacarpal bone where collateral ligaments attach.
5. Sagittal ridge on the distal articular surface of the third metacarpal bone.
6. Medial and lateral palmar margins of the articular fovea on the proximal extremity of the proximal phalanx.
7. Medial and lateral dorsal margins of the articular fovea on the proximal extremity of the proximal phalanx.
8. Medial and lateral condyles (articular surface) of the third metacarpal bone.
9. Articular fovea on the proximal extremity of the proximal phalanx.
10. Medial and lateral palmar tuberosities on the proximal extremity of the proximal phalanx for ligament attachment.
11. Sagittal groove on the proximal articular surface of the proximal phalanx.
12. Bony ridges on the palmar surface of the proximal phalanx for attachment of the middle (oblique) distal sesamoidean ligament.
13. Medullary cavity in the proximal phalanx.

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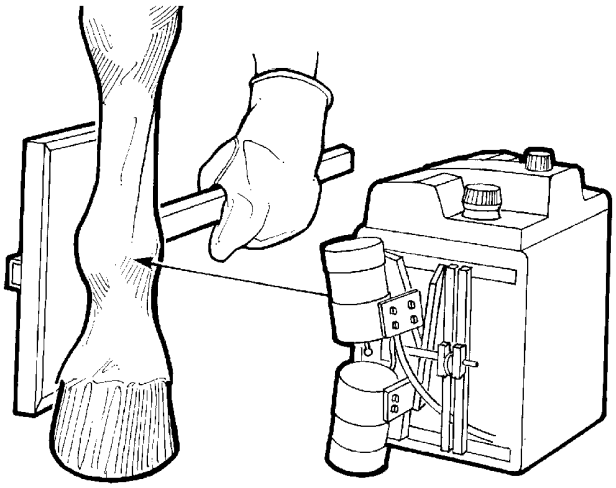


Figure 3.50.

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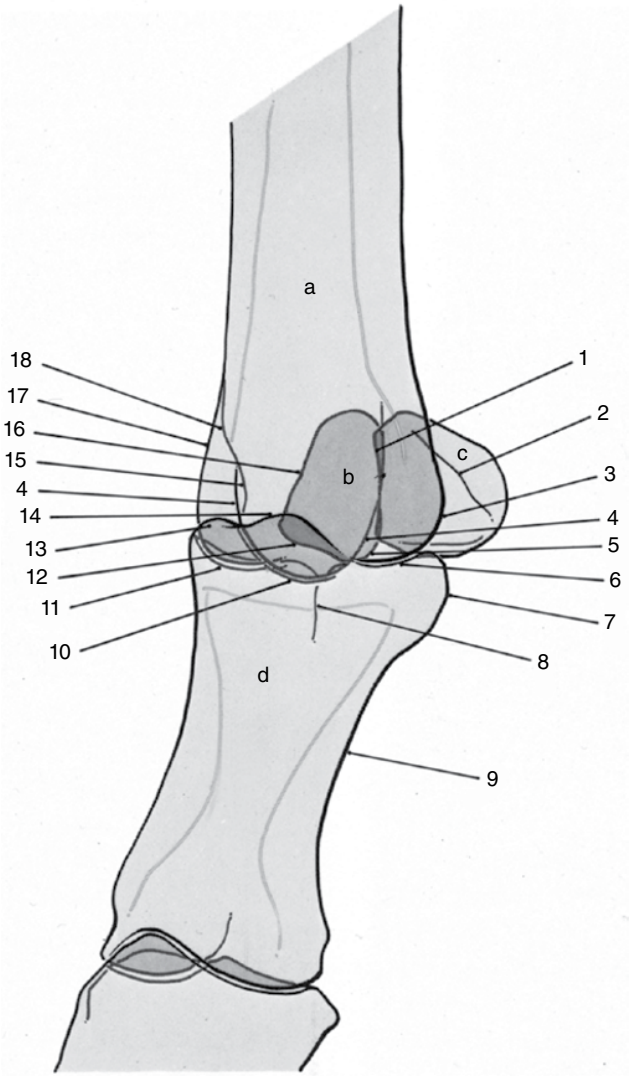


Figure 3.50. (*Continued*) Dorsolateral to palmaromedial oblique (D45L-PaMO) projection of the metacarpophalangeal joint. (a) Third metacarpal bone, (b) medial proximal sesamoid bone, (c) lateral proximal sesamoid bone, and (d) proximal phalanx.

1. Articular surface of the lateral proximal sesamoid bone.
2. The difference in radiographic opacity is caused by a difference in bone thickness on the lateral proximal sesamoid bone. The base and body are more opaque than the apex and peripheral border. The convex shape on the abaxial surface produces the distinct line between the two opacities.
3. Palmar surface of the lateral condyle distal aspect of the third metacarpal bone.
4. Palmar and dorsal aspects of the sagittal ridge on the distal third metacarpal bone.
5. Junction of the peripheral and basilar margins of the medial proximal sesamoid bone.
6. Lateral articular fovea of the proximal extremity of the proximal phalanx.
7. Lateral tuberosity on the proximal palmar aspect of the proximal phalanx.
8. Medial tuberosity on the proximal palmar aspect of the proximal phalanx.
9. Lateral bony ridge for attachment of the middle (oblique) distal sesamoidean ligament.
10. Sagittal groove on the proximal articular surface of the proximal phalanx.
11. Medial articular fovea of the proximal extremity of the proximal phalanx.
12. Basilar margin of the medial proximal sesamoid bone.
13. Medial dorsal margin of the articular fovea on the proximal phalanx.
14. Lateral dorsal margin of the articular fovea on the proximal phalanx. Both margins (13, 14) are visible on correctly exposed and positioned oblique projections of the metacarpophalangeal joint.
15. Depression (concave surface) for attachment of the medial collateral ligament on the distal third metacarpal bone. The visibility and distinctness of the concave line change on different projections. It may be more prominent on some examinations and not visible on others.
16. Abaxial articular margin of the medial proximal sesamoid bone.
17. Dorsal aspect of the medial condyle on the third metacarpal bone.
18. Eminence on the third metacarpal bone for attachment of the medial collateral ligament.

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Figure 3.51.

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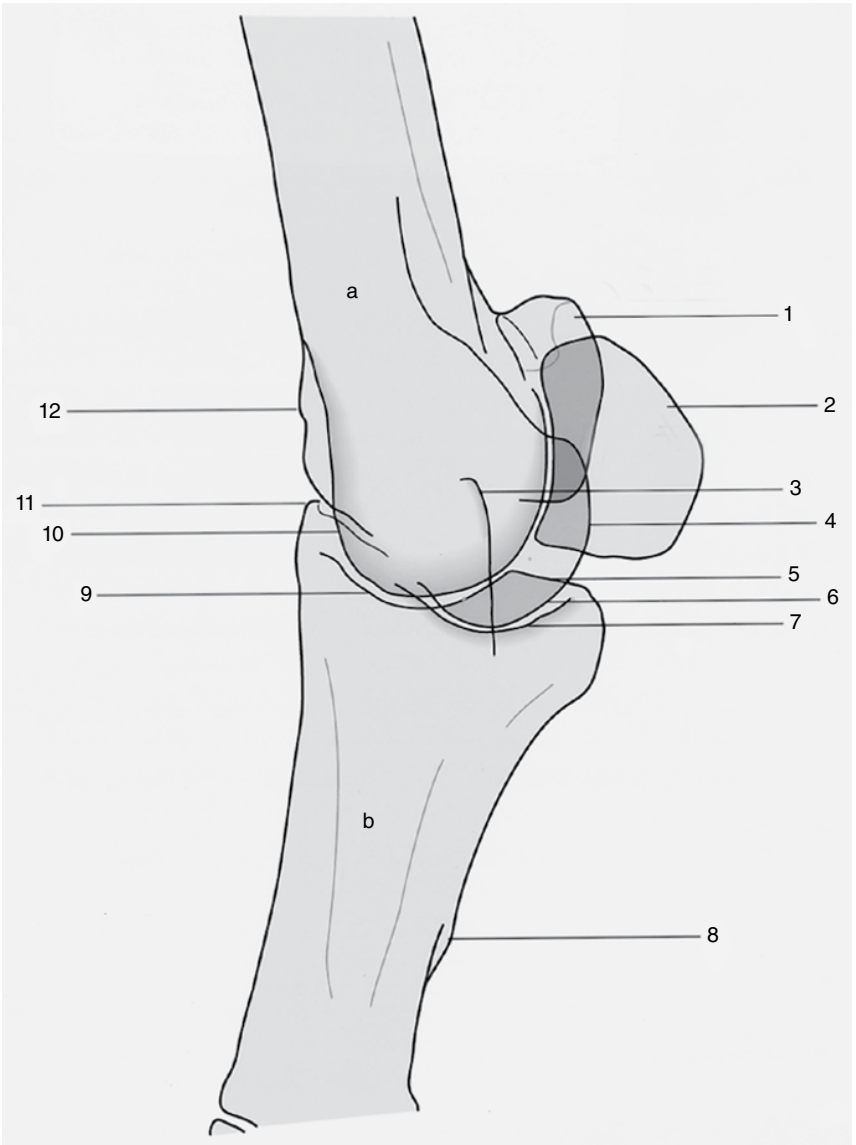


Figure 3.51. (*Continued*) Lateroproximodorsalmediodistopalmar oblique (L20Pr20D-MDiPaO) projection of the metacarpophalangeal joint. (a) Third metacarpal bone and (b) proximal phalanx.

1. Medial proximal sesamoid bone.
2. Peripheral margin of the lateral proximal sesamoid bone.
3. Medial proximal palmar border of the proximal phalanx.
4. Lateral condyle of the third metacarpal bone.
5. Lateral palmar eminence of the proximal phalanx.
6. Lateral condyle (articular surface) of the third metacarpal bone.
7. Lateral articular fovea of the proximal phalanx.
8. Bony ridge for attachment of the middle (oblique) distal sesamoidean ligament.
9. Sagittal groove on the proximal articular surface of the proximal phalanx.
10. Distal surface of the sagittal ridge of the third metacarpal bone.
11. Medial articular margin of the articular fovea on the proximal phalanx.
12. Dorsal border of the medial condyle of the third metacarpal bone.

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Figure 3.52.

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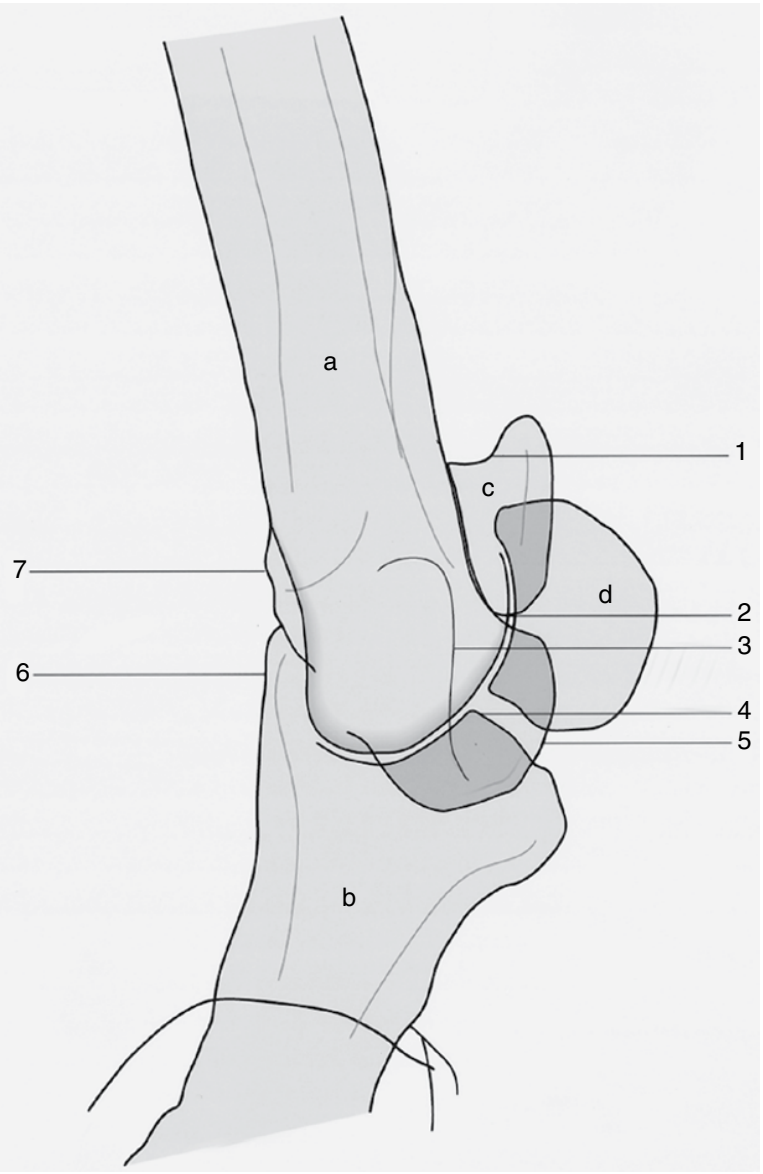


Figure 3.52. (*Continued*) Lateroproximal to distomedial oblique (L45P-DiMO) projection of the metacarpophalangeal joint. (a) Third metacarpal bone, (b) proximal phalanx, (c) medial proximal sesamoid bone, and (d) lateral proximal sesamoid bone.

1. Abaxial surface of the medial proximal sesamoid bone.
2. Palmar surface of the sagittal ridge of the third metacarpal bone.
3. Medial proximal border of the proximal phalanx.
4. Lateral palmar eminence of the proximal phalanx.
5. Lateral condyle of the third metacarpal bone.
6. Proximal medial surface of the proximal phalanx.
7. Dorsal border of the medial condyle of the third metacarpal bone.

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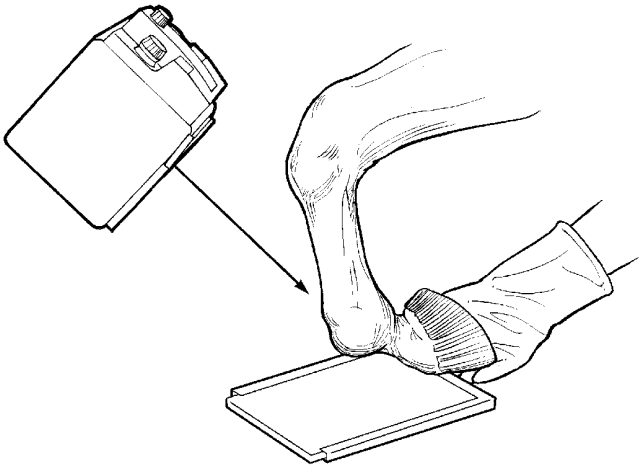


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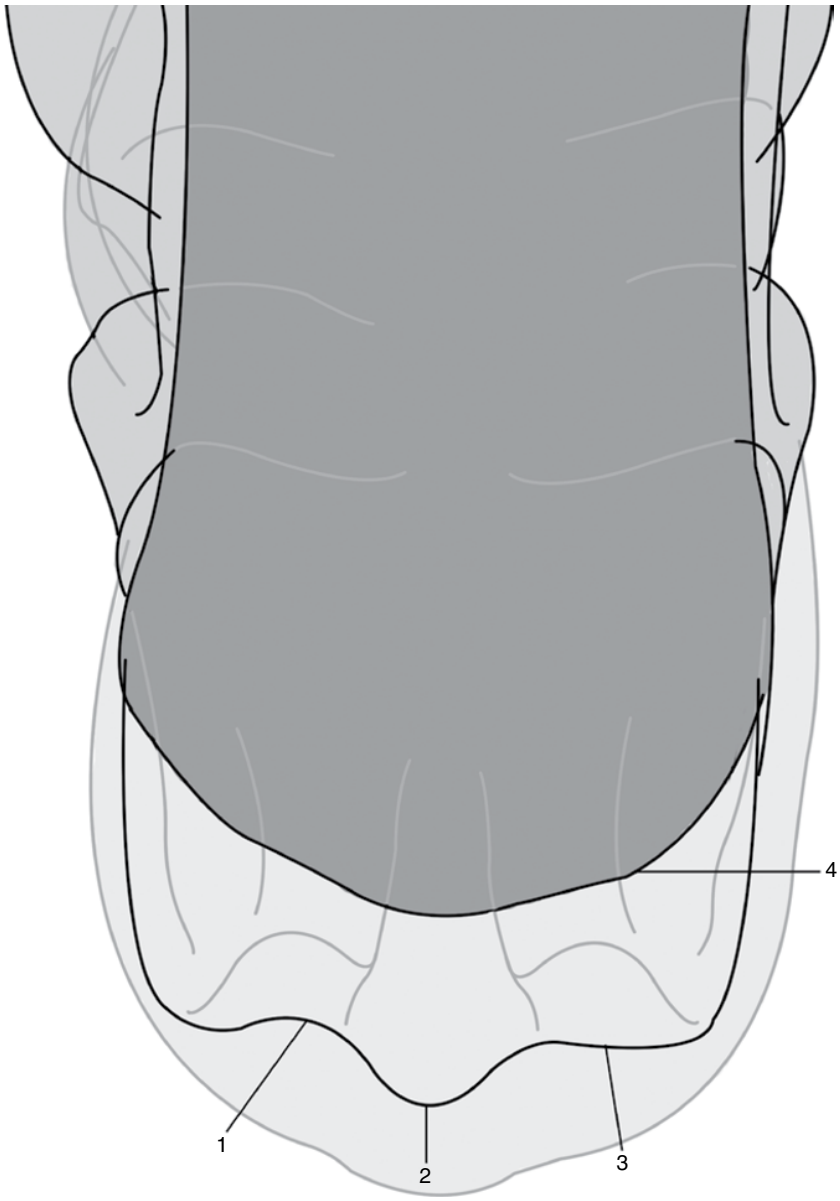
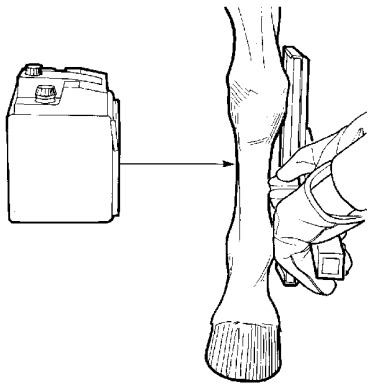


Figure 3.53. (*Continued*) Dorsoproximal to dorsodistal oblique (D45–70Pr-DDiO) projection of the metacarpophalangeal joint. In this projection, the flexed fetlock joint is held on top of the detector. The primary X-ray beam is directed distally in a 45–70° dorsoproximal dorsodistal direction and is centered on the dorsal aspect of the midsagittal ridge of metacarpus/metatarsus for evaluation of the dorsal articular surface of the condyle of MCIII/MTIII.

1. Articular surface of the lateral condyle of the third metacarpal bone.
2. Sagittal ridge on the distal articular surface of the third metacarpal bone.
3. Articular surface of the medial condyle of the third metacarpal bone.
4. Proximal dorsal aspect of the proximal phalanx.

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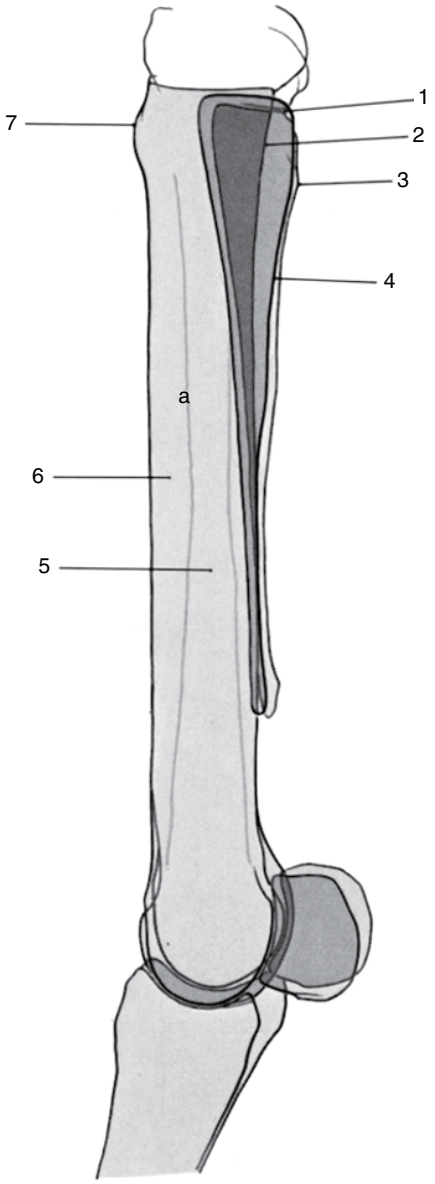


Figure 3.54. Lateromedial (LM) projection of the metacarpus.
(a) Third metacarpal bone.

1. Articulation between the second carpal and the second metacarpal bone.
2. Proximal palmar surface of the third metacarpal bone, where the suspensory ligament (interosseous muscle) attaches. The nutrient foramen is frequently present on the palmar surface of the third metacarpal bone at approximately the junction of the proximal and middle thirds; it may be mistaken for a nondisplaced cortical fracture.
3. Palmar surface of the second metacarpal bone.
4. Palmar aspect of the fourth metacarpal bone. The superimposition of the cortices on the metacarpal bones produces longitudinal lucent lines that may be mistaken for longitudinal fractures.
5. Medullary cavity of the third metacarpal bone.
6. Dorsal cortex of the third metacarpal bone, which is fusiform in shape, i.e. thick in the center and thin toward both extremities.
7. Metacarpal tuberosity.

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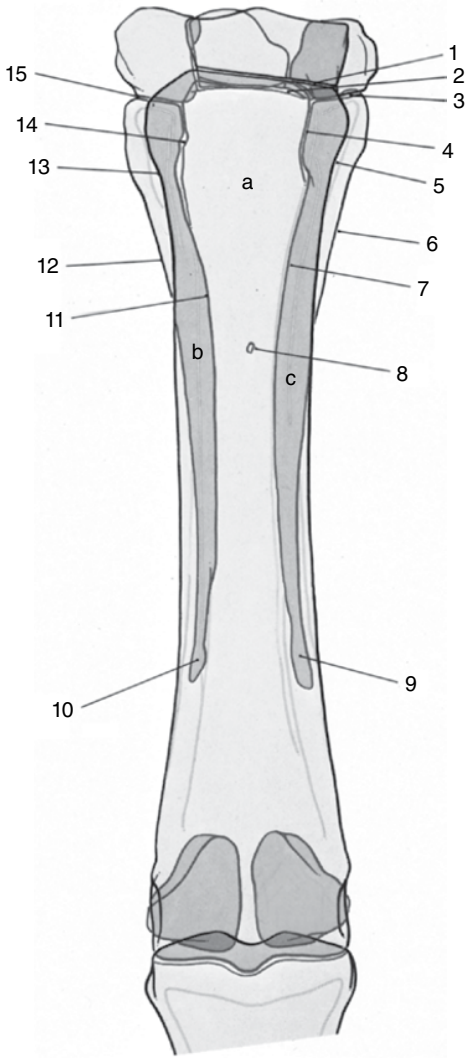
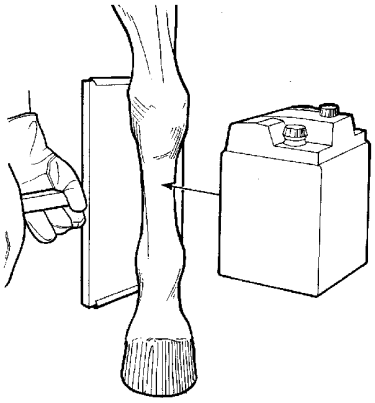


Figure 3.55. Dorsopalmar (DPa) projection of the metacarpus. (a) Third metacarpal bone, (b) fourth metacarpal bone, and (c) second metacarpal bone.

1. Dorsal aspect of the articulation between the third carpal and the third metacarpal bone.
2. Palmar aspect of the articulation between the third carpal and the third metacarpal bone.
3. Articulation between the second carpal and the second metacarpal bone.
4. Articulation between the second and third metacarpal bones.
5. Medioproximal border of the third metacarpal bone.
6. Abaxial border of the second metacarpal bone.
7. Axial border of the second metacarpal bone.
8. End-on projection of the nutrient foramen on the palmar surface of the third metacarpal bone.
9. Distal extent of the second metacarpal bone. The normal shape and position of this bone may be variable. The position may vary in axial and abaxial direction and in proximal or distal direction.
10. Distal extent of the fourth metacarpal bone. The normal shape and position of this bone may be variable. The position may vary in axial and abaxial direction and in proximal or distal direction.
11. Axial border of the fourth metacarpal bone.
12. Abaxial border of the fourth metacarpal bone.
13. Lateral border of the third metacarpal bone.
14. Articulation between the third and fourth metacarpal bones.
15. Articulation between the fourth carpal and the fourth metacarpal bone.

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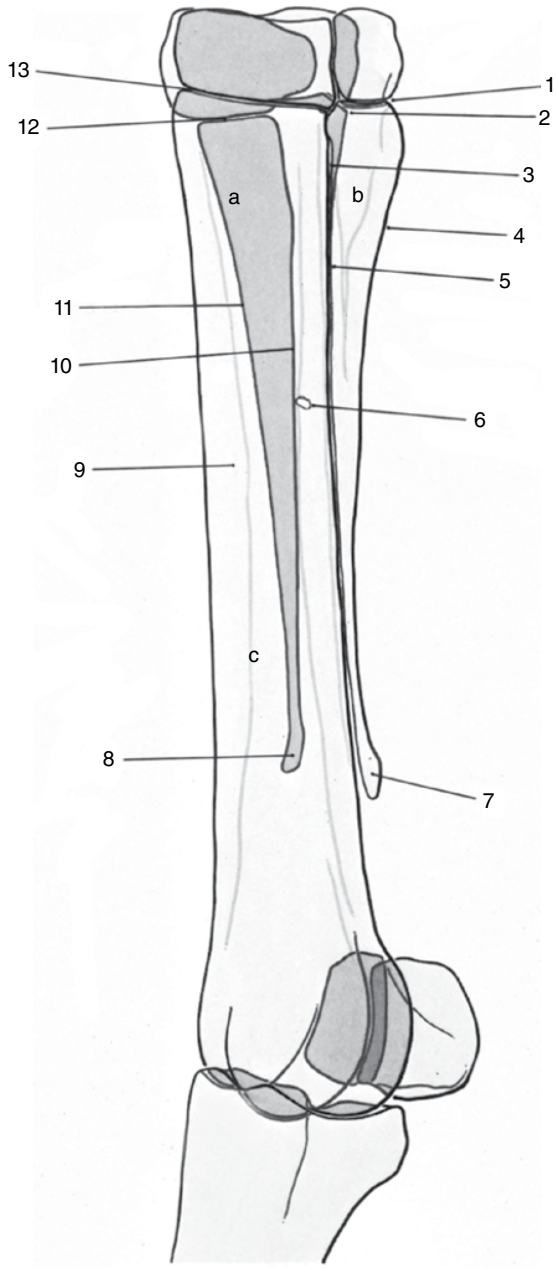
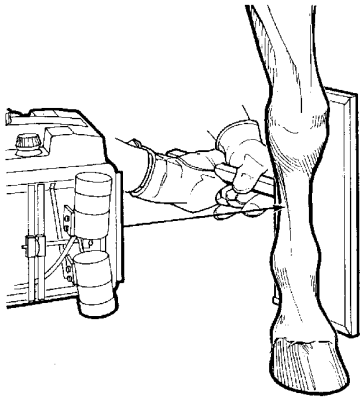


Figure 3.56. Dorsomedial to palmarolateral oblique (D55M-PaLO) projection of the metacarpus. (a) Fourth metacarpal bone, (b) second metacarpal bone, and (c) third metacarpal bone.

1. Articulation between the second carpal and the second metacarpal bone.
2. Proximal palmar medial angle of the third metacarpal bone.
3. Junction between the second and third metacarpal bones.
4. Palmar border of the second metacarpal bone.
5. Dorsal border of the second metacarpal bone.
6. Nutrient foramen on the palmar surface of the third metacarpal bone.
7. Distal end of the second metacarpal bone.
8. Distal end of the fourth metacarpal bone.
9. Dorsolateral cortex of the third metacarpal bone.
10. Palmar surface of the fourth metacarpal bone.
11. Dorsal surface of the fourth metacarpal bone.
12. Articulation between the fourth carpal and the fourth metacarpal bone.
13. Articulation between the third carpal and the third metacarpal bone.

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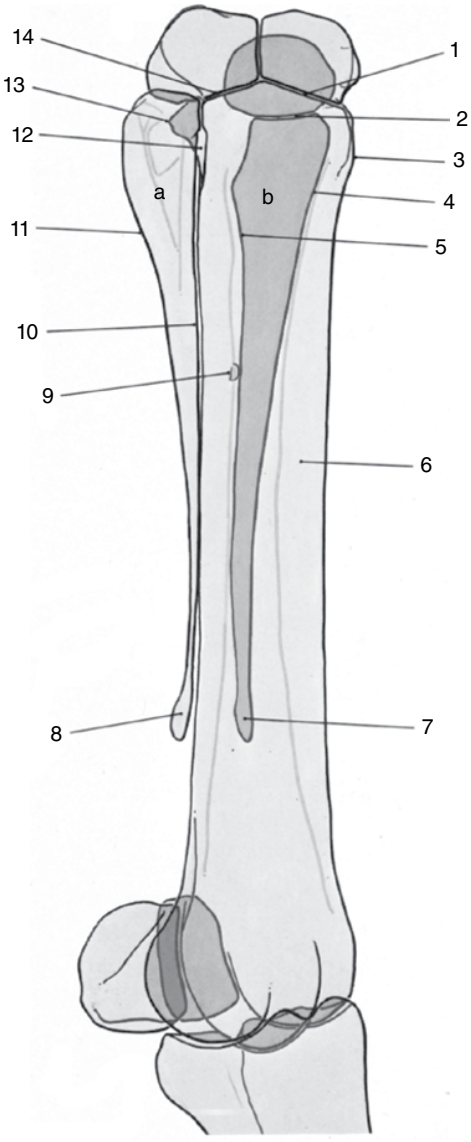
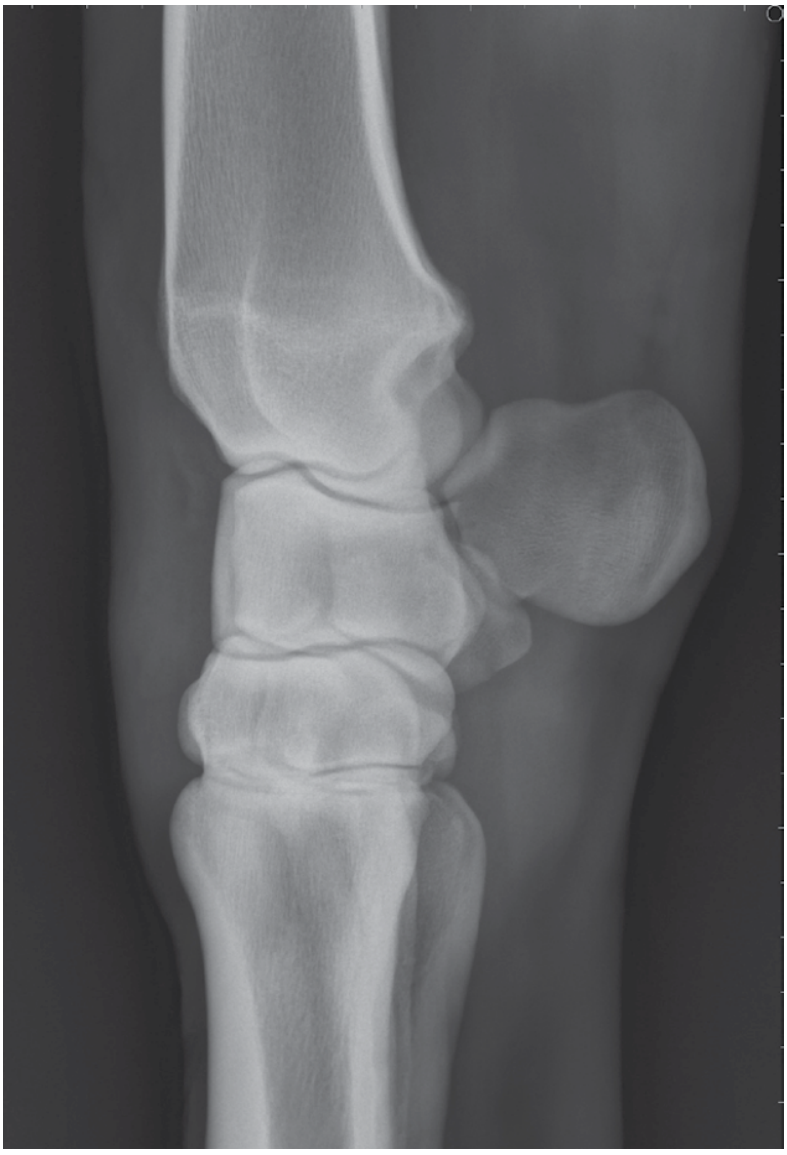


Figure 3.57. Dorsolateral to palmaromedial oblique (D55L-PaMO) projection of the metacarpus. (a) Fourth metacarpal bone, (b) second metacarpal bone, and (c) third metacarpal bone.

1. Articulation between the third carpal and the third metacarpal bone.
2. Articulation between the second carpal and the second metacarpal bone.
3. Metacarpal tuberosity on the dorsomedial surface of the proximal extremity of the third metacarpal bone.
4. Dorsal border of the second metacarpal bone.
5. Palmar border of the second metacarpal bone.
6. Dorsomedial cortex of the third metacarpal bone. The dorsal cortex is normally thick, and the periosteal surface of the cortex should be straight and smooth.
7. Distal end of the second metacarpal bone that may vary in size, shape, and position.
8. Distal ends of the fourth metacarpal bone that may vary in size, shape, and position.
9. Nutrient foramen on the palmar surface of the third metacarpal bone, which may be prominent or not visualized, depending on its size and the projection.
10. Dorsal border of the fourth metacarpal bone.
11. Palmar border of the fourth metacarpal bone.
12. Articulation between the third and fourth metacarpal bones, the visualization of which depends on the incident angle of the primary X-ray beam.
13. Proximal-palmar-lateral angle of the third metacarpal bone.
14. Articulation between the fourth carpal and the third metacarpal bone.

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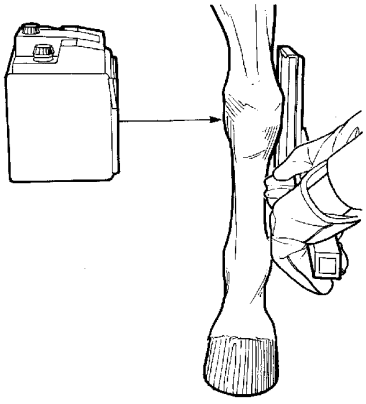


Figure 3.58.

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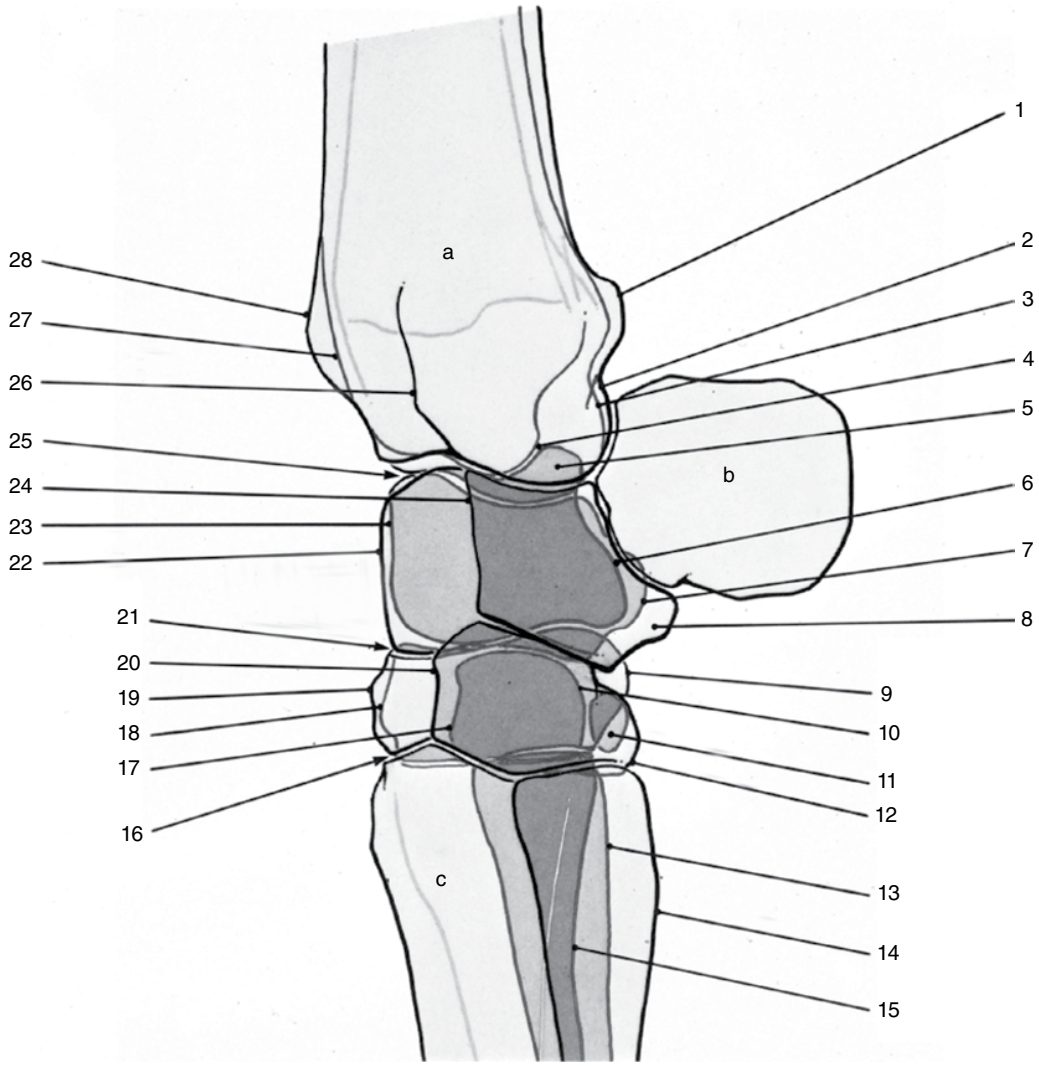


Figure 3.58. (*Continued*) Lateromedial (LM) projection of the carpus. The relative positions of the bony ridges of the distal cranial radius depend on the X-ray beam projection angle. (a) Radius, (b) accessory carpal bone, and (c) third metacarpal bone.

1. Transverse crest, which projects caudad from the closed distal radial physis.
2. Lateral facet of the radial trochlea (lateral styloid process), which articulates with the ulnar carpal bone.
3. Medial facet of the radial trochlea (medial styloid process), which articulates with the radial carpal bone.
4. Intermediate facet of the radial trochlea, which articulates with the intermediate carpal bone.
5. Tuberosity from the proximal palmar aspect of the intermediate carpal bone.
6. Palmar border of the intermediate carpal bone.
7. Palmar border of the radial carpal bone.
8. Palmar border of the ulnar carpal bone. The position of 6, 7, and 8 may change with slight changes in angulation of the X-ray tube or the horse's limb.
9. Palmar border of the third carpal bone.
10. Palmar border of the second carpal bone.
11. First carpal bone, which sometimes is not present.
12. Palmar border of the fourth carpal bone.
13. Proximal palmar border of the second metacarpal bone.
14. Proximal palmar border fourth metacarpal bone.
15. Proximal palmar border of the third metacarpal bone.
16. Carpometacarpal joint.
17. Dorsal border of the second carpal bone.
18. Dorsal border of the third carpal bone.
19. Transverse ridge on the dorsal border of the third carpal bone, which projects with varying degrees of prominence in each horse.
20. Dorsal border of the fourth carpal bone.
21. Midcarpal joint.
22. Dorsal border of the radial carpal bone.
23. Dorsal border of the intermediate carpal bone.
24. Dorsal border of the ulnar carpal bone.
25. Antebrachiocarpal (radiocarpal) joint.
26. Lateral bony ridge of the distal cranial radius, adjacent to the common digital extensor tendon.
27. Bony ridge of the distal cranial radius, adjacent to the medial border of the extensor carpi radialis tendon.
28. Bony ridge of the distal cranial radius between the common digital extensor tendon and the extensor carpi radialis tendon.

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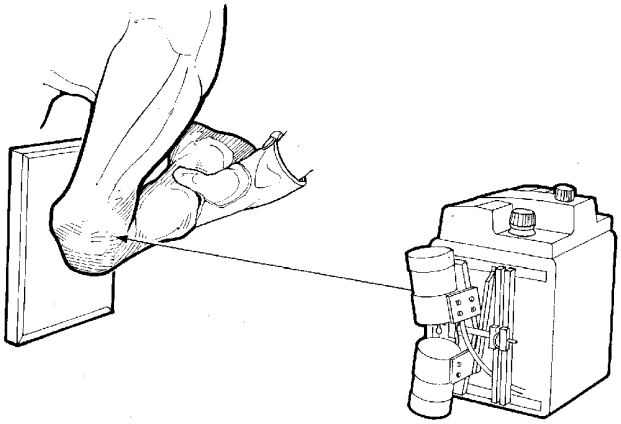


Figure 3.59.

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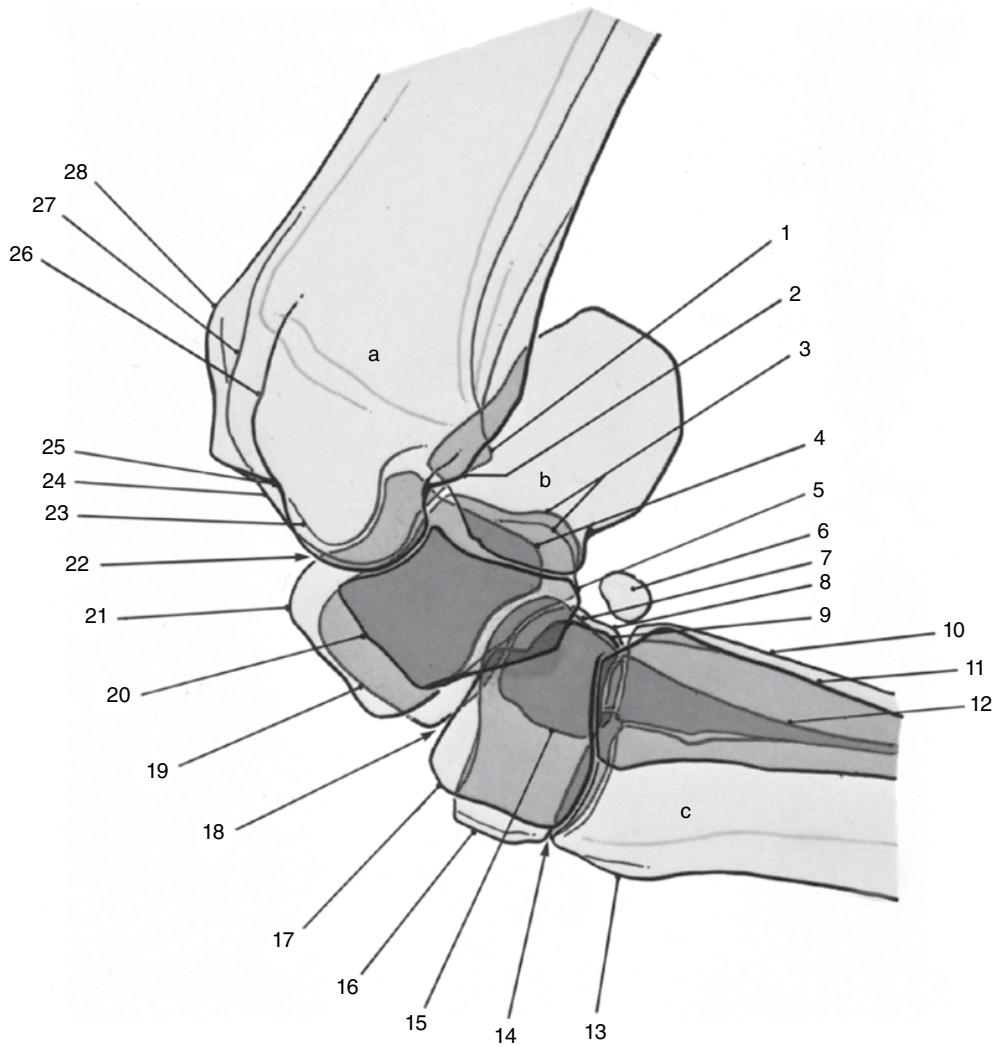


Figure 3.59. (*Continued*) Flexed lateromedial (flexed LM) projection of the carpus. The size and shape of the first and fifth carpal bones may vary, and sometimes the bones are absent. The palmar borders of the carpal and metacarpal bones are closely superimposed in this projection, and the positions may vary slightly with X-ray tube angulation and/or the horse's limb positions. Projection of the bony ridges and facets of the radial trochlea may vary with the positions of the limb and the angle of the X-ray beam. (a) Radius, (b) accessory carpal bone, and (c) third metacarpal bone.

1. Transverse crest, which projects from the caudal aspect of the closed distal radial physis.
2. Ridges produced by the caudal aspect of the medial (medial styloid process) and lateral (lateral styloid process) parts of the radial trochlea.
3. Palmar border of the radial carpal bone.
4. Palmar border of the intermediate carpal bone.
5. Palmar border of the ulnar carpal bone.
6. First carpal bone.
7. Palmar border of the fourth carpal bone.
8. Palmar border of the third carpal bone.
9. Palmar border of the second carpal bone.
10. Proximal palmar border of the second metacarpal bone.
11. Proximal palmar border of the fourth metacarpal bone.
12. Proximal palmar border of the third metacarpal bone.
13. Metacarpal tuberosity, which may vary in prominence.
14. Carpometacarpal joint. Numerous joint space lines are produced by the irregular contour of the bones forming this joint.
15. Dorsal border of the second carpal bone.
16. Dorsal border of the third carpal bone.
17. Dorsal border of the fourth carpal bone. The proximal dorsal aspect of the fourth carpal bone (17) projects proximal to the third carpal bone (16) on the flexed lateral projection.
18. Middle carpal joint.
19. Dorsal border of the radial carpal bone.
20. Dorsal borders of the ulnar carpal bone.
21. Dorsal borders of the intermediate carpal bone. The dorsal borders of the radial (19) and intermediate (21) carpal bones are closely superimposed and may vary slightly. The intermediate carpal bone usually projects proximal to the radial carpal bone on the flexed lateral projection.
22. Antebrachiocarpal (radiocarpal) joint.
23. Intermediate facet of the radial trochlea, which articulates with the intermediate carpal bone (21).
24. Medial border of the radial trochlea.
25. Lateral border of the radial trochlea.
26. Lateral bony ridge adjacent to the lateral border of the common digital extensor tendon.
27. Bony ridge adjacent to the medial border of the extensor carpi radialis.
28. Bony ridge between the lateral digital extensor tendon and the extensor carpi radialis tendon.

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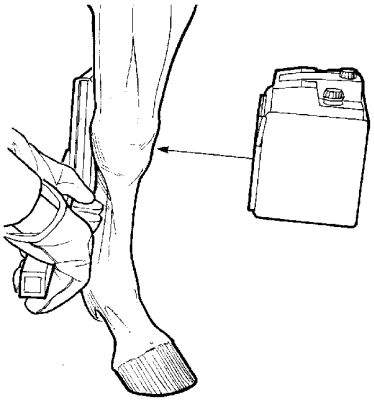


Figure 3.60.

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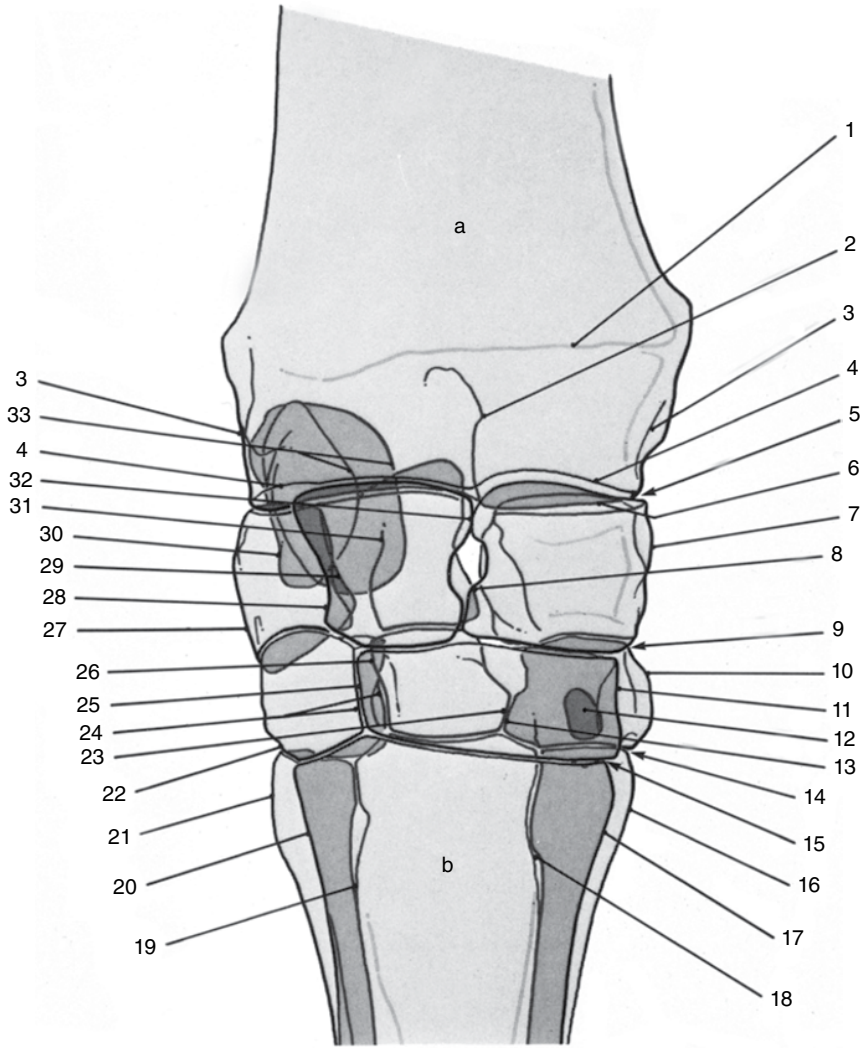


Figure 3.60. (*Continued*) Dorsopalmar (DPa) projection of the carpus. (a) Radius and (b) third metacarpal bone.

1. Physeal scar in the distal extremity of the radius after physeal closure.
2. Caudolateral border of the medial facet (medial styloid process) on the radial trochlea.
3. Depression in the medial and lateral styloid processes for attachment of medial and lateral collateral ligaments. Their appearances and apparent depths can change with a slight obliqueness of the limb.
4. Cranial articular margin on the distal radius.
5. Antebrachiocarpal (radiocarpal) joint.
6. Medial facet (medial styloid process) on the distal radial trochlea, which articulates with the radial carpal bone (7 and 8).
7. Medial border of the radial carpal bone.
8. Lateral border of the radial carpal bone.
9. Midcarpal joint. The different levels of the joint space are the result of many bones forming the articular surfaces and of slight angulation of the X-ray tube or positions of the horse's limb.
10. Medial border of the second carpal bone.
11. Dorsomedial border of the third carpal bone.
12. First carpal bone superimposed over the second and third carpal bone.
13. Lateral border of the second carpal bone.
14. Palmar aspect of the carpometacarpal joint space.
15. Dorsal aspect of the carpometacarpal joint space.
16. Proximomedial border of the second metacarpal bone.
17. Proximomedial border of the third metacarpal bone.
18. Articulation between the second and third metacarpal bones.
19. Articulation between the third and fourth metacarpal bones.
20. Lateral border of the third metacarpal bone.
21. Lateral border of the fourth metacarpal bone.
22. Lateral border of the fourth carpal bone.
23. Medial border of the palmar process on the third carpal bone.
24. Medial border of the fourth carpal bone.
25. Dorsolateral border of the third carpal bone.
26. Lateral border of the palmar process on the third carpal bone.
27. Lateral border of the ulnar carpal bone.
28. Lateral border of the intermediate carpal bone.
29. Medial border of the ulnar carpal bone.
30. Lateral border of the accessory carpal bone.
31. Lateral border of the palmar tuberosity on the intermediate carpal bone.
32. Medial border of the intermediate carpal bone.
33. Concave medial surface of the accessory carpal bone.

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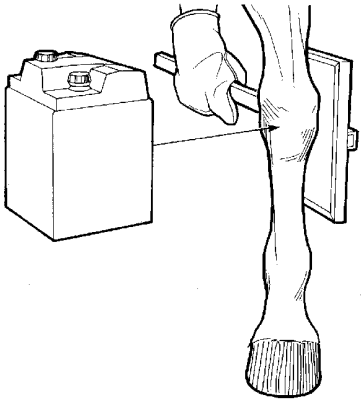


Figure 3.61.

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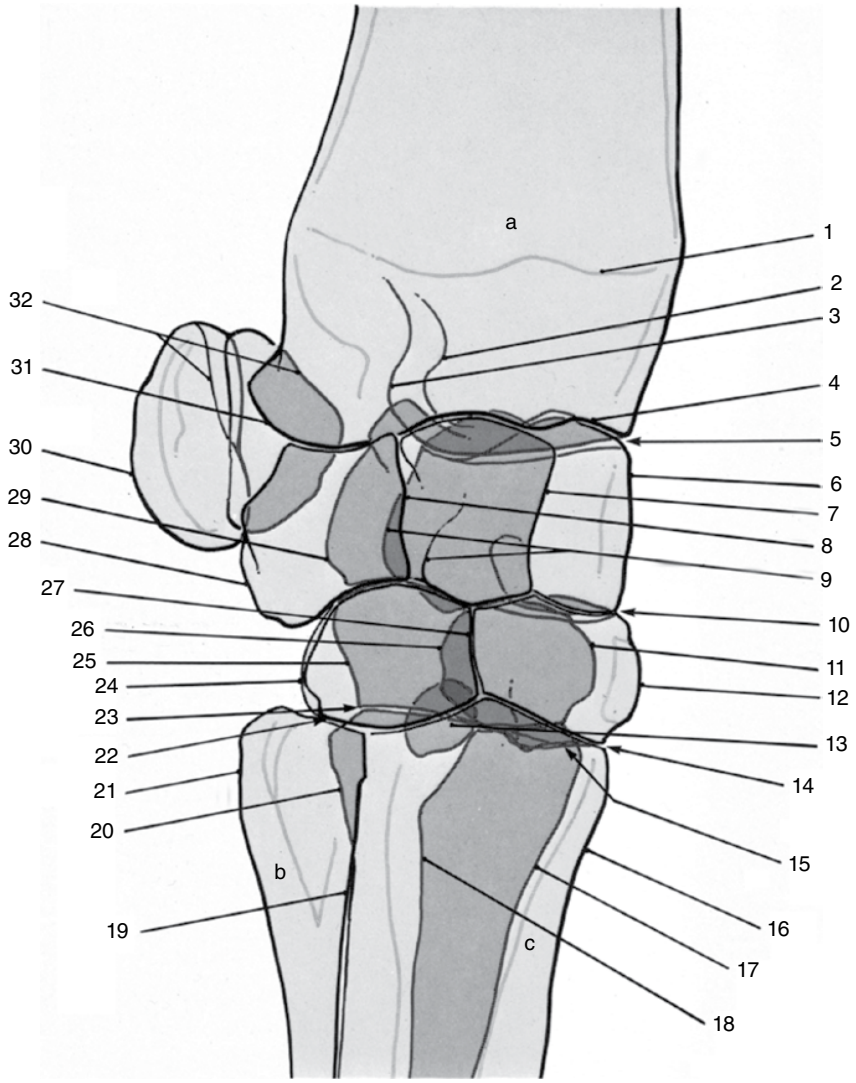


Figure 3.61. (*Continued*) Dorsolateral to palmaromedial oblique (D45L-PaMO) projection of the carpus. (a) Radius, (b) fourth metacarpal bone, and (c) third metacarpal bone.

1. Physeal scar remaining after distal radial physeal closure.
2. Caudal aspect of the intermediate facet on the radial trochlea.
3. Caudal aspect of the medial facet (medial styloid process) on the radial trochlea.
4. Cranial articular margin on the radial trochlea.
5. Dorsomedial aspect of the antebrachiocarpal (radiocarpal) joint.
6. Dorsomedial border of the radial carpal bone.
7. Dorsomedial border of the intermediate carpal bone.
8. Dorsomedial border of the ulnar carpal bone.
9. Tubercles on the palmar surface of the radiocarpal bone.
10. Dorsomedial aspect of the midcarpal joint.
11. Dorsomedial border of the second carpal bone.
12. Transverse ridge on the dorsomedial border of the third carpal bone.
13. First carpal bone, which is difficult to see on this projection because of superimposition.
14. Medial aspect of the carpometacarpal joint.
15. Dorsal aspect of the carpometacarpal joint. The multiple joint spaces are associated with the carpometacarpal joint and vary in appearance on different projection angles.
16. Dorsomedial border of the third metacarpal bone.
17. Dorsomedial border of the second metacarpal bone.
18. Palmarolateral border of the second metacarpal bones.
19. Dorsomedial border of the fourth metacarpal bone.
20. Palmarolateral border of the third metacarpal bone.
21. Palmarolateral border of the fourth metacarpal bone.
22. Palmarolateral aspect of the carpometacarpal joint between the fourth carpal bone and the third and fourth metacarpal bones.
23. Palmar aspect of the carpometacarpal joint between the third carpal and the third metacarpal bone.
24. Tubercle on the palmarolateral border of the fourth carpal bones.
25. Palmarolateral border of the third carpal bone.
26. Palmarolateral border of the second carpal bone.
27. Dorsomedial border of the fourth carpal bone.
28. Palmarolateral border of the ulnar carpal bone.
29. Palmarolateral border of the intermediate carpal bone.
30. Palmarolateral border of the accessory carpal bone.
31. Lateral facet (lateral styloid process) on the radial trochlea.
32. Medial, concave surface of the accessory carpal bone.

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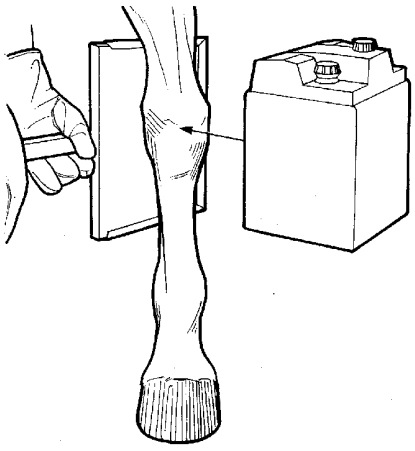


Figure 3.62.

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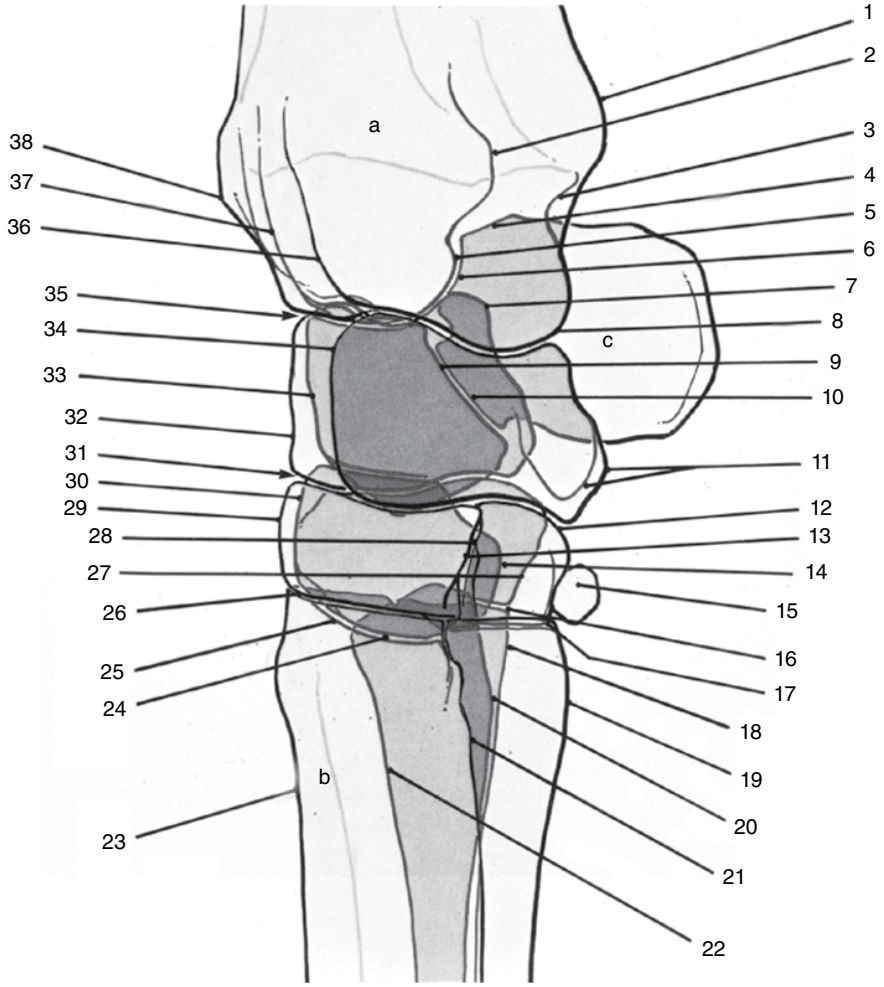


Figure 3.62. (*Continued*) Dorsomedial to palmarolateral oblique (D60M-PaLO) projection of the carpus. The relative positions of the bony ridges on the distocranial aspect of the radius change slightly with different X-ray beam projections. (a) Radius, (b) third metacarpal bone, and (c) accessory carpal bone.

1. Bony projection from the mediocaudal surface of the radius.
2. Transverse crest proximal to the lateral facet (lateral styloid process) of the radial trochlea.
3. Indentation proximal to the medial facet (medial styloid process) of the radial trochlea.
4. Proximal border of the accessory carpal bone.
5. Caudal border of the lateral and intermediate facets of the radial trochlea superimposed.
6. Proximal articular surface of the accessory carpal bone.
7. Palmaromedial border of the intermediate carpal bone.
8. Medial facet (medial styloid process) of the radial trochlea.
9. Palmaromedial border of the ulnar carpal bone.
10. Distal articular surface of the accessory carpal bone.
11. Palmaromedial border of the radial carpal bone.
12. Palmaromedial border of the second carpal bone.
13. Palmaromedial border of the third carpal bone.
14. Palmaromedial border of the fourth carpal bone.
15. First carpal bone.
16. Carpometacarpal articulation between the third carpal and the third metacarpal bone.
17. Carpometacarpal articulation between the second carpal and the second metacarpal bone.
18. Palmaromedial border of the third metacarpal bone.
19. Palmaromedial border of the second metacarpal bone.
20. Palmaromedial border of the fourth metacarpal bone.
21. Dorsolateral border of the second metacarpal bone.
22. Dorsolateral border of the fourth metacarpal bone.
23. Dorsolateral border of the third metacarpal bone.
24. Carpometacarpal articulation between the fourth carpal and the fourth metacarpal bone.
25. Carpometacarpal articulation between the fourth carpal and the third metacarpal bone.
26. Carpometacarpal articulation between the third carpal and the third metacarpal bone.
27. Palmaromedial border of the third carpal bone.
28. Dorsal border of the second carpal bone.
29. Dorsal border of the third carpal bone.
30. Dorsal border of the fourth carpal bone.
31. Dorsolateral aspect of the midcarpal joint.
32. Dorsal border of the intermediate carpal bone.
33. Dorsal border of the ulnar carpal bone.
34. Dorsal border of the radial carpal bone. The relative position and appearance of the dorsal borders of 32, 33, and 34 may change with slight projection differences of the X-ray beam.
35. Antebrachiocarpal (radiocarpal) joint.
36. Bony ridge forming the medial border of the groove for the common digital extensor tendon.
37. Bony ridge along the lateral border of the extensor carpi radialis tendon.
38. Bony ridge between the grooves for the common digital extensor and the extensor carpi radialis tendons.

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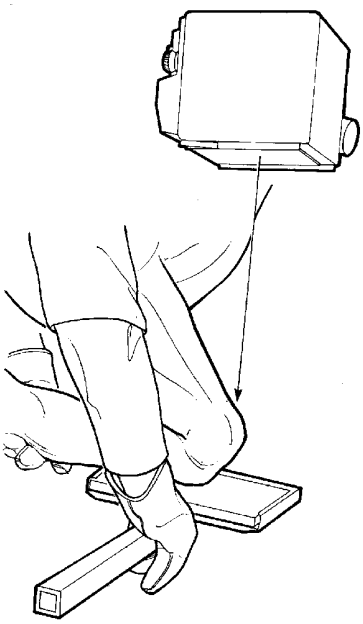


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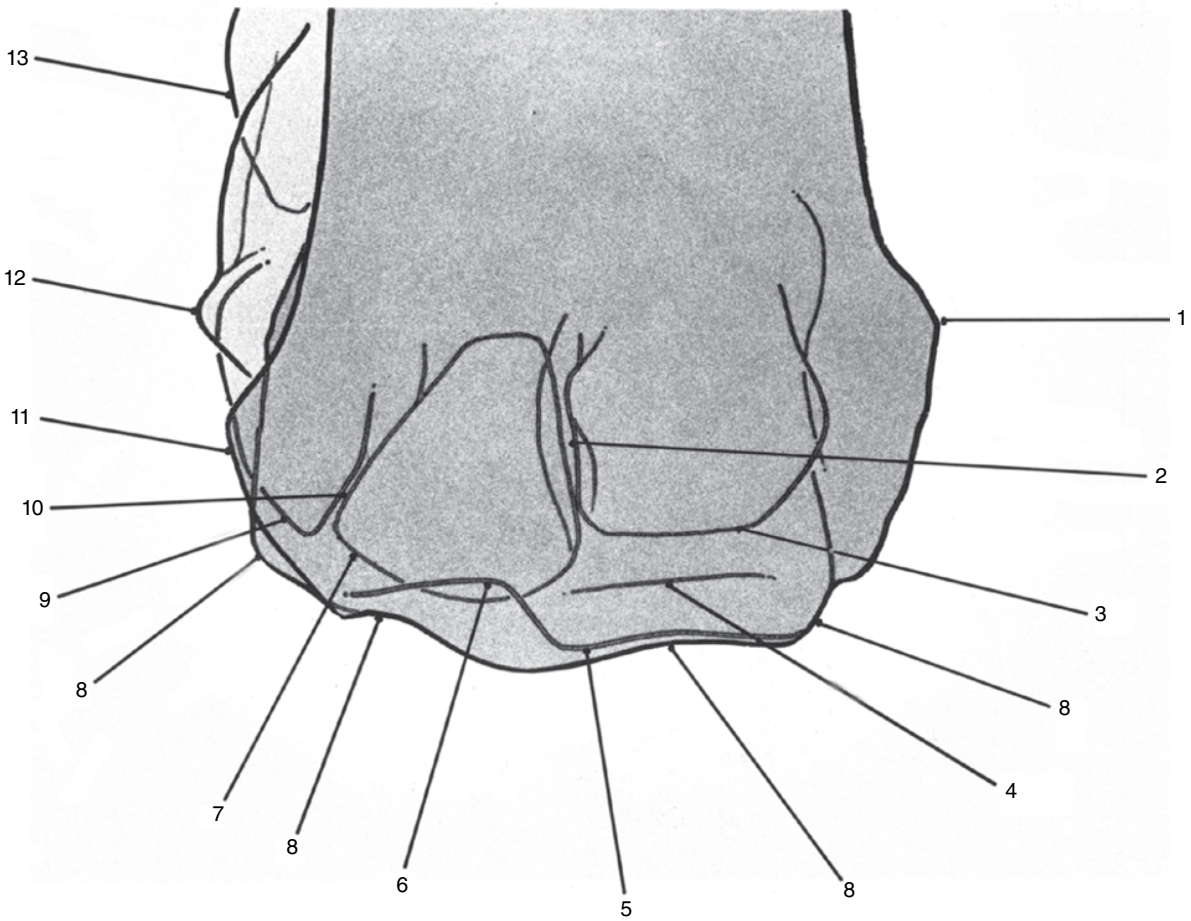
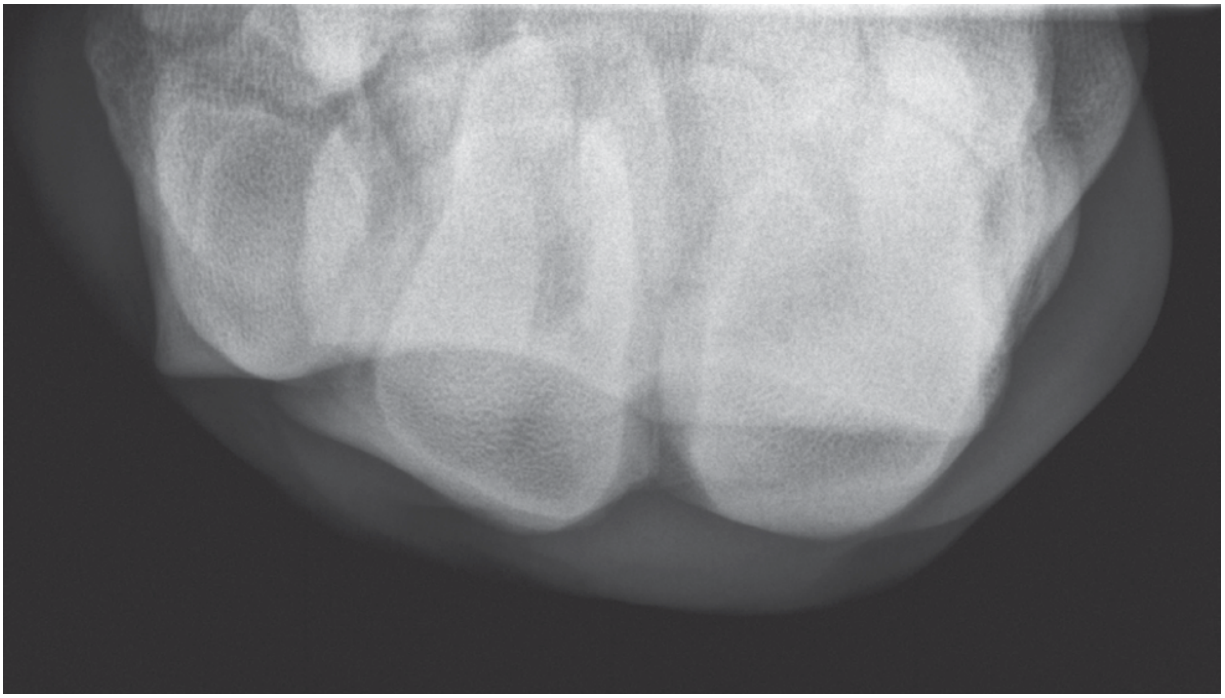


Figure 3.63. (*Continued*) Flexed dorsoproximal to dorsodistal oblique (flexed D80Pr-DDiO) projection of the distal radius.

1. Tuberosity on the distomedial aspect of the radius for attachment of the medial collateral ligament.
2. Junction between the radial and intermediate carpal bones.
3. Dorsal border of the radial carpal bone.
4. Dorsoproximal border of the radial carpal bone.
5. Medial facet (medial styloid process) of the radial trochlea.
6. Intermediate facet of the radial trochlea.
7. Dorsal border of the intermediate carpal bone.
8. Dorsal articular margin of the radial trochlea.
9. Dorsal border of the ulnar carpal bone.
10. Junction between the ulnar and intermediate carpal bones.
11. Tuberosity on the distal lateral radius for attachment of the lateral collateral ligament.
12. Lateral border of the accessory carpal bone.
13. Proximolateral border of the fourth metacarpal bone.

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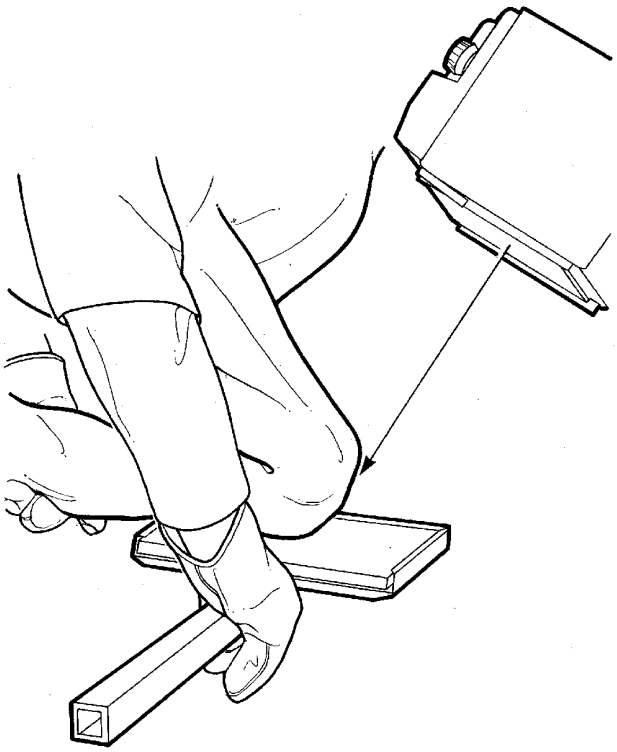


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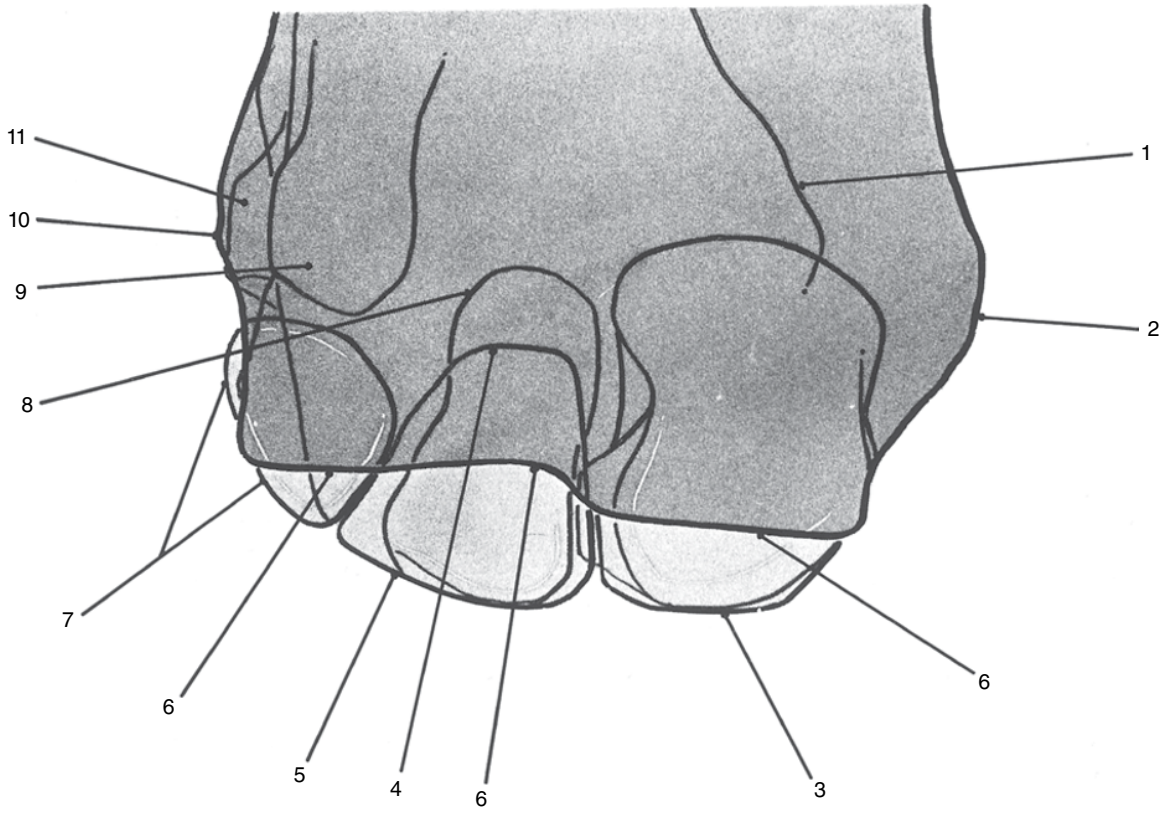


Figure 3.64. (*Continued*) Flexed dorsoproximal to dorsodistal oblique (flexed D55Pr-DDiO) projection of the proximal row of carpal bones. The position of the distal extremity of the radius relative to the proximal carpal bones varies with position of the horse's limb and the angulation of the primary X-ray beam. Slight changes in either may cause different degrees of superimposition of the radius and carpal bones.

1. Medial border of the second metacarpal bone.
2. Tuberosity on the distomedial aspect of the radius for attachment of the medial collateral ligament.
3. Dorsal border of the radial carpal bone.
4. Proximopalmar border of the intermediate carpal bone.
5. Dorsal border of the intermediate carpal bone.
6. Radial trochlea.
7. Dorsolateral border of the ulnar carpal bone.
8. Palmarodistal border of the intermediate carpal bone.
9. Proximal aspect of the fourth metacarpal bone.
10. Lateral tuberosity on the distal extremity of the radius for attachment of the lateral collateral ligament.
11. Accessory carpal bone.

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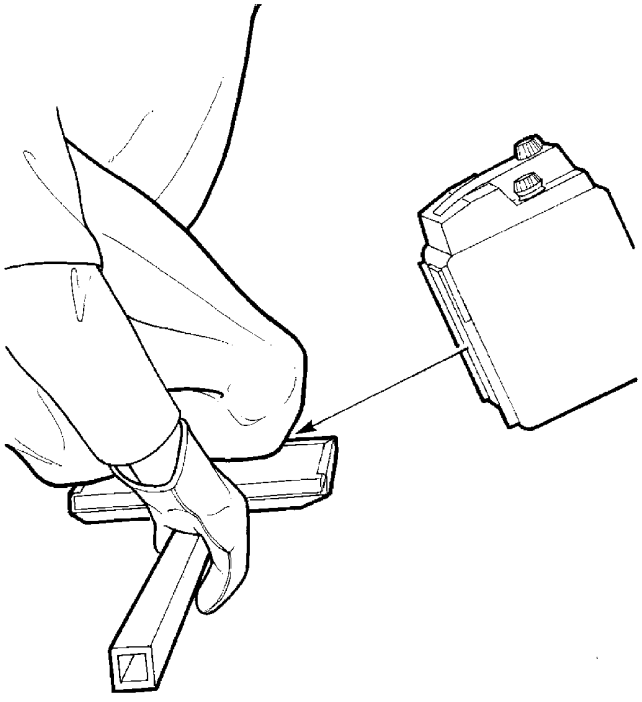


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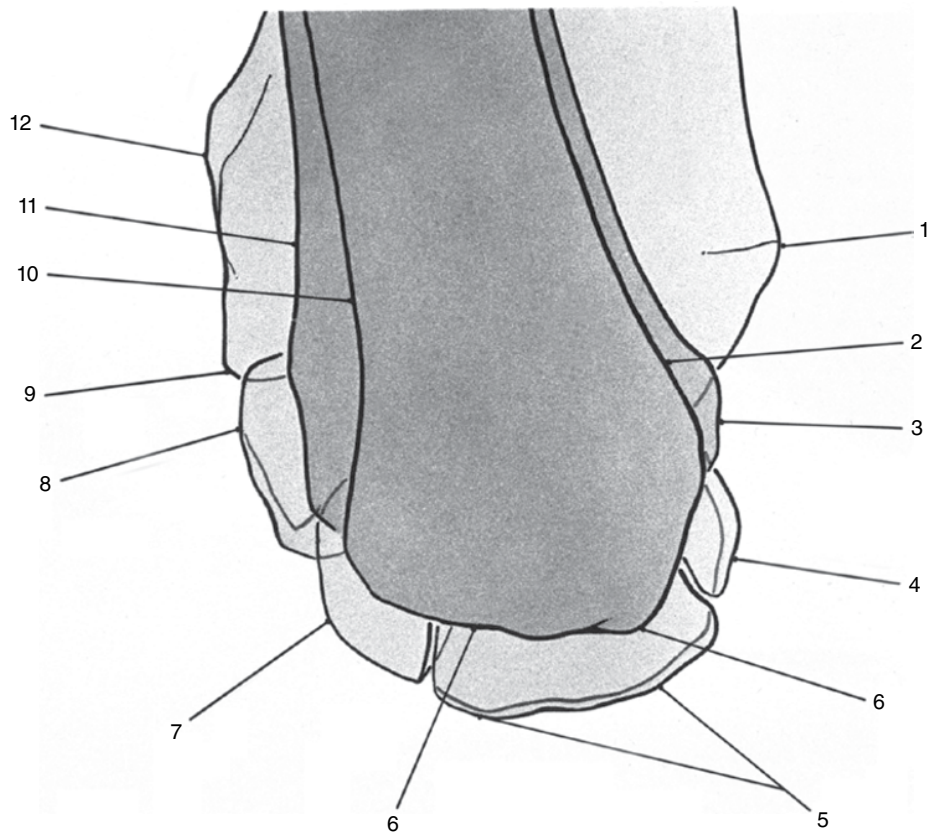
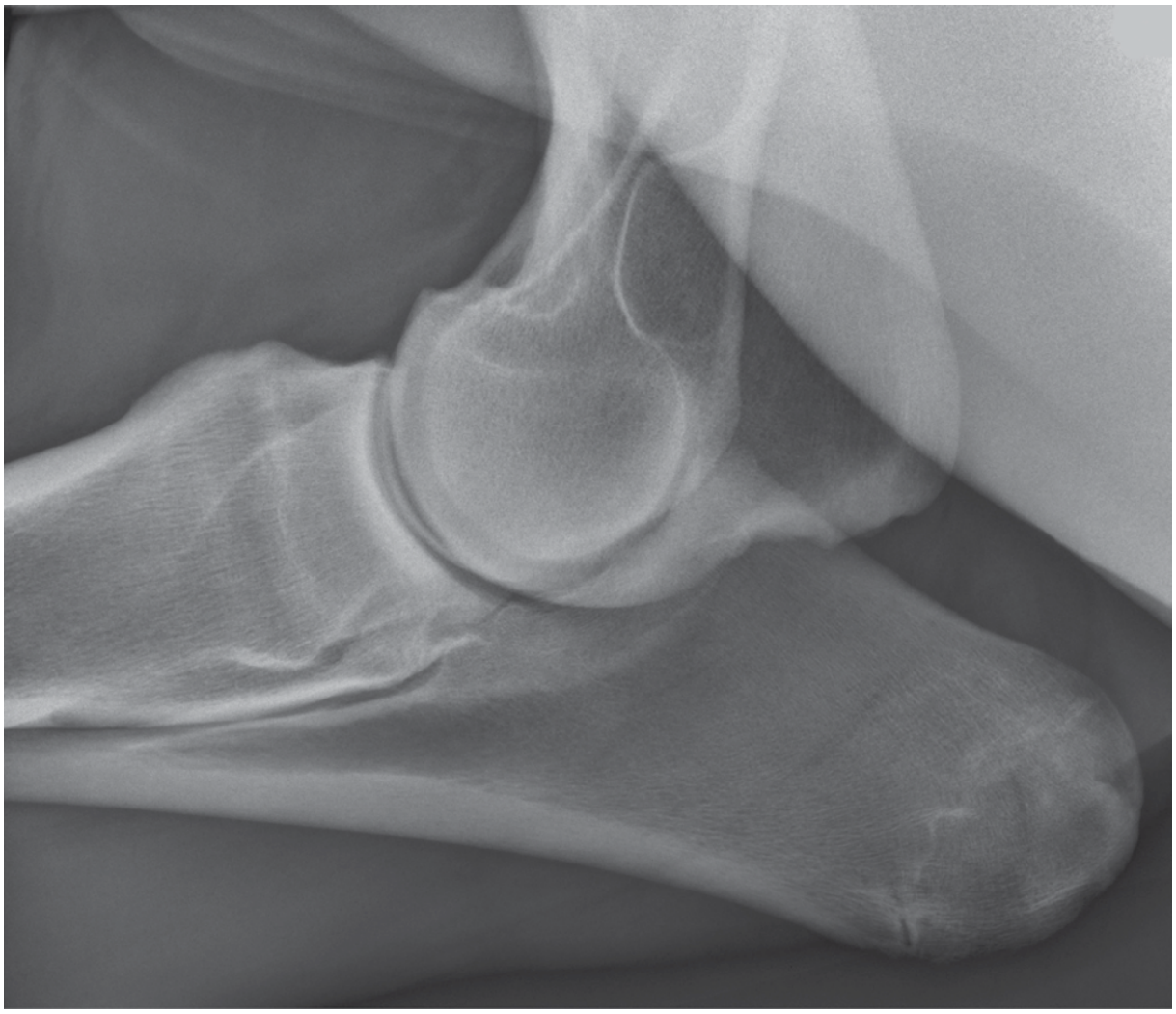


Figure 3.65. (*Continued*) Flexed dorsoproximal to dorsodistal oblique (flexed D30Pr-DDiO) projection of the distal row of carpal bones. The apparent shape of the third carpal bone may be changed by X-ray tube angulation and the position of the horse's limb and may appear more elongated than shown here. The dorsal cortex and medullary cavity should be evident on the medial aspect of the normal third carpal bone if properly positioned.

1. Medial tuberosity on the distal radius for attachment of the medial collateral ligament.
2. Medial border of the third metacarpal bone.
3. Medial border of the second metacarpal bone.
4. Second carpal bone.
5. Dorsal border of the third carpal bone.
6. Superimposed dorsal border of the proximal carpal bones and the third metacarpal bone.
7. Fourth carpal bone.
8. Lateral border of the accessory carpal bone.
9. Lateral aspect (lateral styloid process) of the radial trochlea.
10. Lateral border of the third metacarpal bone.
11. Lateral border of the fourth metacarpal bone.
12. Lateral tuberosity on the distal extremity of the radius for attachment of the lateral collateral ligament.

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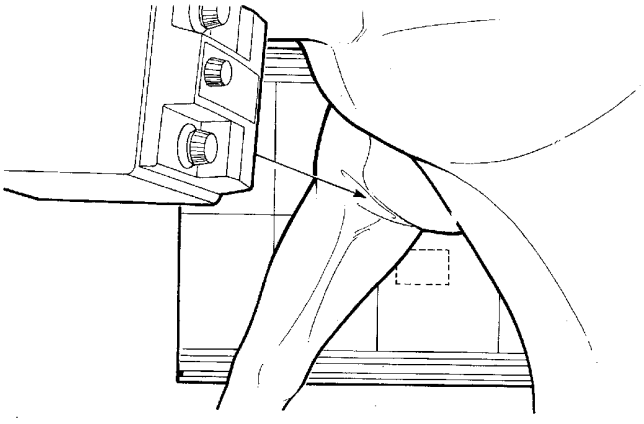


Figure 3.66.

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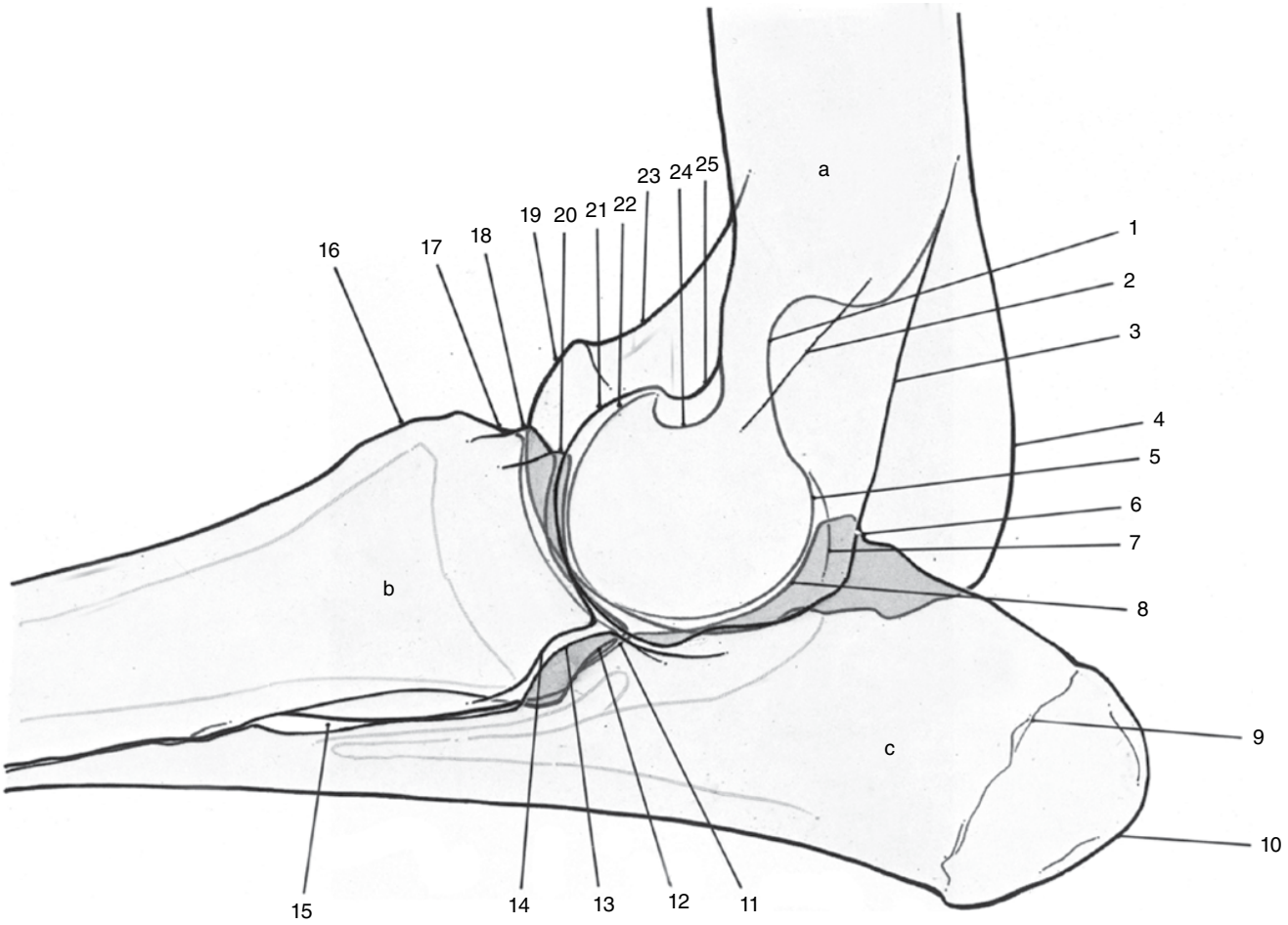


Figure 3.66. (*Continued*) Mediolateral (ML) projection of the humeroulnar and humeroradial joint (elbow). (a) Humerus, (b) radius, and (c) ulna.

1. Floor of the olecranon fossa.
2. Lateral supracondylar crest on the distal extremity of the humerus.
3. Lateral epicondyle of the distal humerus.
4. Medial epicondyle of the distal humerus.
5. Sagittal trochlear groove on the medial humeral condyle.
6. Anconeal process of the ulna.
7. Articular surface of the trochlea (medial condyle) on the humerus.
8. Trochlear notch (ulnar articular surface).
9. Growth plate (physis) in the proximal ulna.
10. Olecranon tuberosity.
11. Medial aspect of the coronoid process of the ulna.
12. Middle caudal border of the radial head.
13. Lateral aspect of the coronoid process of the ulna.
14. Laterocaudal border of the radial head.
15. Interosseous space between the radius and ulna.
16. Radial tuberosity.
17. Craniomedial border of the radial head.
18. Midcranial border of the radial head.
19. Trochlea (medial condyle) of the humerus.
20. Cranial lateral border of the radial head.
21. Capitulum of the humerus.
22. Cranial surface (floor) of the sagittal groove on the trochlea of the humerus.
23. Medial border of the radial fossa.
24. Floor of the radial fossa.
25. Lateral border of the radial fossa.

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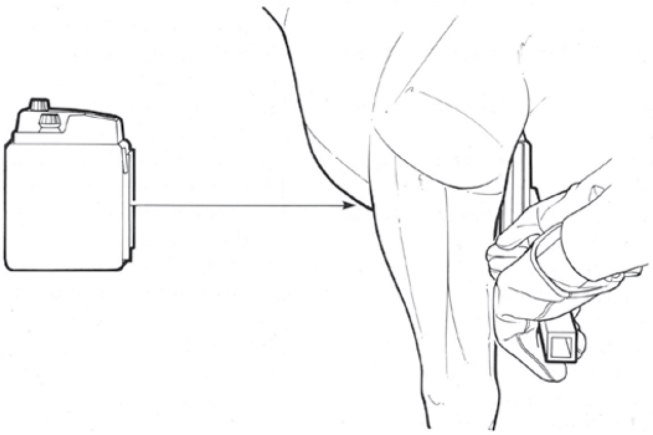


Figure 3.67.

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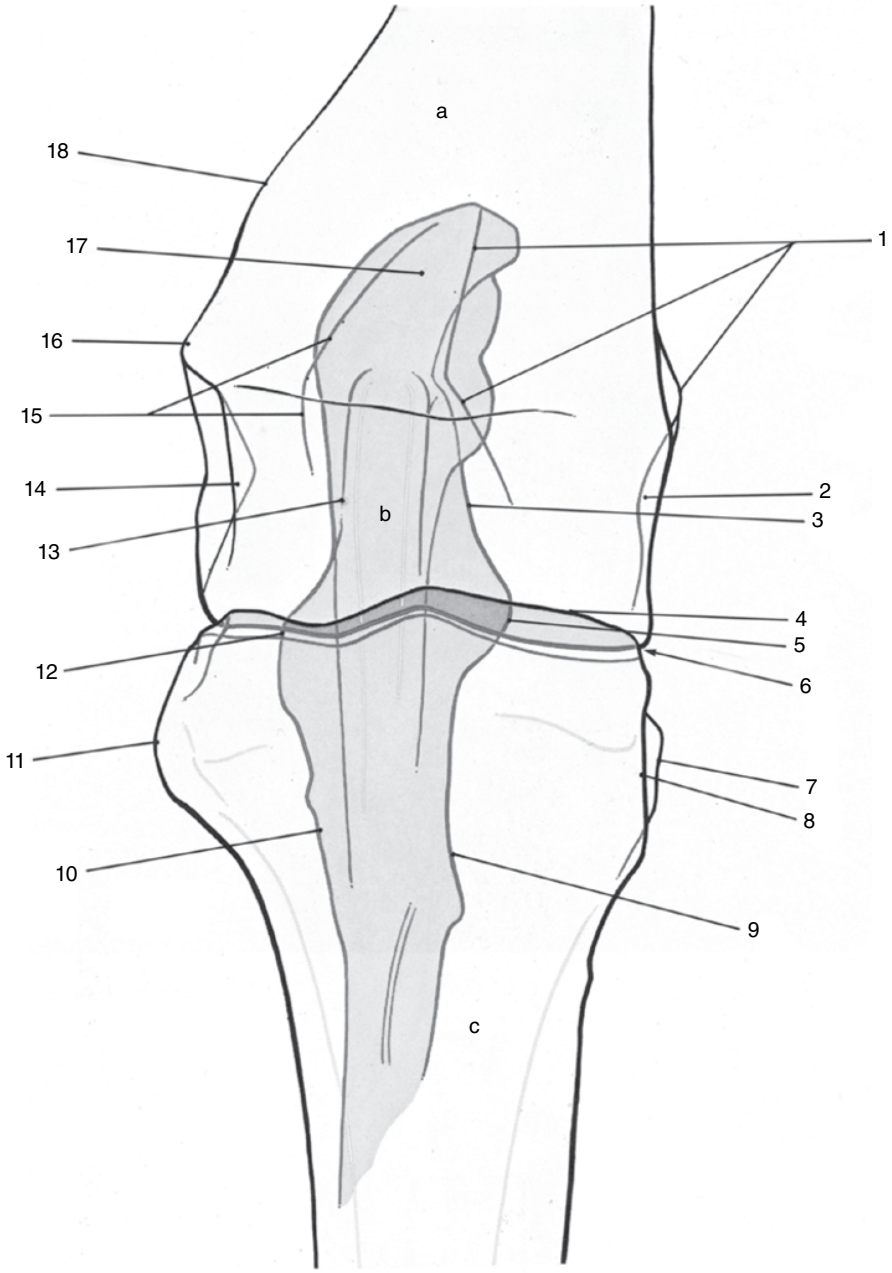


Figure 3.67. (*Continued*) Craniocaudal (CrCa) projection of the humeroulnar and humeroradial joint (elbow). (a) Humerus, (b) ulna, and (c) radius.

1. Medial epicondyle of the humerus, which is large and superimposed over the distal extremity of the humerus except for a slight convex projection on the distal medial humerus where the medial collateral ligament attaches.
2. Depression on the distomedial aspect of the humerus.
3. Medial border of the trochlear notch on the ulna.
4. Caudal margin of the capitular fovea (proximal radial articulation).
5. Medial aspect of the coronoid process of the ulna.
6. Humeroradial articulations.
7. Radial tuberosity.
8. Medial tuberosity of the radius for collateral ligament attachment.
9. Medial border of the ulna.
10. Lateral border of the ulna.
11. Lateral tuberosity of the radius for collateral ligament attachment.
12. Lateral aspect of the coronoid process of the ulna.
13. Lateral border of the trochlear notch on the ulna.
14. Depression for attachment of the lateral collateral ligament.
15. Lateral border of the olecranon fossa.
16. Lateral epicondyle of the humerus.
17. Olecranon tuberosity of the ulna.
18. Lateral supracondylar crest.

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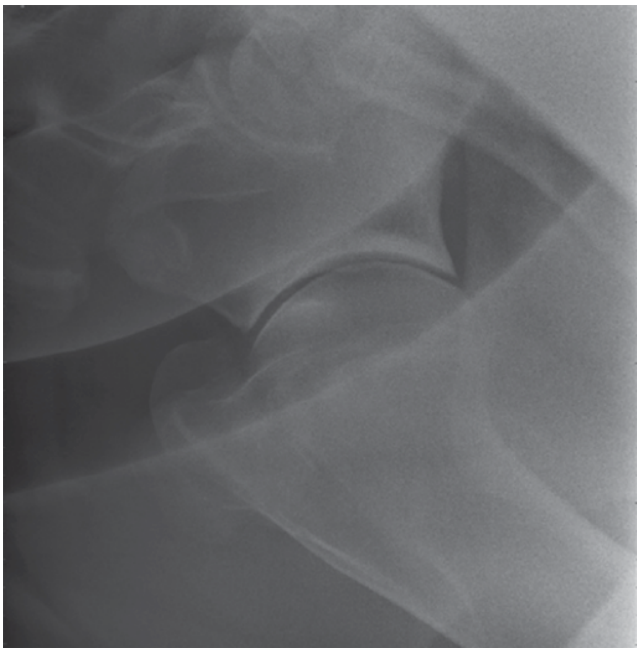




Figure 3.68.

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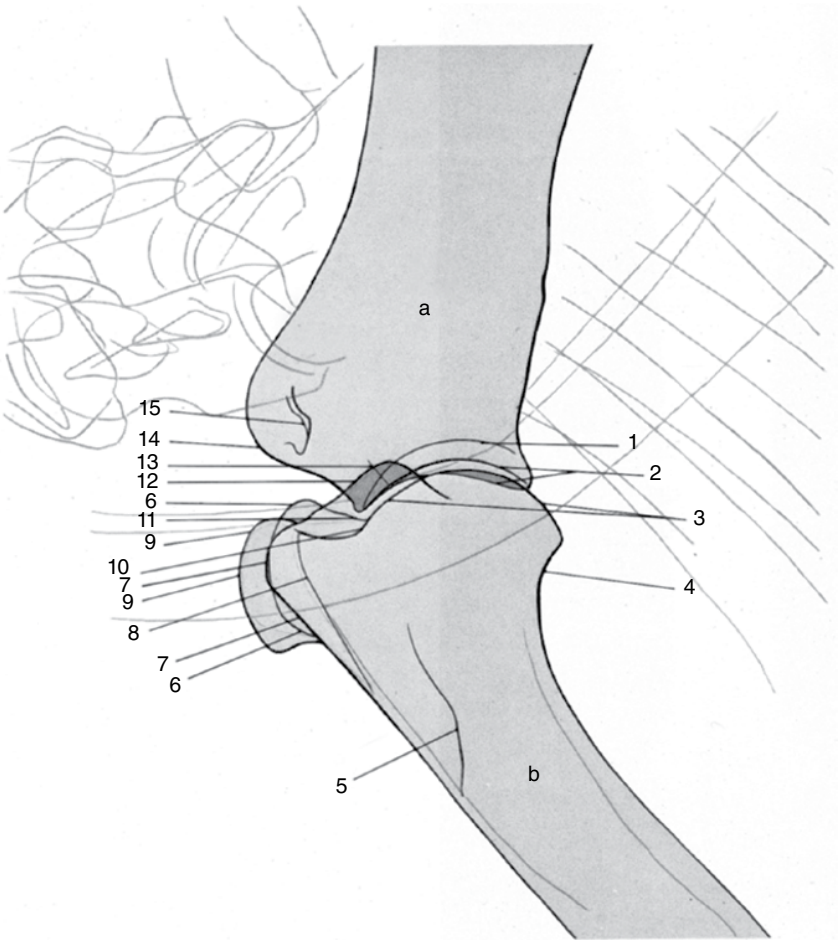


Figure 3.68. (*Continued*) (A–C) Mediolateral (ML) projection of the scapulohumeral joint (shoulder) (A) radiographic positioning to acquire radiograph (B), anatomic detail of radiograph (C). (a) Scapula and (b) humerus.

1. Subchondral bone on the concave surface of the glenoid cavity.
2. Medial and lateral borders of the glenoid cavity.
3. Cranial and caudal borders of the humeral head.
4. Caudal border of the humeral neck.
5. Deltoid tuberosity superimposed on the humerus.
6. Proximal and distocranial borders of the lesser (medial) tubercle.
7. Cranial border of the greater (lateral) tubercle.
8. Floor of the intertuberal groove between the lateral and intermediate tubercles.
9. Intermediate tubercle.
10. Fossa between the tubercles and the humeral head.
11. Caudal part of the lesser (medial) tubercle.
12. Caudal part of the greater (lateral) tubercle.
13. Glenoid notch, which is more or less apparent, depending on the X-ray beam projection angle and development in the horse.
14. Supraglenoid tubercle.
15. Coracoid process.

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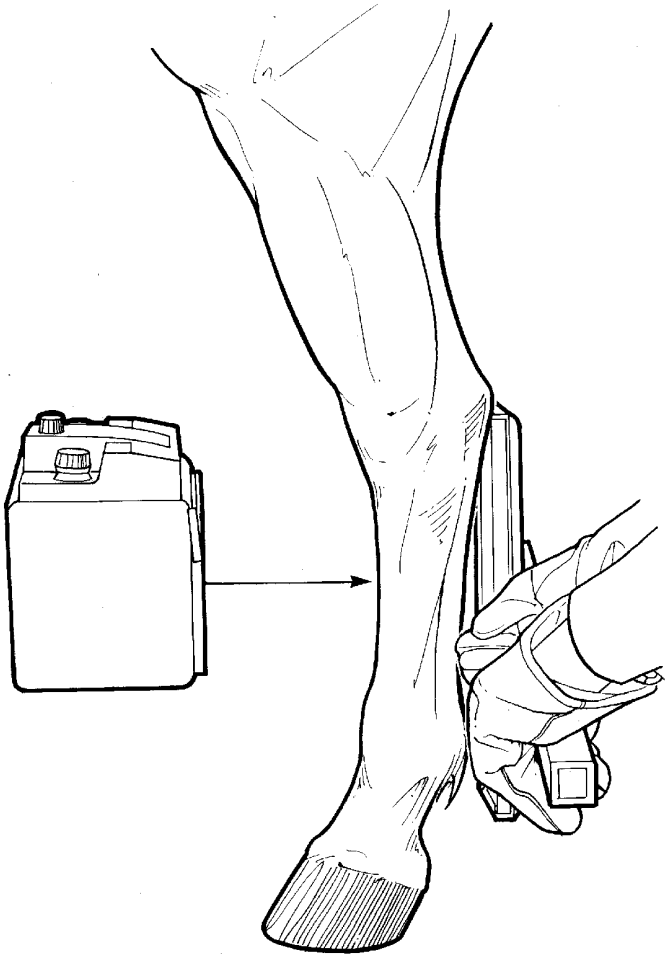


Figure 3.69.

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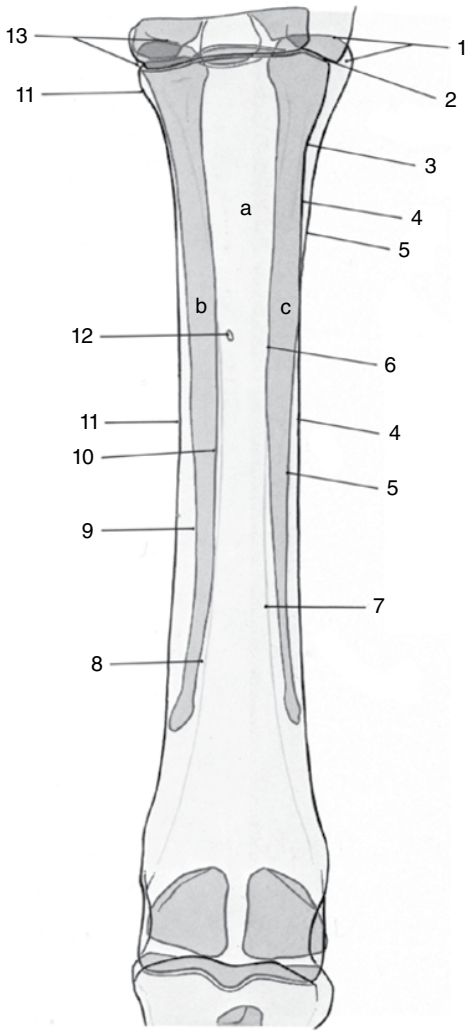
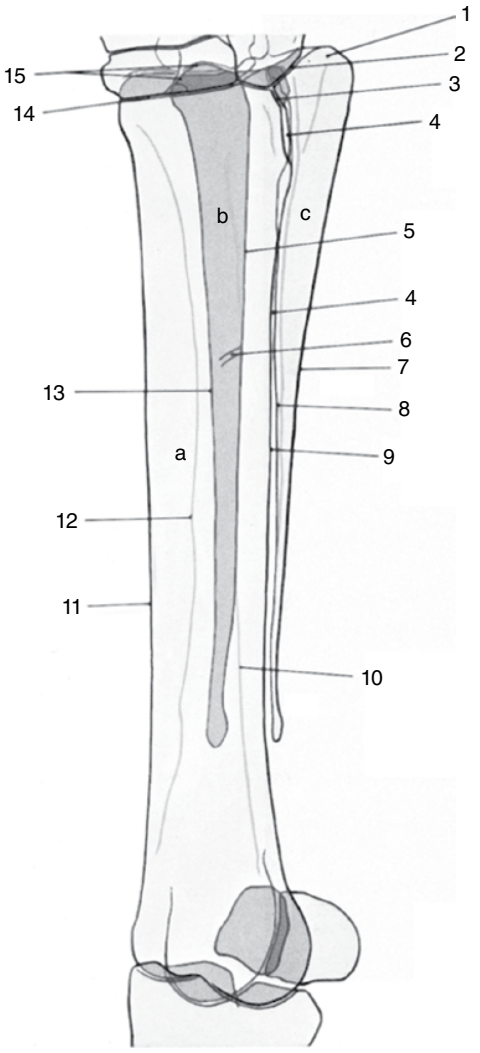


Figure 3.69. (*Continued*) Dorsoplantar (DPl) projection of the metatarsus. (a) Third metatarsal bone, (b) second metatarsal bone, and (c) fourth metatarsal bone.

1. Proximal extremity (base) of the fourth metatarsal bone, which is larger than the second metatarsal bone and is superimposed over the third and fourth tarsal bones.
2. Articulation between the fourth tarsal and the third metatarsal bone.
3. Lateroproximal border of the third metatarsal bone.
4. Lateral surface of the third metatarsal bone.
5. Abaxial surface of the fourth metatarsal bone.
6. Axial surface of the fourth metatarsal bone.
7. Endosteal surface on the lateral cortex of the third metatarsal bone.
8. Endosteal surface on the medial cortex of the third metatarsal bone.
9. Abaxial surface of the second metatarsal bone.
10. Axial surface of the second metatarsal bone.
11. Medial surface of the third metatarsal bone.
12. Nutrient foramen on the plantar surface of the third metatarsal bone.
13. Proximal extremity (base) of the second metatarsal bone, which is superimposed over the fused first and second tarsal bones and the third tarsal bone.

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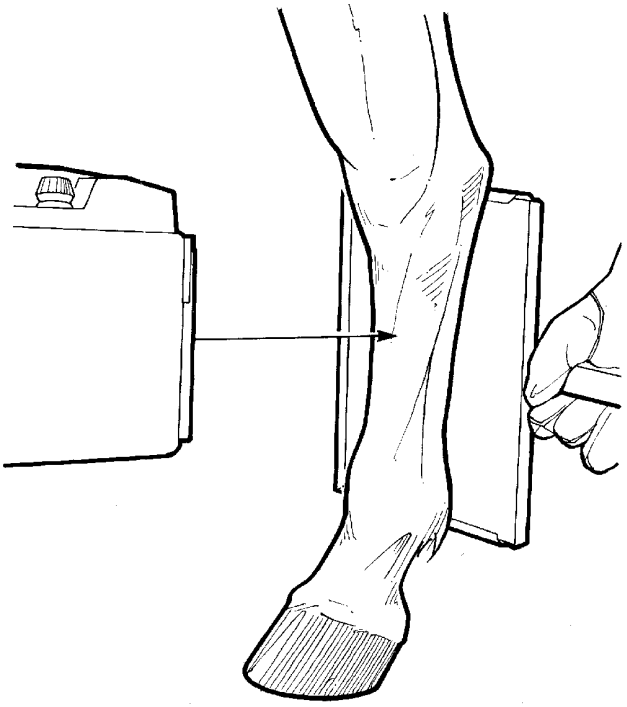


Figure 3.70. Dorsolateral to plantaromedial oblique (D45L-PlMO) projection of the metatarsus. (a) Third metatarsal bone, (b) second metatarsal bone, and (c) fourth metatarsal bone.

1. Proximal extremity (base) of fourth metatarsal bone.
2. Articulation between the fourth metatarsal and the fourth tarsal bone.
3. Articulation between the fourth and third metatarsal bones.
4. Interosseous space between the third and fourth metatarsal bones.
5. Plantarolateral border of second metatarsal bone.
6. Nutrient foramen on the plantar surface of the third metatarsal bone.
7. Plantarolateral border of fourth metatarsal bone.
8. Dorsomedial border of fourth metatarsal bone.
9. Plantarolateral border of third metatarsal bone.
10. Endosteal surface on the plantarolateral cortex of third metatarsal bone.
11. Dorsomedial surface of third metatarsal bone.
12. Endosteal surface on the dorsomedial cortex of third metatarsal bone.
13. Dorsomedial surface of second metatarsal bone.
14. Articulation between the third tarsal and the third metatarsal bone.
15. Proximal extremity (base) of the second metatarsal bone superimposed over the distal tarsal bones.

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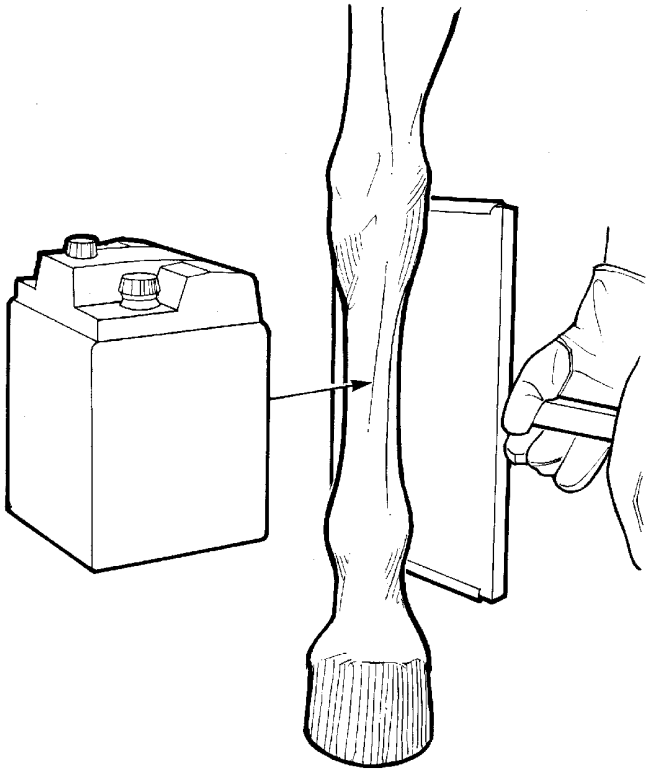
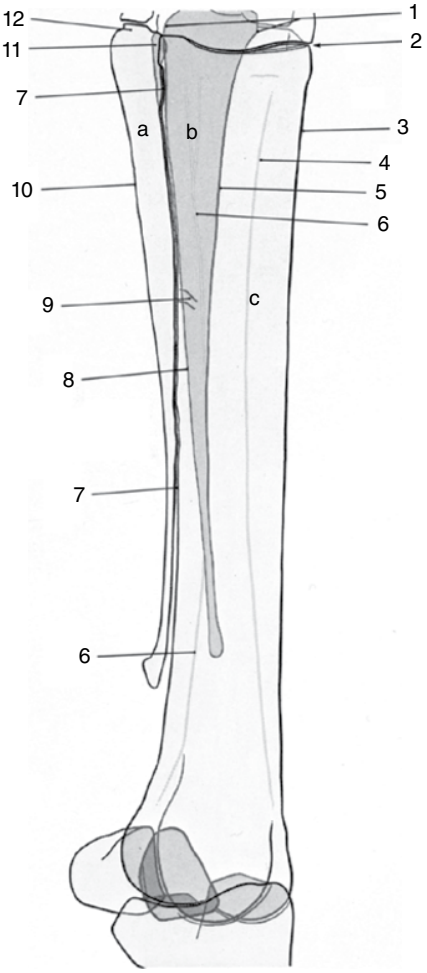


Figure 3.71.

Figure 3.71. (*Continued*) Dorsomedial to plantarolateral oblique (D45M-PlLO) projection of the metatarsus. (a) Second metatarsal bone, (b) fourth metatarsal bone, and (c) third metatarsal bone.

1. Proximal extremity (base) of the fourth metatarsal bone.
2. Articulation between the third tarsal and the third metatarsal bone.
3. Dorsolateral surface of the third metatarsal bone.
4. Endosteal surface on the dorsolateral cortex of the third metatarsal bone.
5. Dorsolateral surface of the fourth metatarsal bone.
6. Endosteal surface on the plantar medial cortex of the third metatarsal bone.
7. Interosseous space between the second and third metatarsal bones.
8. Plantaromedial surface of the fourth metatarsal bone.
9. Nutrient foramen on the plantar surface of the third metatarsal bone.
10. Plantaromedial surface of the second metatarsal bone.
11. Articulation between second and third metatarsal bones.
12. Proximal extremity (base) of the second metatarsal bone.



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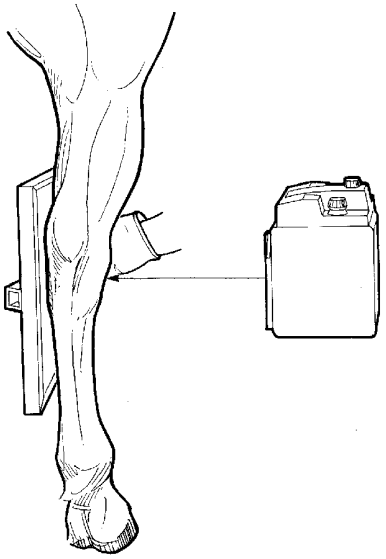


Figure 3.72.

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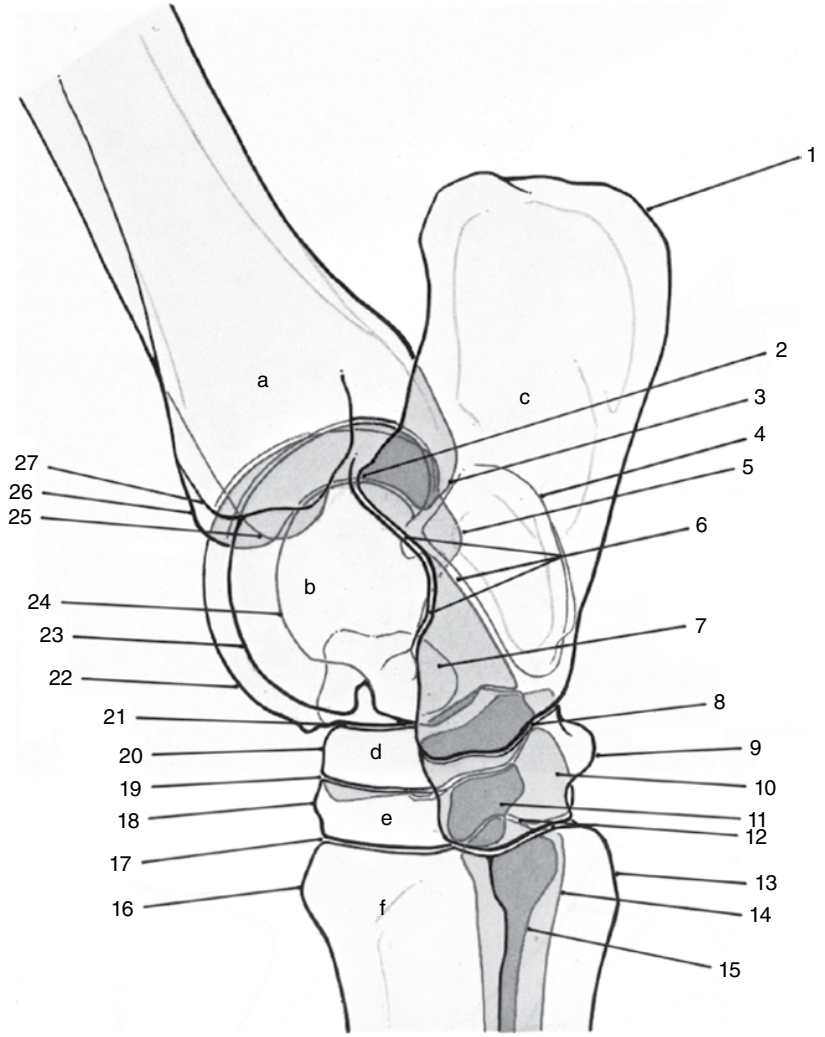


Figure 3.72. (*Continued*) Lateromedial (LM) projection of the tarsus. Depending on obliquity of the radiographic projection, the cranial borders of the malleoli and trochlear ridges may be in different relative positions. (a) Tibia, (b) talus, (c) calcaneus, (d) central tarsal bone, (e) third tarsal bone, and (f) third metatarsal bone.

1. Calcaneal tuber.
2. Coracoid process of the calcaneus.
3. Caudal intermediate part of the tibial cochlea.
4. Sustentaculum tali of the calcaneus.
5. Proximomedial tuberosity of the talus for ligamentous attachments (superficial short medial collateral ligament).
6. Articulation between the talus and the calcaneus. All of these joint surfaces may not be distinguishable on any one radiograph; they should not be mistaken for fractures.
7. Distomedial tuberosity of the talus for ligamentous attachment (dorsal tarsal ligament).
8. Articulation between the calcaneus and the fourth tarsal bone.
9. Plantar surface of the fourth tarsal bone.
10. Second tarsal bone. The first and second tarsal bones are fused and project as different densities because of superimposition.
11. First tarsal bone.
12. Plantar aspect of the tarsometatarsal articulations.
13. Plantar border of the fourth metatarsal bone. The fourth metatarsal bone is larger than the second metatarsal bone and projects on the plantar surface.
14. Plantar border of the second metatarsal bone.
15. Plantar border of the third metatarsal bone.
16. Dorsoproximal ridge on the third metatarsal bone for attachment of the tibialis cranialis.
17. Articulation between the third tarsal and the third metatarsal bone.
18. Dorsal border of the third tarsal bone.
19. Articulation between the third and central tarsal bones.
20. Dorsal border of the central tarsal bone.
21. Articulation between the central tarsal bone and the talus.
22. Medial trochlear ridge of the talus. The small bony projection on the distal part of the medial trochlear ridge is variable in size and shape and should not be mistaken for a periarticular osteophyte or any other bony abnormality.
23. Lateral trochlear ridge of the talus.
24. Depth of the trochlear groove between the trochlear ridges on the talus.
25. Distocranial border of the medial tibial malleoli.
26. Cranial intermediate part of the tibial cochlea.
27. Distocranial border of the lateral tibial malleoli.

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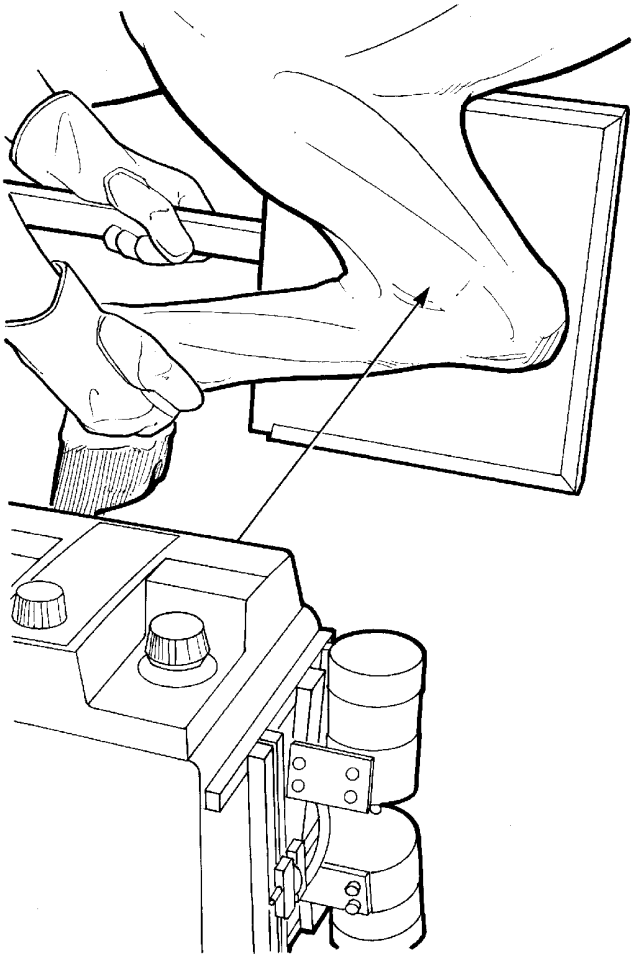


Figure 3.73.

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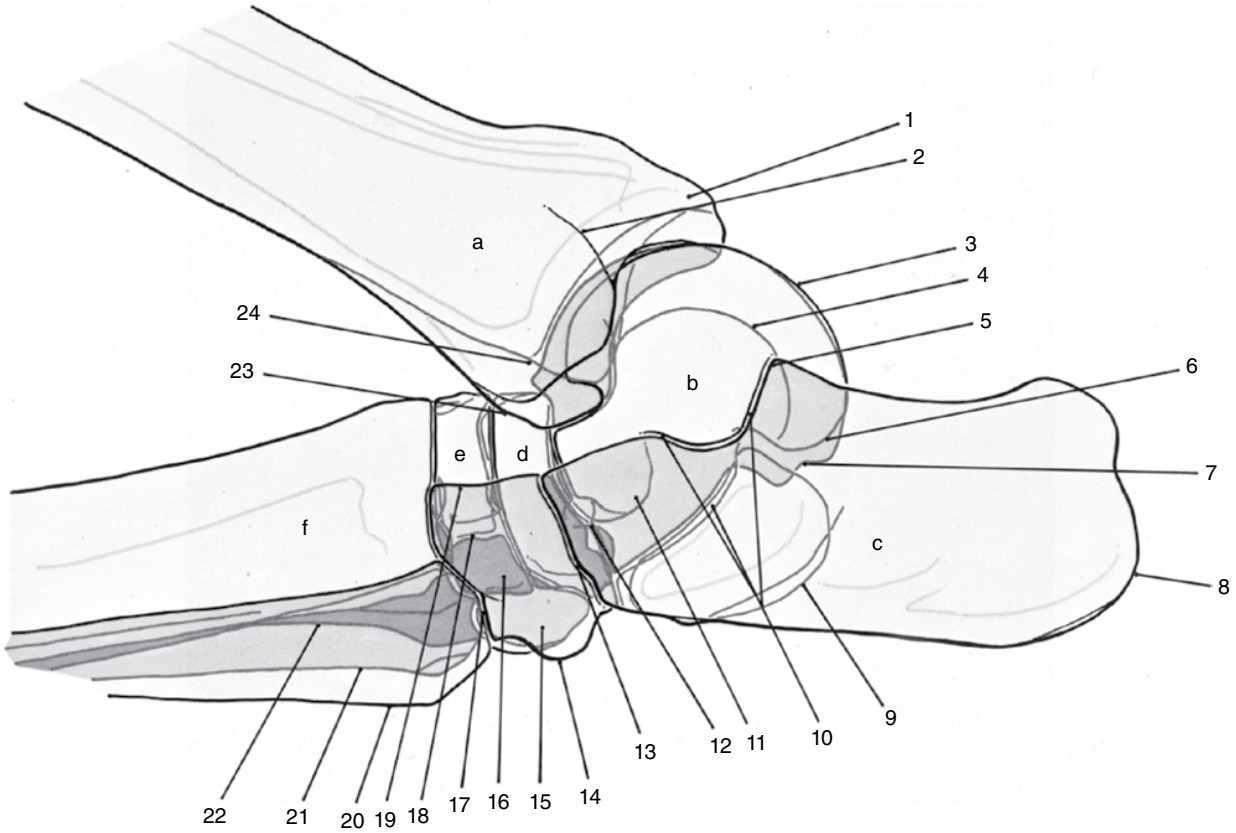


Figure 3.73. (*Continued*) Flexed lateromedial (flexed LM) projection of the tarsus. (a) Tibia, (b) talus, (c) calcaneus, (d) central tarsal bone, (e) third tarsal bone, and (f) third metatarsal bone.

1. Caudal intermediate part of the tibial cochlea.
2. Distocaudal border of the lateral tibial malleolus.
3. Superimposed medial and lateral trochlear ridges of the talus.
4. Depth of the trochlear groove between the trochlear ridges of the talus.
5. Coracoid process of the calcaneus.
6. Plantar border of the lateral trochlear ridge on the talus.
7. Plantar border of the medial trochlear ridge on the talus.
8. Calcaneal tuber.
9. Sustentaculum tali.
10. Articulations between the talus and calcaneus, the visualization of which depends on the projection angle.
11. Distomedial tuberosity of the talus for ligamentous attachment (dorsal tarsal ligament).
12. Articulation between the talus and the central tarsal bone.
13. Articulation between the calcaneus and the fourth tarsal bone.
14. Plantar border of the fourth tarsal bone.
15. Second tarsal bone. The first and second tarsal bones are fused.
16. First tarsal bone.
17. Tarsometatarsal articulation.
18. Junction between the first and third tarsal bones, which is not always distinctly visible. Do not mistake the junction, when present, for a slab fracture.
19. Dorsal border of the fourth tarsal bone.
20. Plantar border of the fourth metatarsal bone.
21. Plantar border of the second metatarsal bone.
22. Plantar border of the third metatarsal bone. The superimposed plantar border of the third metatarsal bone and the dorsal borders of the second and fourth metatarsal bones may produce pseudolongitudinal fracture lines.
23. Cranial intermediate part of the tibial cochlea.
24. Cranial border of the medial tibial malleolus.

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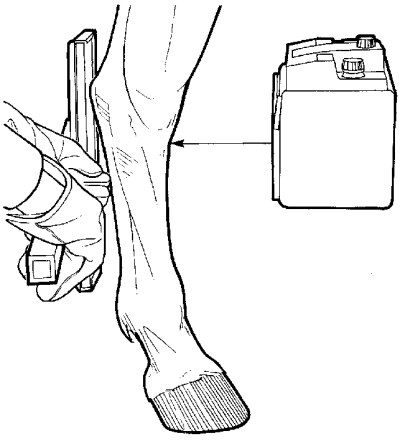


Figure 3.74.

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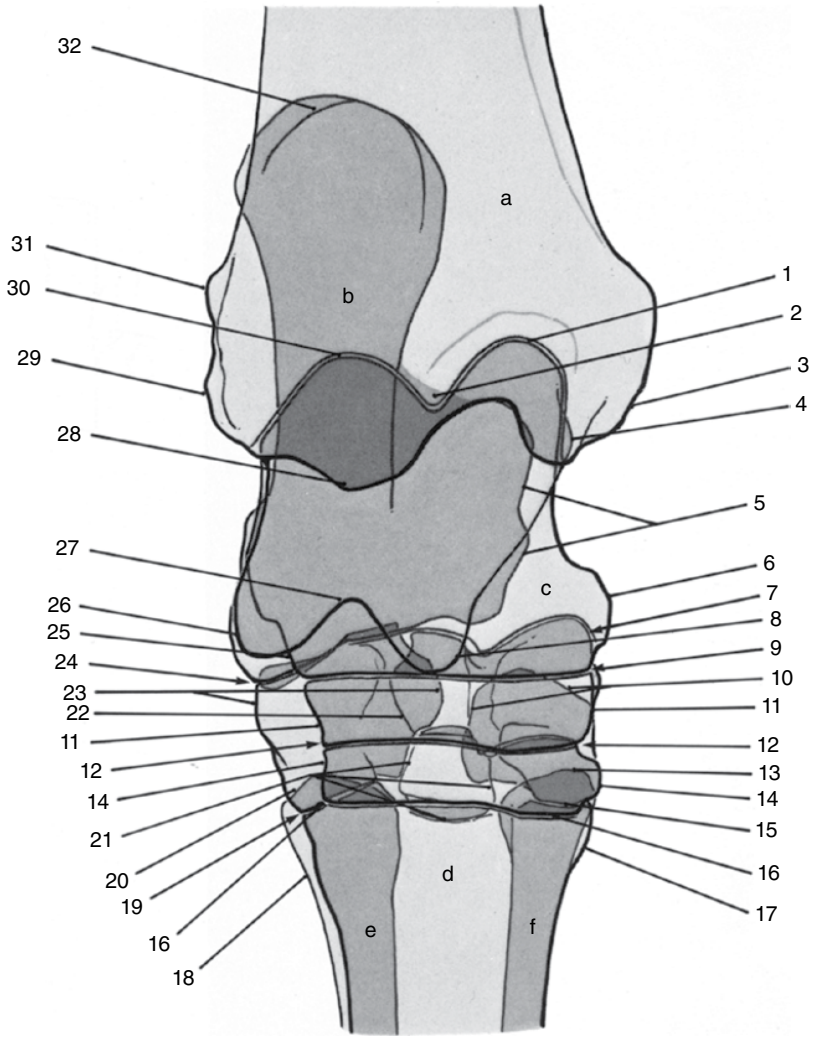


Figure 3.74. (*Continued*) Dorsoplantar (DPl) projection of the tarsus. The bones and joints of the tarsus are irregular and have complex shapes; thus, their appearance can vary with little change in projection angle. Therefore, careful observation is necessary when the tarsus is being evaluated radiographically. (a) Tibia, (b) calcaneus, (c) talus, (d) third metatarsal bone, (e) fourth metatarsal bone, and (f) second metatarsal bone.

1. Articulation between the medial trochlear ridge of the talus and the medial cochlear groove of the tibia (tarsocrural joint).
2. Intermediate ridge of the tibial cochlea.
3. Medial malleolus on the distal tibia, where the medial collateral ligaments attach.
4. Proximal medial tuberosity on the talus, where the superficial short medial collateral ligament attaches.
5. Sustentaculum tali.
6. Distal medial tuberosity on talus for ligamentous attachment.
7. Plantar medial aspect of the talocalcaneocentral (proximal intertarsal) joint.
8. Distomedial border of the medial trochlear ridge on the talus.
9. Dorsomedial aspect of the talocalcaneal central (proximal intertarsal) joint. The difference in position of the plantar and dorsal aspects of this joint is caused by the curved contour of the articular surfaces in the proximal intertarsal joint.
10. Medial and lateral borders of the second tarsal bone.
11. Medial and lateral borders of the central tarsal bone.
12. Articulation between the third and central tarsal bones (tarsodistal or distal intertarsal joint).
13. Proximal border of the second metatarsal bone superimposed on the first and third tarsal bones.
14. Medial and lateral borders of the third tarsal bone.
15. Articulation between the second metatarsal and the fused first and second tarsal bones.
16. Medial and lateral aspects of the articulation between the third tarsal and the third metatarsal bone.
17. Proximomedial border of the second metatarsal bones.
18. Proximolateral border of the fourth metatarsal bone.
19. Articulation between the fourth tarsal and the fourth metatarsal bone (tarsometatarsal joint).
20. Proximal border of the fourth metatarsal bone superimposed over the fourth tarsal bone.
21. Bony prominence on the plantar surface of the third tarsal bone.
22. Prominence on the plantar surface of the central tarsal bone.
23. Lateral and medial borders of the fourth tarsal bone.
24. Articulation of the fourth tarsal bone and the calcaneus (calceneoquartal or proximal intertarsal joint).
25. Distal lateral border of the talus.
26. Lateral trochlear ridge of the talus.
27. Groove between medial and lateral trochlear ridges on the talus.
28. Caudal aspect of the intermediate ridge on the tibial cochlea.
29. Cranial part of the lateral malleolus.
30. Articulation between the lateral trochlear ridge on the talus and the lateral tibial cochlear groove in the tarsocrural (tibiotarsal) joint.
31. Caudal part of the lateral malleolus.
32. Calcaneal tuber.

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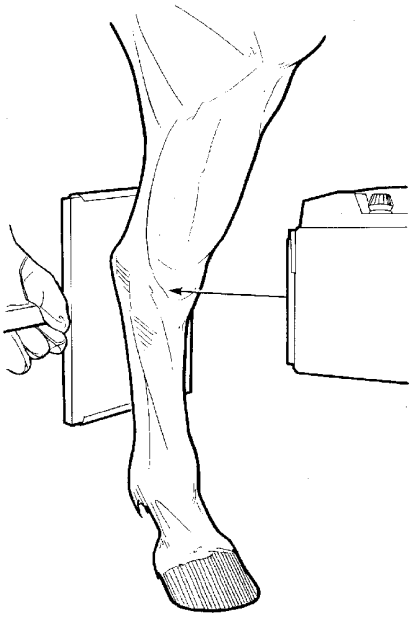


Figure 3.75.

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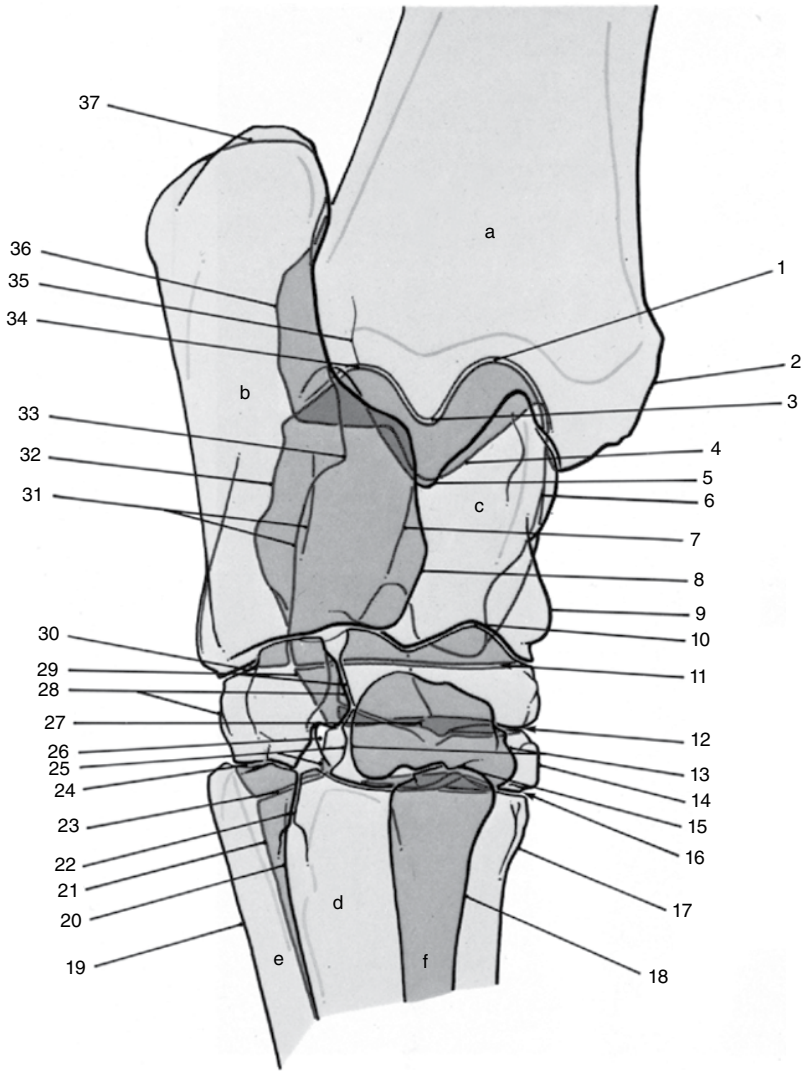
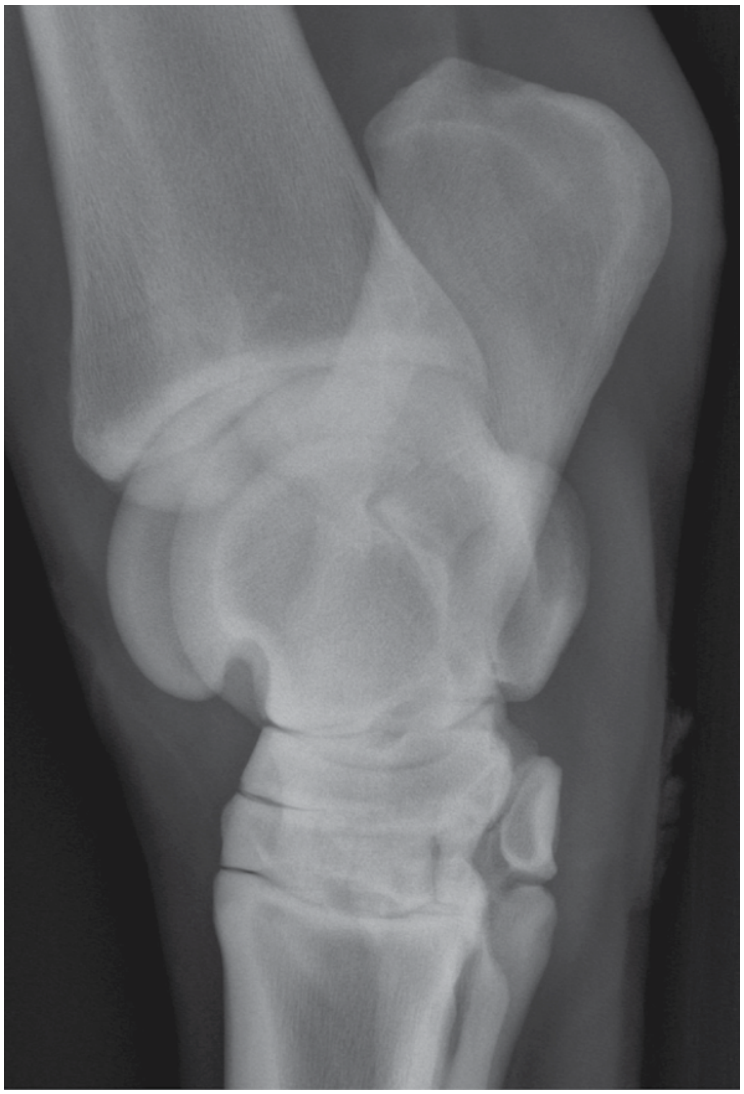


Figure 3.75. (*Continued*) Dorsolateral to plantaromedial oblique (D35L-PIMO) projection of the tarsus. (a) Tibia, (b) calcaneus, (c) talus, (d) third metatarsus, (e) fourth metatarsal bone, and (f) second metatarsal bone.

1. Articulation between the medial trochlear ridge of the talus and the medial tibial cochlear groove in the tarsocrural (tibiotarsal) joint.
2. Medial malleolus.
3. Articulation between the intermediate ridge of the tibial cochlea and the groove in the trochlea tali in the tarsocrural joint.
4. Cranial aspect of the intermediate ridge on the tibial cochlea.
5. Caudal aspect of the intermediate ridge on the tibial cochlea.
6. Medial trochlear ridge of the talus.
7. Lateral trochlear ridge of the talus.
8. Dorsomedial border of the sustentaculum tali.
9. Distomedial tuberosity of the talus for ligamentous attachment.
10. Plantaromedial aspect of the talocalcaneocentral (proximal intertarsal) joint.
11. Dorsomedial aspect of the talocalcaneocentral (proximal intertarsal) joint.
12. Dorsomedial aspect of the articulations between the central and third tarsal bone (centrodistal or distal intertarsal joint).
13. Medial and lateral borders of the fused first and second tarsal bones.
14. Prominent ridge for ligamentous attachment on the dorsomedial surface of the third tarsal bone.
15. Articulation between the fused first and second tarsal bones and the second metatarsal bone.
16. Dorsomedial aspect of the articulation between the metatarsal bone and the third metatarsal bone (tarsometatarsal joint).
17. Dorsomedial border of the third metatarsal bone.
18. Dorsomedial border of the fourth metatarsal bone.
19. Plantarolateral border of the fourth metatarsal bone.
20. Interosseous space between the third and fourth metatarsal bone.
21. Lateral border of the third metatarsal bone dorsal to the fourth metatarsal bone.
22. Articulation between the third and fourth metatarsal bone.
23. Articulation between the fourth tarsal and the third metatarsal bone.
24. Articulation between the fourth tarsal and the fourth metatarsal bone.
25. Plantarolateral and plantaromedial borders of the third tarsal bone.
26. Vascular tarsal canal, which encloses the perforating tarsal artery and vein and a deep perineal nerve branch. It may be more or less apparent on this projection, depending on the projection angle.
27. Nonvascular area between the central and the third tarsal bones. These nonarticular areas may simulate subchondral bone lysis and must be differentiated from erosion by their location.
28. Plantarolateral and dorsomedial borders of the fourth tarsal bone.
29. Plantarolateral and plantaromedial borders of the central tarsal bone.
30. Articulation between the calcaneus and the fourth tarsal bone (calcaneoquarteral or proximal intertarsal joint).
31. Borders of the sinus tarsi, which is a space between the calcaneus and talus that appears as a more or less lucent region.
32. Plantarolateral border of the sustentaculum tali.
33. Coracoid process of the calcaneus.
34. Articulation between the lateral trochlear ridge of the talus and the lateral tibial cochlear groove in the tarsocrural joint (tibiotarsal joint).
35. Cranial part of the lateral malleolus.
36. Caudal part of the lateral malleolus.
37. Calcaneal tuber.

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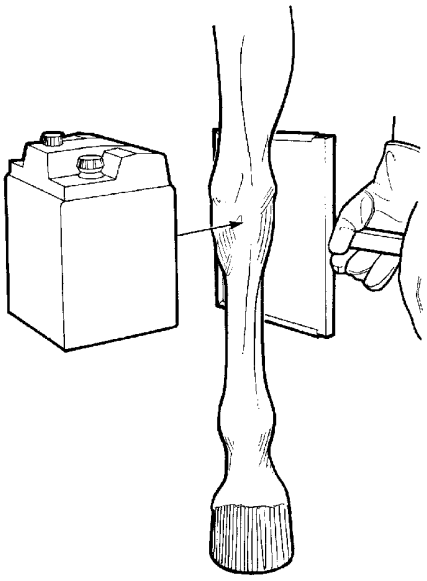


Figure 3.76.

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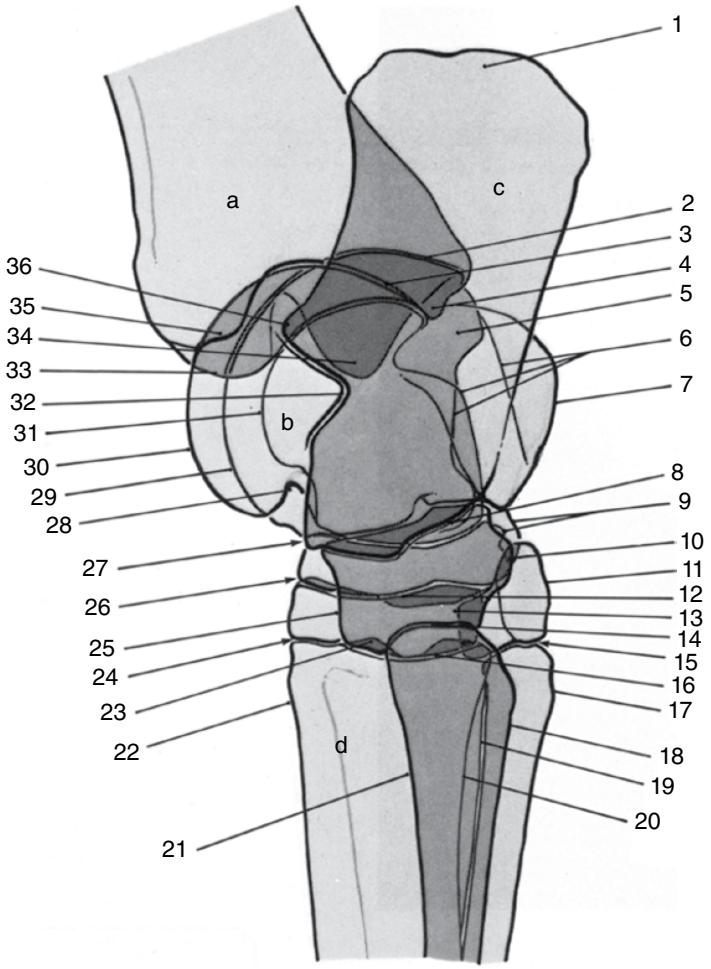
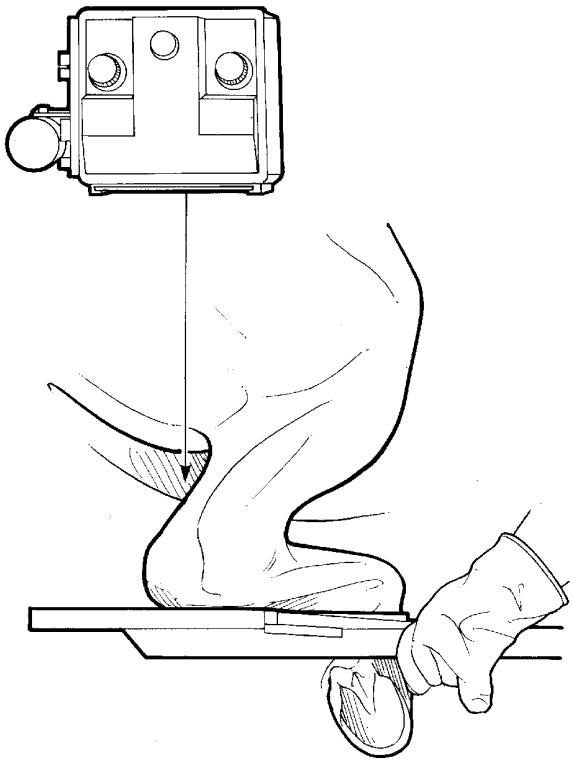


Figure 3.76. (*Continued*) Dorsomedial to plantarolateral (D55MPILO) projection of the tarsus. (a) Tibia, (b) talus, (c) calcaneus, and (d) third metatarsal bone.

1. Calcaneal tuber
2. Articulation between the medial trochlear ridge of the talus and the medial tibial cochlear groove of the tarsocrural joint.
3. Articulation between the lateral trochlea of the talus and the lateral tibial cochlear groove of the tarsocrural joint.
4. Caudal aspect of the intermediate ridge of the tibial cochlea.
5. Proximomedial tuberosity of the talus.
6. Plantar borders of the talus superimposed on the calcaneus.
7. Plantar border of the sustentaculum tali.
8. Nonarticular depression between the talus and the central tarsal bone. These nonarticular depressions, which appear more of less distinct depending on the angle of the X-ray beam projection, may simulate subchondral bone lysis.
9. Medial and lateral plantar borders of the central tarsal bone.
10. Plantar border of the fourth tarsal bone.
11. Plantar border of the fused first and second tarsal bone.
12. Area of nonarticular depression between the central and third tarsal bones.
13. Junction between the fused first and second tarsal bones and the medial plantar border of the third tarsal bone.
14. Articulation between the fourth tarsal and the fourth metatarsal bone.
15. Articulation between the fused first and second tarsal bone and the second metatarsal bone.
16. Articulation between the fourth tarsal bone and the third metatarsal bone.
17. Plantar border of the second metatarsal bone.
18. Plantar border of the fourth metatarsal bone.
19. Interosseous space between the second and third metatarsal bones.
20. Plantar border of the third metatarsal bone.
21. Dorsolateral border of the fourth metatarsal bone.
22. Dorsolateral border of the third metatarsal bone.
23. Nonarticular depressions in the adjacent surface of the third tarsal and the third metatarsal bone.
24. Dorsolateral aspect of the articulation between the third tarsal and the third metatarsal bone (tarsometatarsal joint).
25. Dorsolateral border of the fourth tarsal bone.
26. Dorsolateral aspect of the articulation between the central and the third tarsal bone (centrodistal or distal intertarsal bone).
27. Dorsolateral aspect of the talocalcaneal central (proximal intertarsal joint).
28. Notch at the distal aspect of the lateral trochlear ridge on the talus.
29. Medial trochlear ridge of the talus.
30. Lateral trochlear ridge of the talus.
31. Depth of the groove between the medial and lateral trochlear ridges on the talus.
32. Articulation between the talus and the calcaneus (talocalcaneal articulation), the visibility of which depends on the X-ray beam projection.
33. Cranial aspect of the intermediate ridge on the tibial cochlea.
34. Medial malleolus superimposed over the talus and calcaneus.
35. Lateral malleolus superimposed over the intermediate tibial cochlear ridge and the lateral trochlear ridge of the talus.
36. Coracoid process of the calcaneus.

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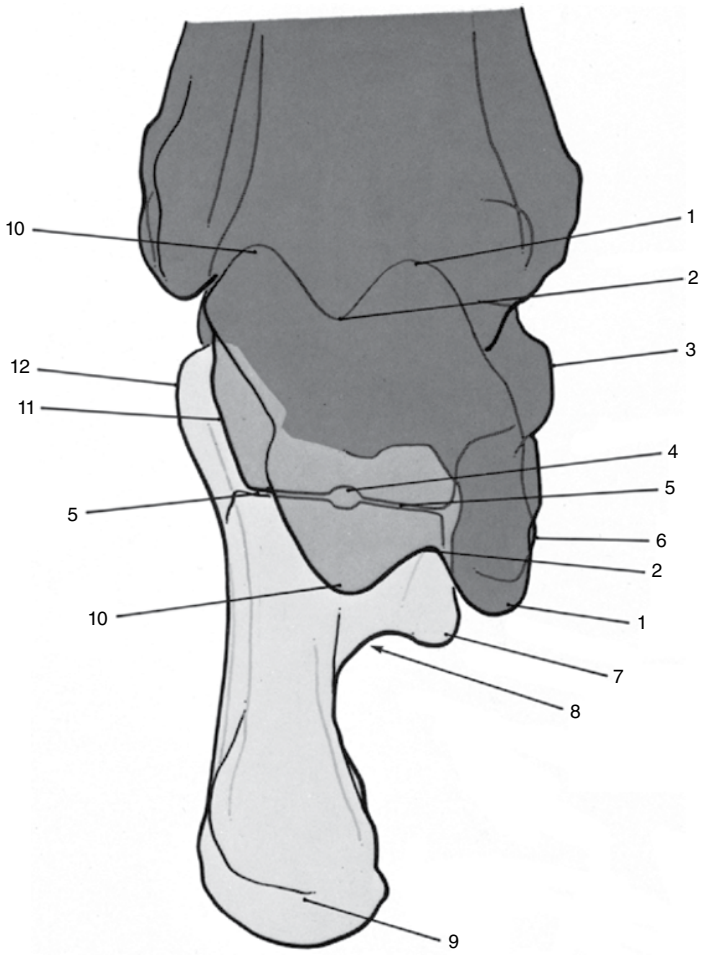


Figure 3.77. Flexed dorsoplantar (flexed DP) projection of the tarsus.

1. Medial trochlear ridge on the talus.
2. Groove between the medial and lateral trochlear ridges of the talus.
3. Distomedial tuberosity of the talus.
4. Nonarticular depressions between the talus and calcaneus. This opening communicates with the sinus tarsi.
5. Medial and lateral aspects of the articulation between the talus and calcaneus.
6. Proximomedial tuberosity of the talus.
7. Sustentaculum tali.
8. Tarsal groove for the deep digital flexor principal tendon.
9. Calcaneal tuber.
10. Lateral trochlear ridge on the talus.
11. Areas of attachment of the lateral collateral ligament on the talus.
12. Areas of attachment of the lateral collateral ligament on the calcaneus.

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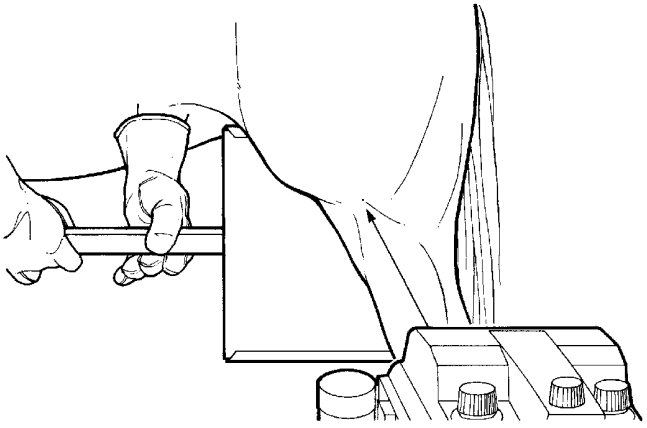


Figure 3.78.

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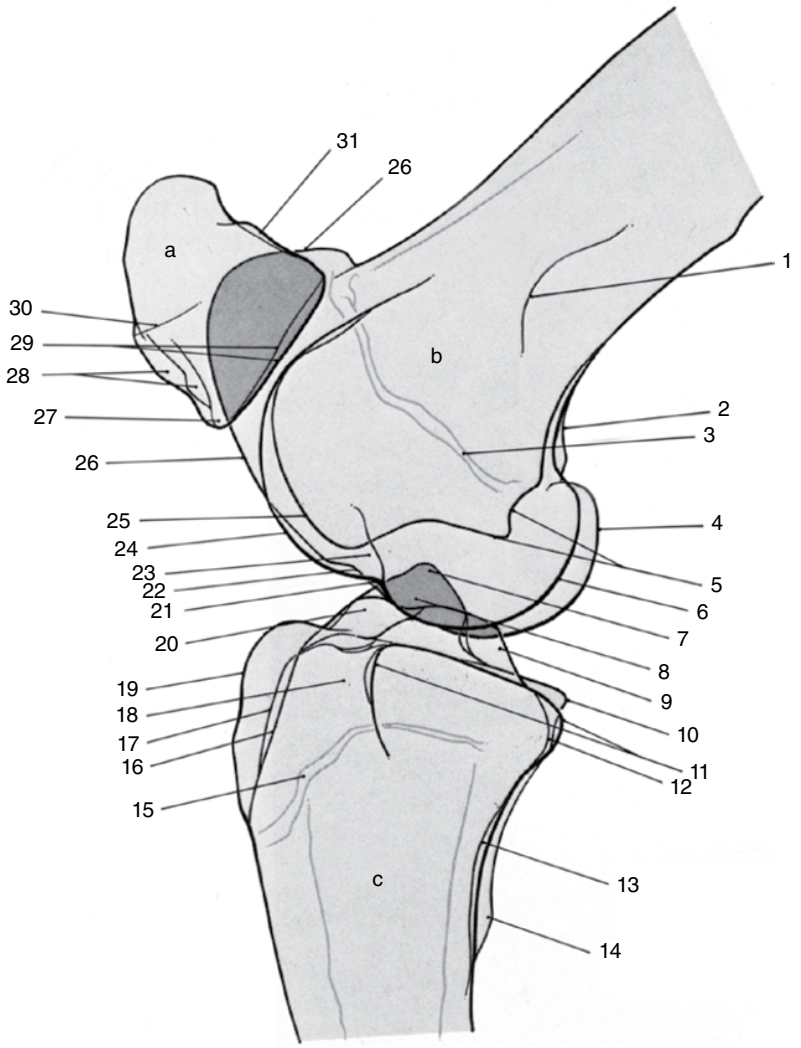


Figure 3.78. (*Continued*) Lateromedial (LM) projection of the femorotibial and femoropatellar joints (stifle joint). (a) Patella, (b) femur, and (c) tibia.

1. Supracondyloid fossa.
2. Medial supracondyloid tuberosity.
3. Distal femoral growth plate.
4. Medial femoral condyle.
5. Intercondyloid fossa.
6. Lateral femoral condyle.
7. Medial tubercle on the intercondyloid eminence of the tibia.
8. Central intercondylar area.
9. Medial part of the articular surface on the lateral tibial condyle.
10. Medial tibial condyle.
11. Lateral tibial condyle.
12. Popliteal notch.
13. Concavity of the popliteal incisure.
14. Tubercle on the caudal medial surface of the tibia.
15. Growth plate on the proximal tibia.
16. Groove for the medial patellar ligament.
17. Medial part of the tibial tuberosity.
18. Extensor sulcus.
19. Lateral part of the tibial tuberosity.
20. Lateral tubercle on the intercondyloid eminence of the tibia.
21. Ridge connecting the lateral trochlear ridge and the lateral condyle on the femur.
22. Ridge connecting the medial trochlear ridge and the medial condyle on the femur.
23. Extensor fossa.
24. Lateral femoral trochlear ridge.
25. Compact bone in the femoral trochlear between the lateral and medial trochlear ridge.
26. Medial femoral trochlear ridge.
27. Apex of the patella.
28. Areas of ligamentous attachment on the cranial surface of the patella.
29. Articular surfaces of the patella.
30. Edge of the medial articular surface and medial border of the patella.
31. Base of the patella.

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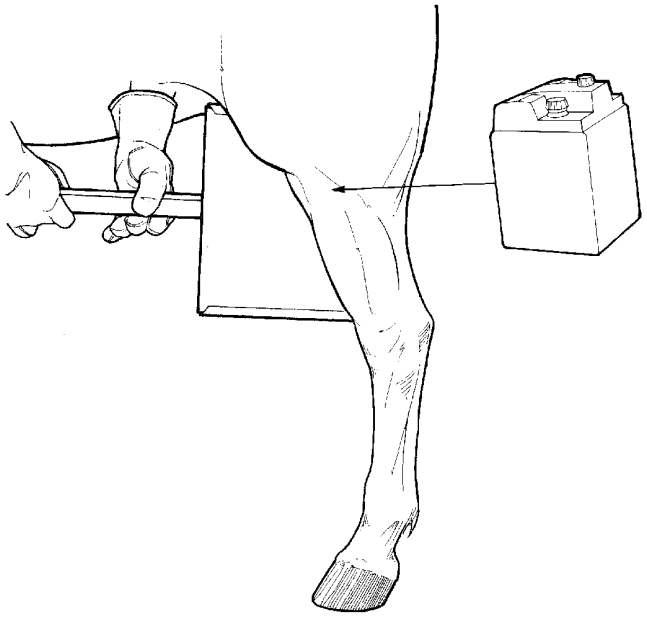


Figure 3.79.

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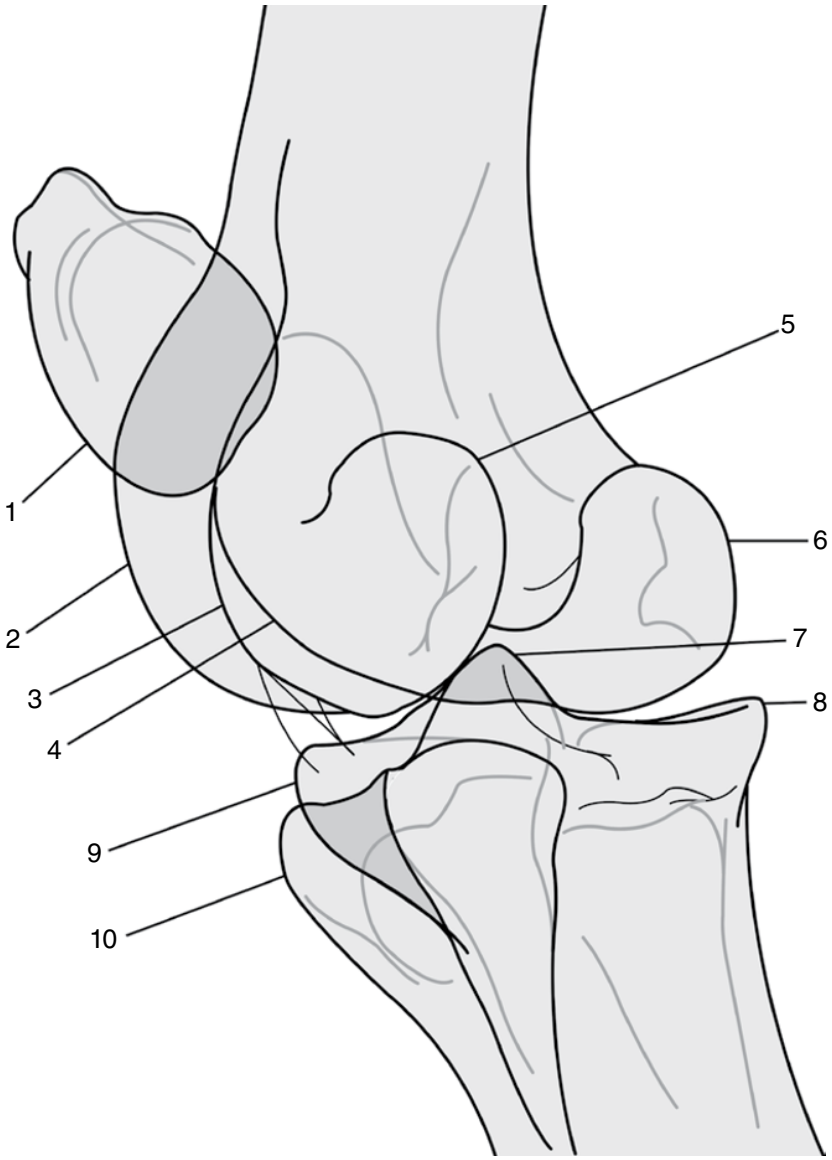


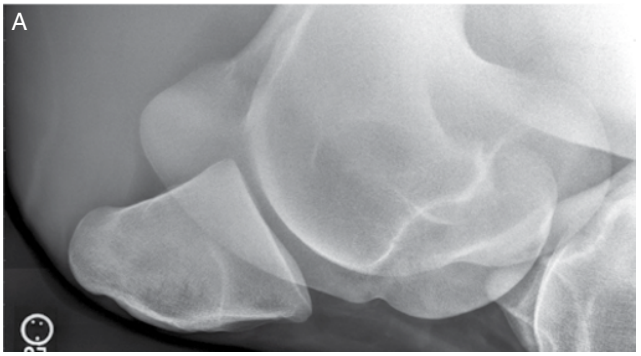
Figure 3.79. (*Continued*) Laterocaudal to craniomedial oblique (L45Ca-CrMO) projection of the stifle joint. From a straight lateromedial position, the primary X-ray beam is directed parallel to the ground with a 45° caudal angle and is centered on the caudal aspect of the stifle joint for a different approach in the evaluation of the medial femoral condyle. This projection also shows a large area of the weight-bearing articular surface of the medial femoral condyle free of superimposition with the lateral condyle and can be an alternative to the flexed projection. However, in both projections, the medial femoral condyle is projected differently, and one lesion may be seen in one and not the other.

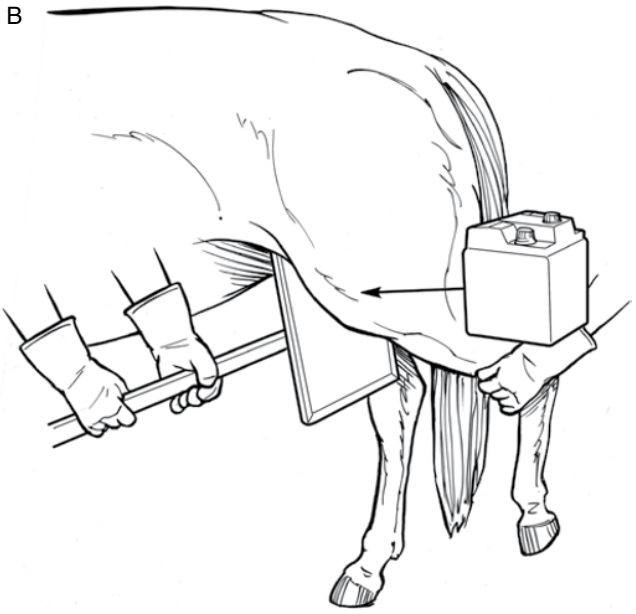
1. Patella.
2. Lateral femoral trochlear ridge.
3. Intercondylar groove.
4. Medial femoral trochlear ridge.
5. Lateral femoral condyle.
6. Medial femoral condyle.
7. Medial tubercle on the intercondyloid eminence of the tibia.
8. Medial tibial condyle.
9. Lateral tibial condyle.
10. Tibial tuberosity.

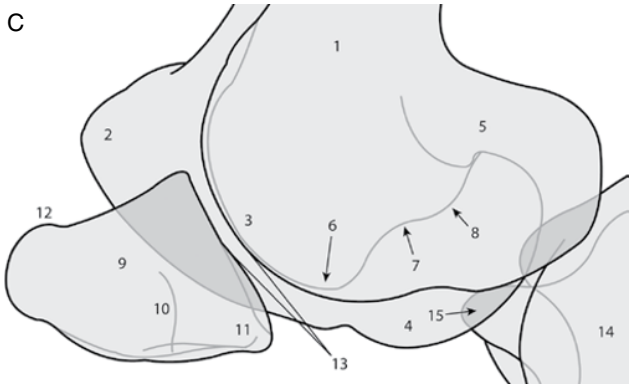
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Figure 3.80. (A, B, and C). Flexed lateromedial (flexed L0-20Cr-CaMO) projection of the femorotibial and femoropatellar joints (stifle). Projections (A and C) show the articular surface of the medial femoral condyle free of superimposition with other osseous structures. This projection can be performed in two different ways. The limb can be flexed and abducted and the X-ray beam directed parallel to the ground with a 0° to 20° laterocranial obliquity (B); if the limb cannot be abducted, an approximately 10° distal obliquity (L0-20Cr10Di-PrMO) should be added to the X-ray beam direction.

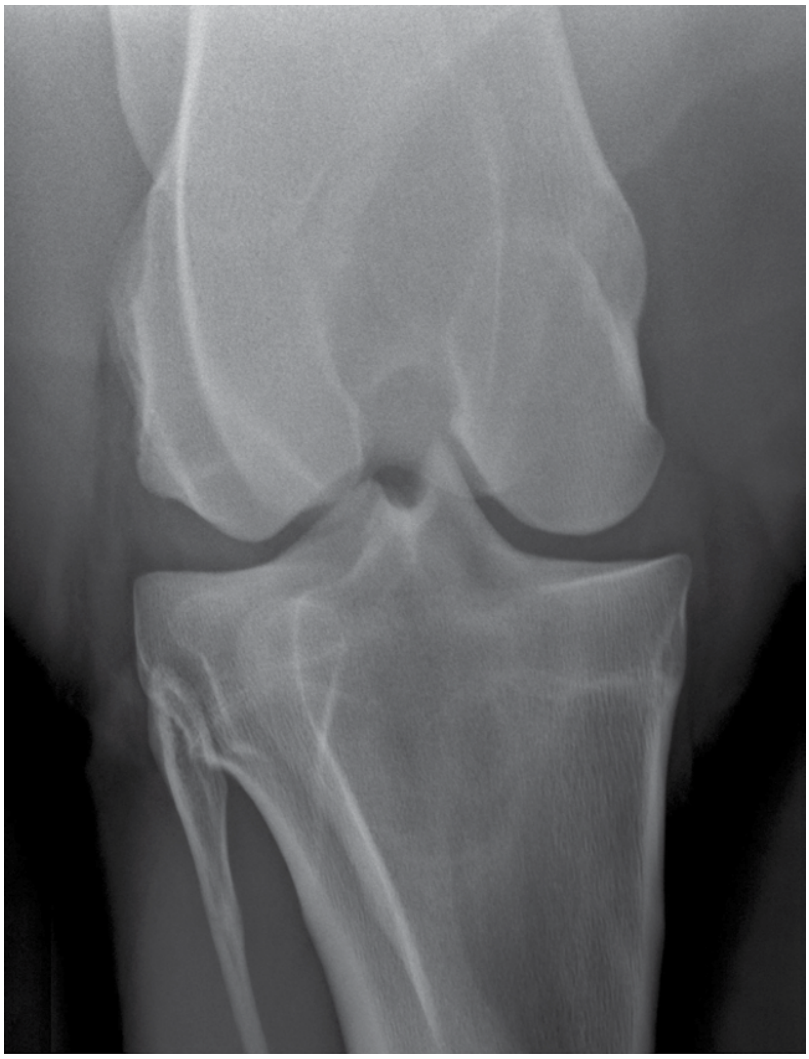
1. Femur
2. Medial trochlea
3. Lateral trochlea
4. Medial condyle
5. Lateral condyle
6. Intertrochlear groove
7. Extensor fossa
8. Intercondylar fossa
9. Patella
10. Medial articular surface
11. Apex
12. Base
13. Articular surfaces
14. Tibia
15. Medial tibial eminence







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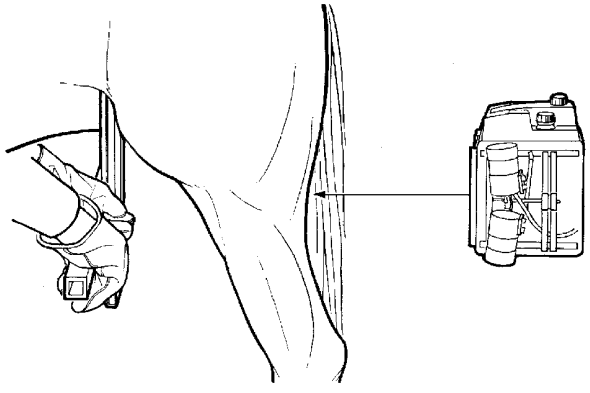


Figure 3.81.

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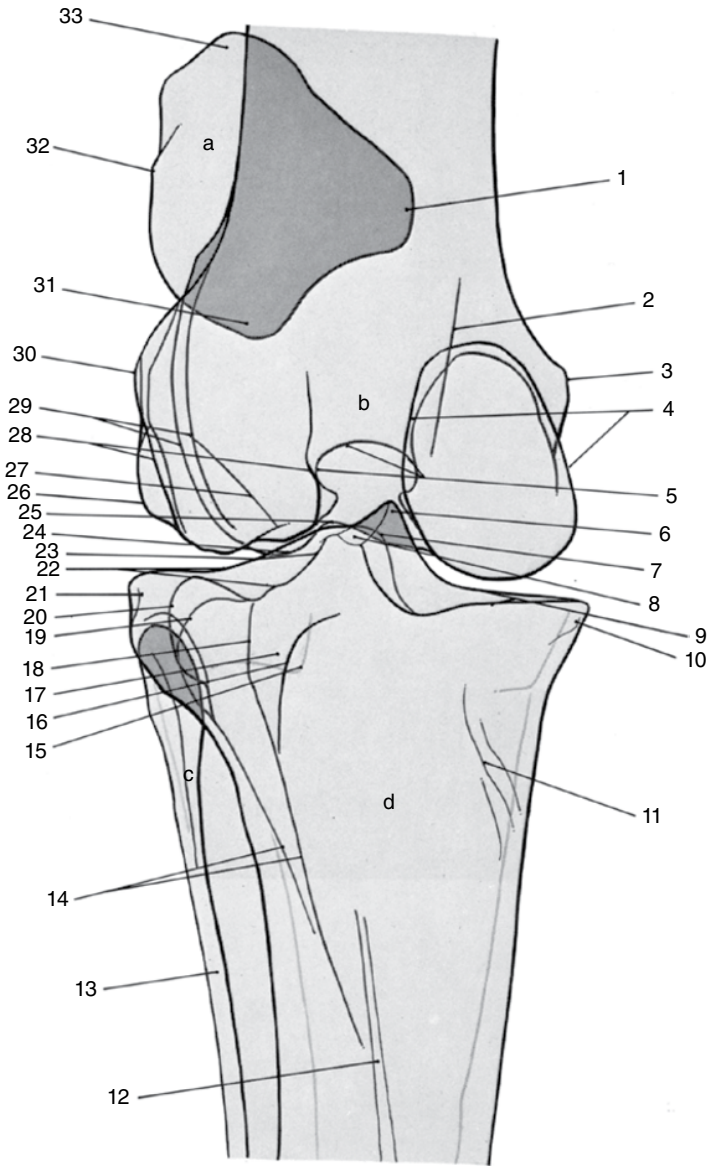
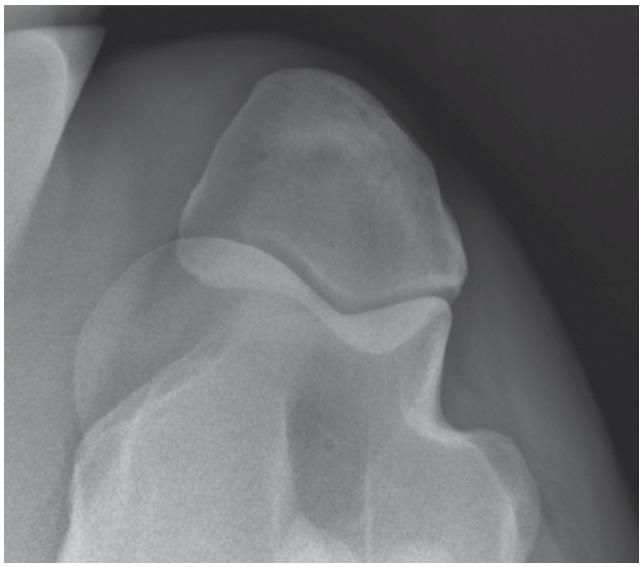
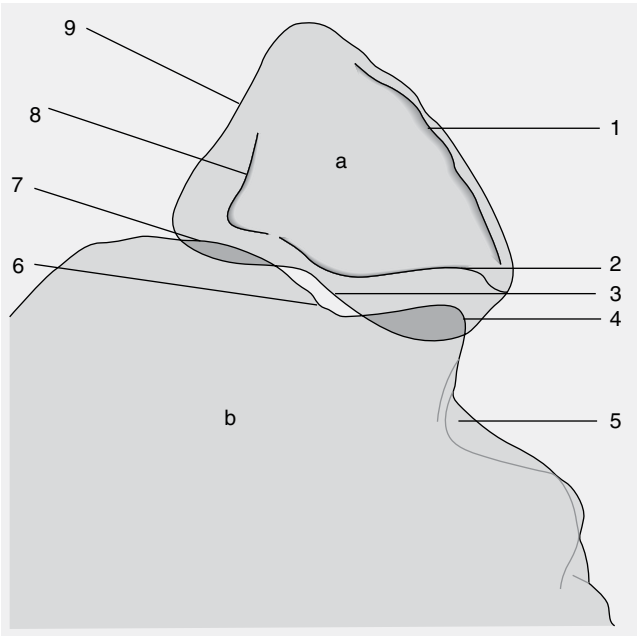


Figure 3.81. (*Continued*) Caudocranial (CaCr) projection of the femorotibial and femoropatellar joints (stifle). The entire patella may or may not be seen depending on the X-ray exposure. The patella is normally located to the lateral side of the distal aspect of the femur. The fibula may be a complete bone (as here), but it is usually rudimentary with only the proximal part present or with one or two transverse lines that give the mistaken appearance of fractures. (a) Patella, (b) femur, (c) fibula, and (d) tibia.

1. Medial angle of the patella. A large cartilaginous process extends from the medial angle of the patella and is not visible radiographically.
2. Lateral border of the medial trochlear ridge on the distal femur, the visibility of which depends on the X-ray exposure.
3. Medial epicondyle for ligamentous attachment.
4. Medial and lateral borders of the medial femoral condyle.
5. Intercondyloid fossa on the caudal aspect of the distal femur.
6. Medial tubercle on the intercondylar eminence of the proximal tibia.
7. Lateral tubercle on the intercondylar eminence of the proximal tibia.
8. Central intercondylar area.
9. Cranial and caudal borders of the articular surface of the medial tibial condyle.
10. Medial tibial condyle.
11. Tubercle on the caudal medial tibial surface.
12. Muscular lines on the caudal tibial surface.
13. Fibula.
14. Tibial crest.
15. Bony margin of the extensor sulcus.
16. Medial part of the tibial tuberosity.
17. Groove between the medial and lateral parts of the tibial tuberosity for the medial patellar ligament.
18. Medial border of the lateral part of the tibial tuberosity.
19. Cranial aspect of the lateral tibial condyle.
20. Lateral proximal border of the lateral part of the tibial tuberosity.
21. Caudal aspect of the lateral tibial condyle.
22. Cranial and caudal articular surfaces on the lateral tibial condyle.
23. Articular surface on the medial part of the lateral tibial condyle.
24. Distal aspect of the lateral trochlear ridge on the femur.
25. Distal aspect of the groove between the distal femoral trochlear ridges.
26. Lateral femoral epicondyle for ligamentous attachment.
27. Bony borders of the extensor fossa on the distal femur.
28. Lateral and medial borders of the lateral femoral condyle.
29. Lateral trochlear ridge on the distal extremity of the femur.
30. Proximolateral border of the lateral femoral condyle.
31. Apex of the patella.
32. Lateral angle and the patella.
33. Base of the patella.

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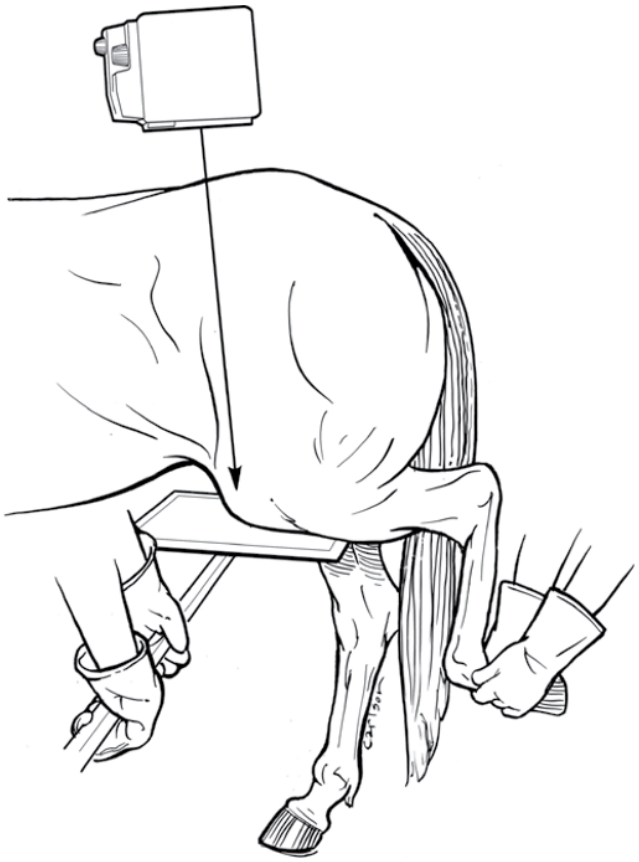
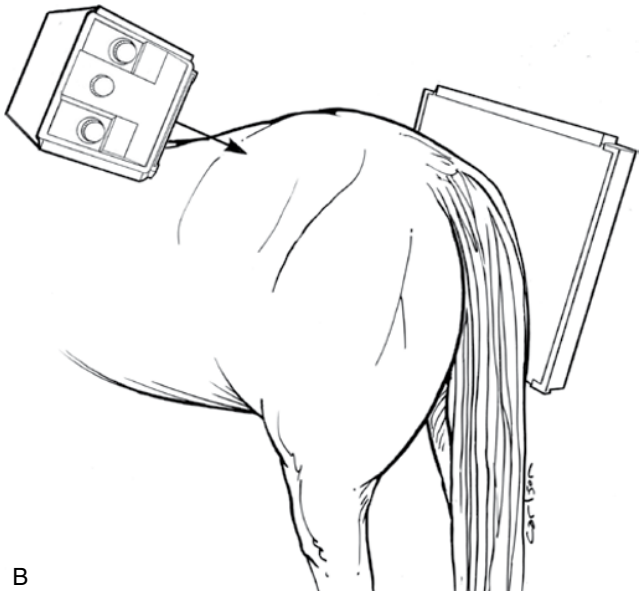


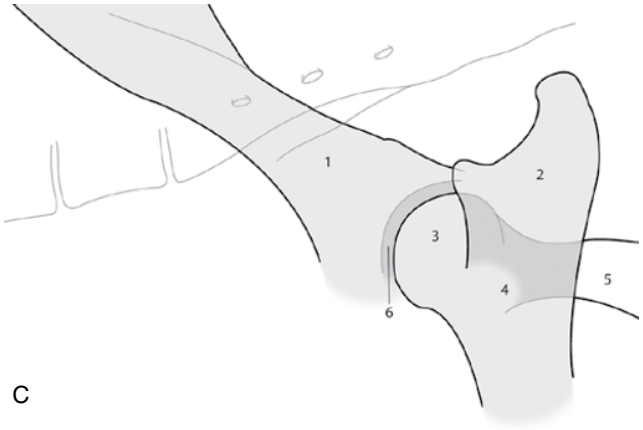
Figure 3.82. Cranioproximal to craniodistal oblique (CrPR-DiCrO) projection of the patella. (a) Patella and (b) femur.
1. Dorsal surface and area of ligament attachment on the patella.
2. Distal articular surface on the patella.
3. Proximal articular surface on the patella.
4. Lateral femoral trochlear ridge.
5. Extensor fossa.
6. Trochlear groove between the medial and lateral trochlear ridges.
7. Medial femoral trochlear ridge.
8. Distal medial border of the patella.
9. Proximal medial border of the patella.

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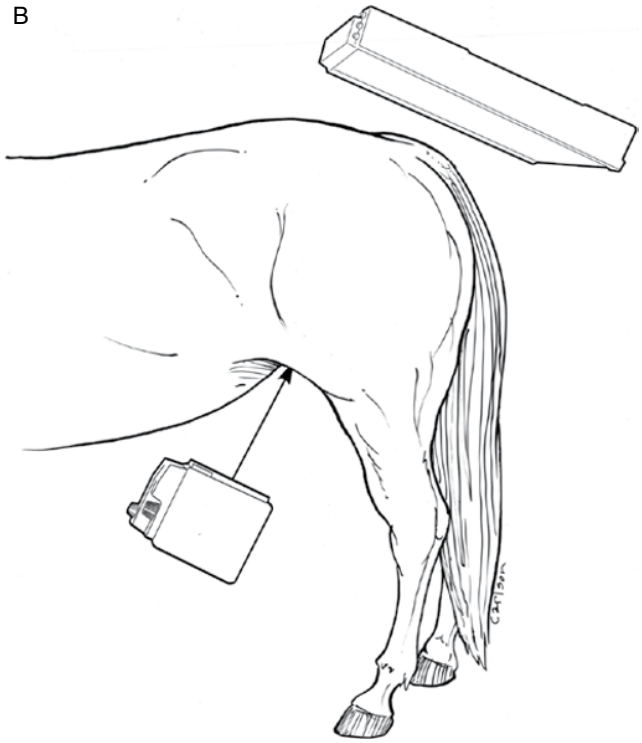
Figure 3.83. (A, B, and C). Dorsal 30° lateral to lateroventral oblique view of the coxofemoral joint.
1. Ilium
2. Greater trochanter of femur
3. Femoral head
4. Femoral neck
5. Ischium
6. Acetabulum











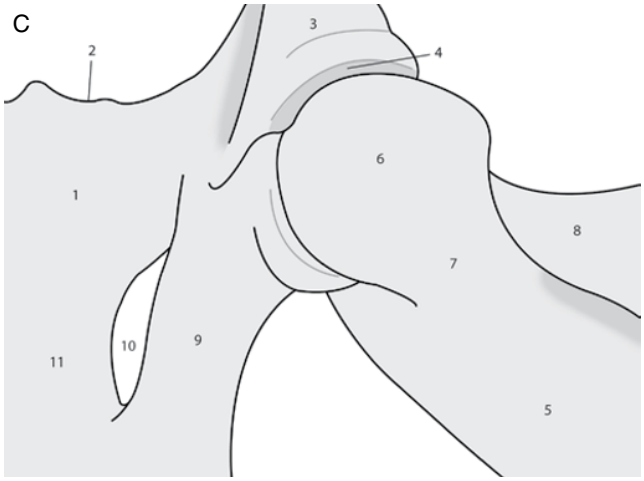
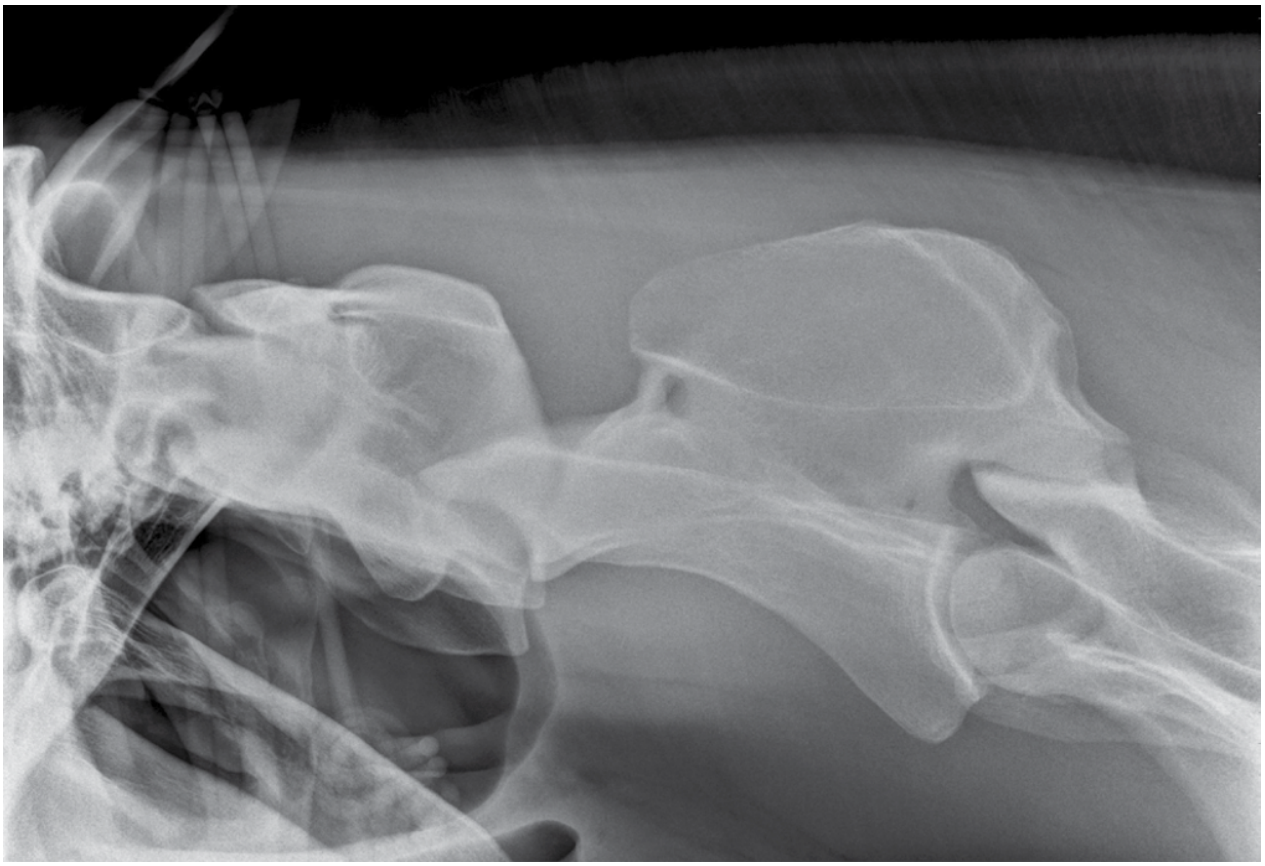


Figure 3.84. (A, B, and C) Ventral 15–30° medial to dorsolateral projection of the pelvis.

1. Pubis
2. Cranial border of pubis
3. Ilium
4. Acetabular rim
5. Femur
6. Femoral head
7. Femoral neck
8. Greater trochanter
9. Ischium
10. Obturator foramen
11. Sacrum

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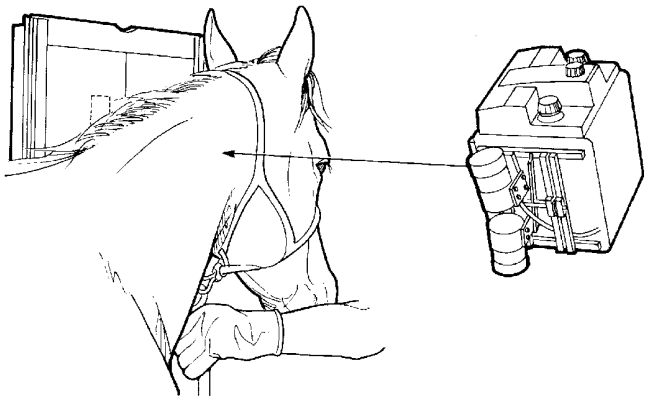


Figure 3.85.

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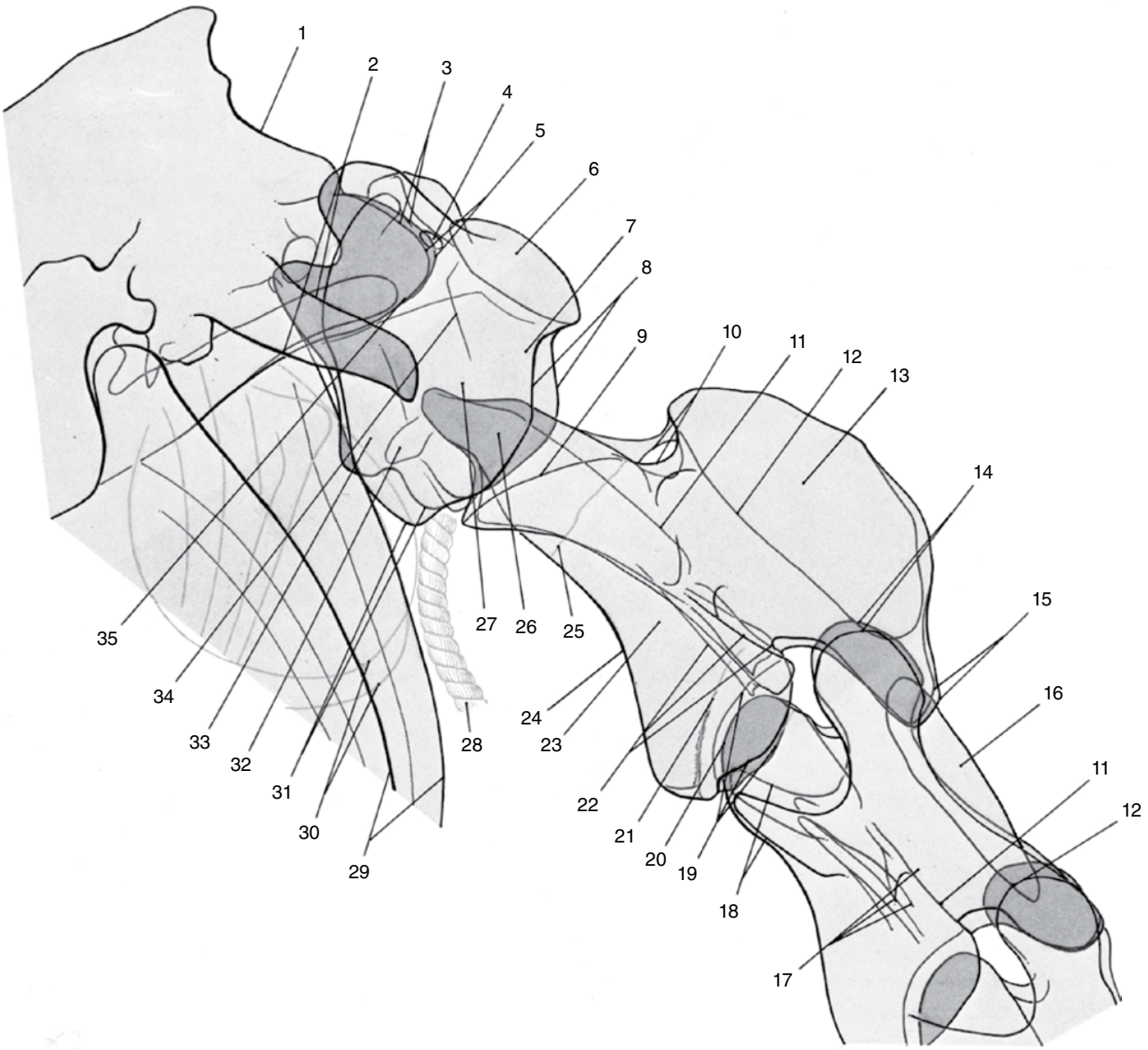


Figure 3.85. (*Continued*) Right-to-left lateral (Rt-LeL) projection of the cranial cervical vertebral column (C1–C3).

1. Occipital bone.
2. Jugular process.
3. Dorsal surface of the right and left occipital condyles.
4. Right and left margins of the lateral foramen of the atlas.
5. Caudal borders of the occipital condyle (atlantooccipital articulation).
6. Dorsal tubercle of the atlas.
7. Atlas.
8. Caudal margins of the articular fovea.
9. Cranial articular process of the axis.
10. Margins of the lateral vertebral foramen.
11. Ventral margin of the vertebral column.
12. Dorsal margin of the vertebral column.
13. Dorsal spinous process of the axis.
14. Right and left cranial articular fovea of C3.
15. Caudal articular fovea of the axis.
16. Dorsal spinous process of C3.
17. Base of the transverse processes of C3.
18. Transverse process of C3.
19. Concave margins of the caudal extremity of the axis.
20. Convex cranial extremity of C3.
21. Caudal growth plate of the axis.
22. Multiple linear opacities produced by the wide bases on the right and left transverse processes of the axis.
23. Axis.
24. Ventral border of the axis.
25. Cranial growth plate of the axis.
26. Dens of the axis.
27. Atlas.
28. Shadow produced by braided rope used for alter.
29. Right and left rami of the mandible.
30. Right and left ventral margins of the guttural pouches.
31. Right and left ventrocaudal margins of the atlas.
32. Shadow caused by margins of the transverse foramen.
33. Base of the wings of the atlas.
34. Shadow formed by the concavity of the atlantal fossa.
35. Right and left caudal margins of the occipital condyles.

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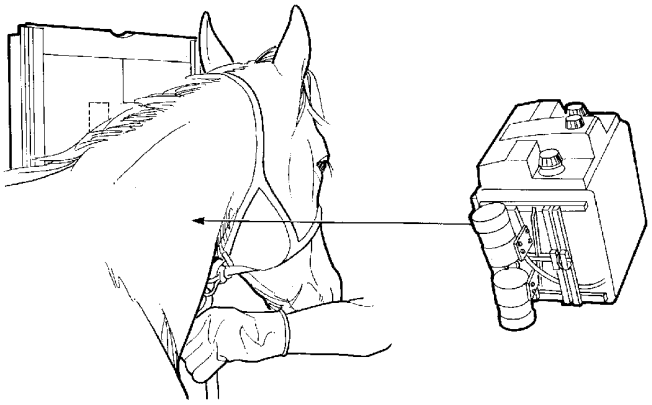


Figure 3.86.

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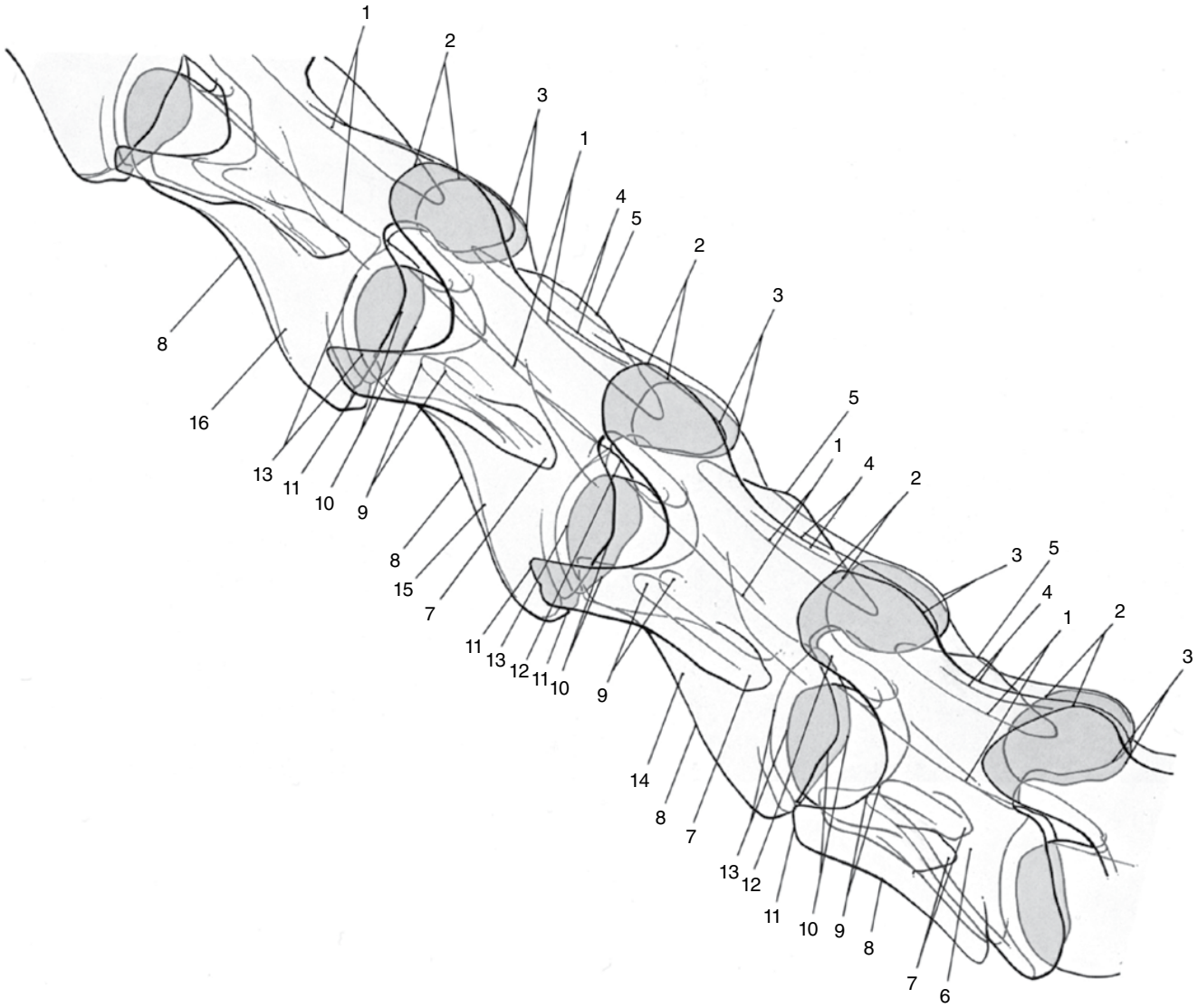
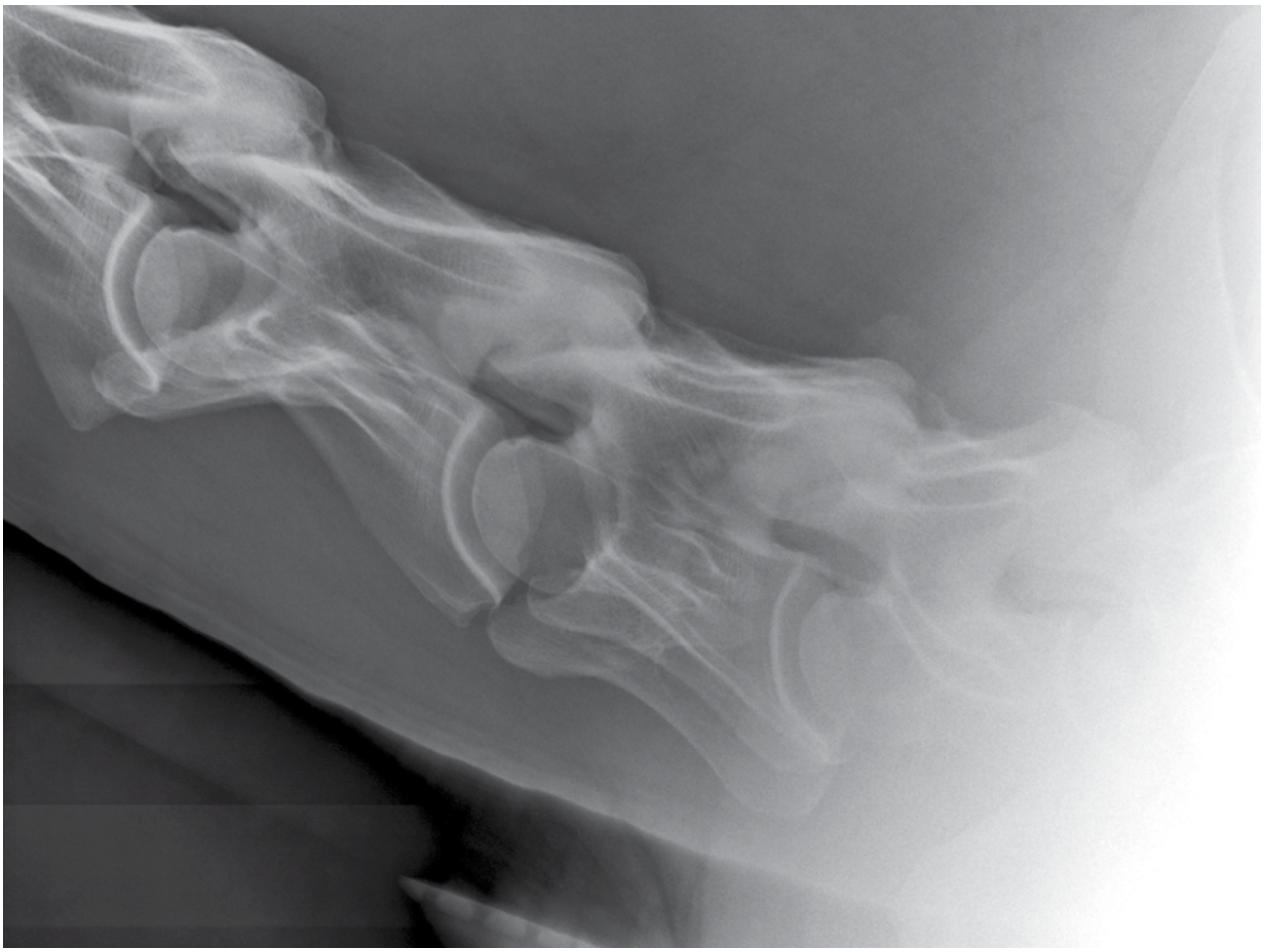


Figure 3.86. (*Continued*) Right-to-left lateral (Rt-LeL) projection of the mid-cervical spine (C3–C6).

1. Dorsal and ventral borders of the vertebral canal.
2. Right and left cranial articular processes.
3. Right and left caudal articular processes.
4. Shadows formed by vertebral arches on either side of the spinous processes.
5. Dorsal spinous processes of C4, C5, and C6.
6. C6.
7. Bases of lateral transverse vertebral processes.
8. Ventral borders of the vertebrae.
9. Bases of the transverse processes, which also form the ventral and dorsal margins of transverse foramina.
10. Caudal extremities of the vertebrae.
11. Transverse processes.
12. Dorsal tuberculums.
13. Concave borders of the caudal extremities.
14. C5.
15. C4.
16. C3.

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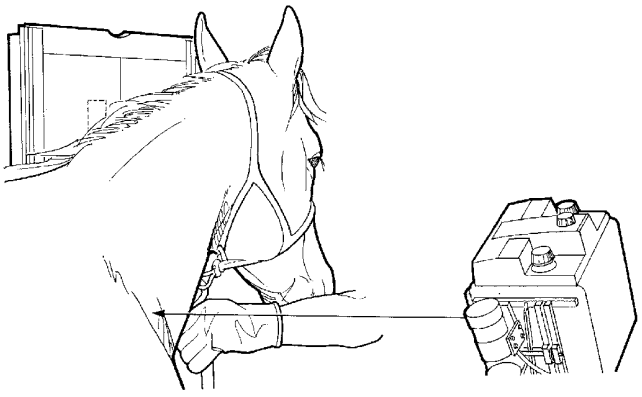


Figure 3.87.

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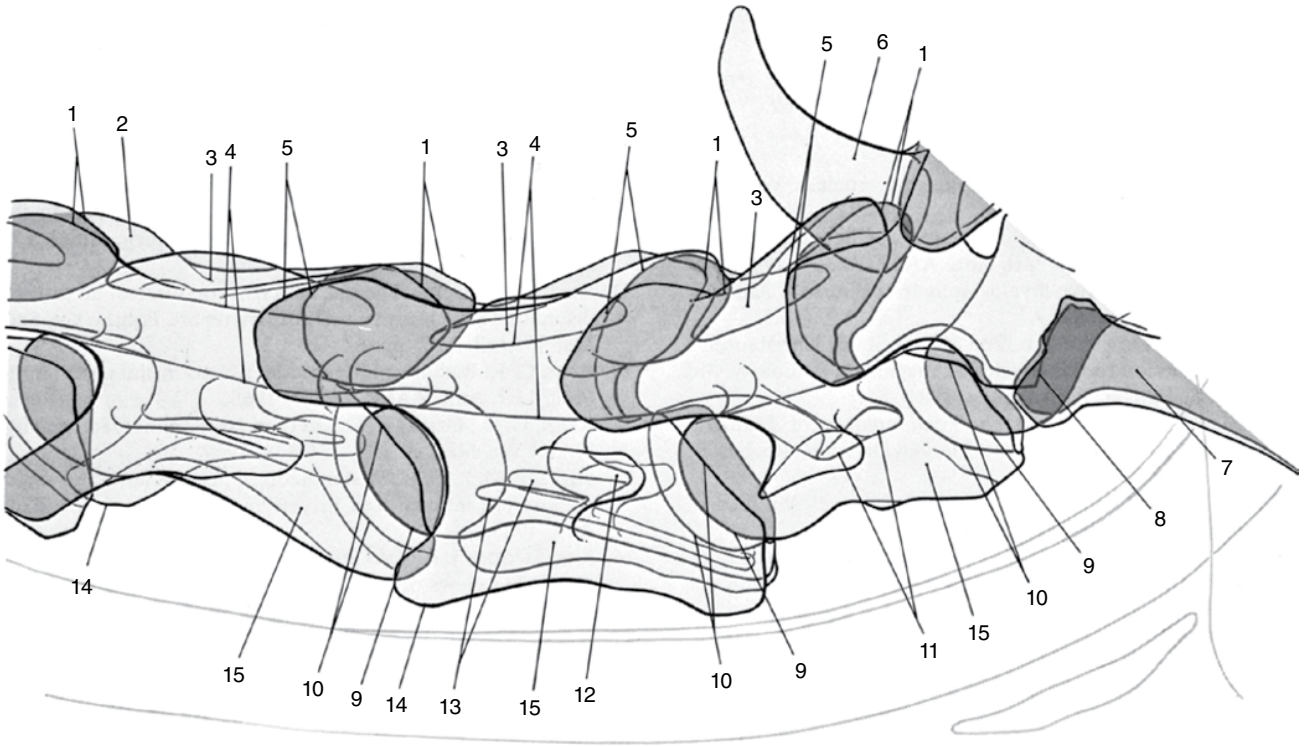
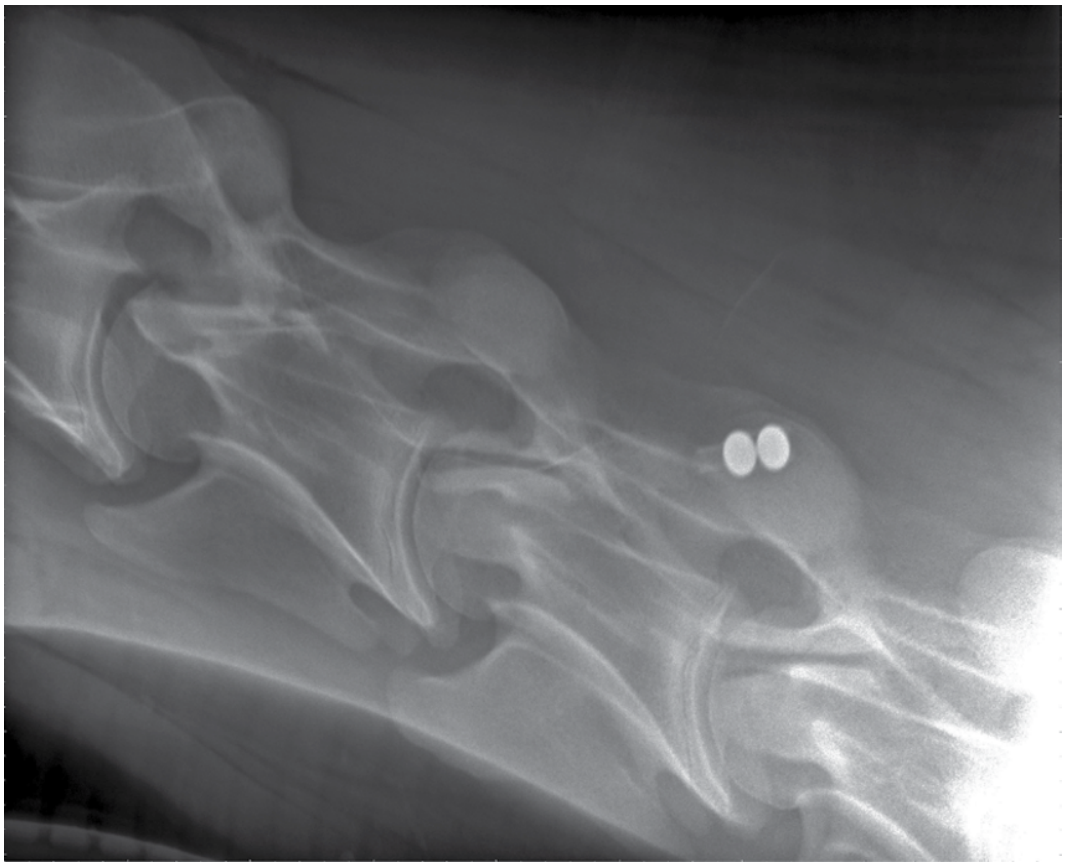


Figure 3.87. (*Continued*) Right-to-left lateral (Rt-LeL) projection of the caudal cervical spine (C5–C7).

1. Caudal articular processes.
2. Vertebral lamina on either side of the spinous process.
3. Dorsal spinous processes and dorsal laminae.
4. Dorsal and ventral borders of the vertebral canal.
5. Cranial articular border.
6. Dorsal spinous process of T1.
7. First rib.
8. Tubercle of the first rib.
9. Cranial extremities of the vertebrae.
10. Caudal extremities of the vertebrae.
11. Bases of the transverse processes.
12. Shadow of the transverse foramen.
13. Bases of the trifid transverse processes of C6.
14. Cranial part of the transverse processes of C5 and C6.
15. C5, C6, and C7.

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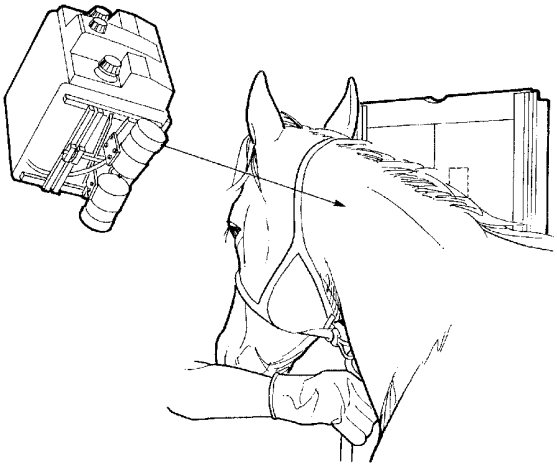


Figure 3.88.

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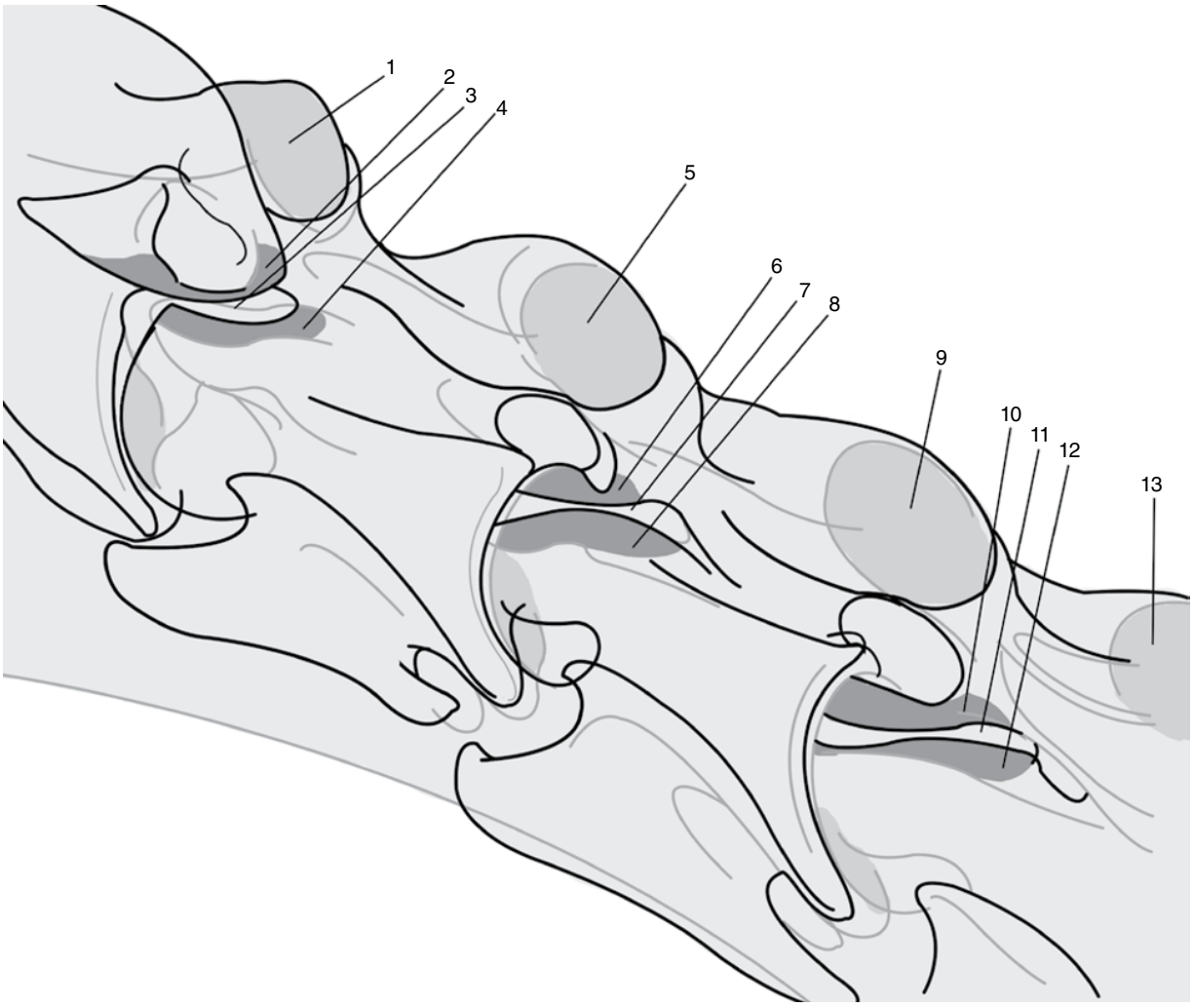
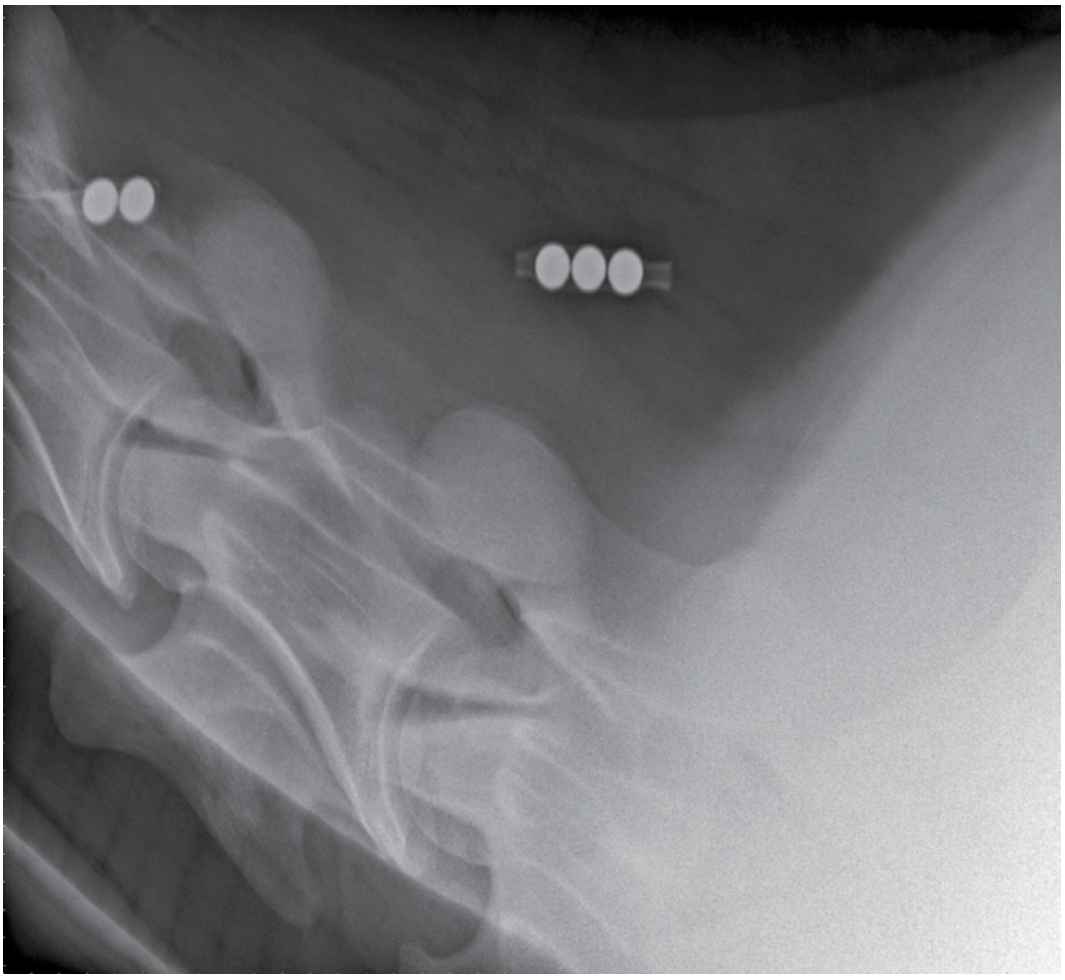


Figure 3.88.

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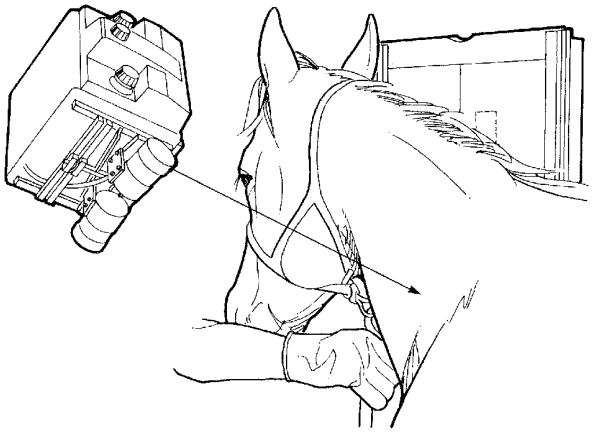


Figure 3.88. (*Continued*)

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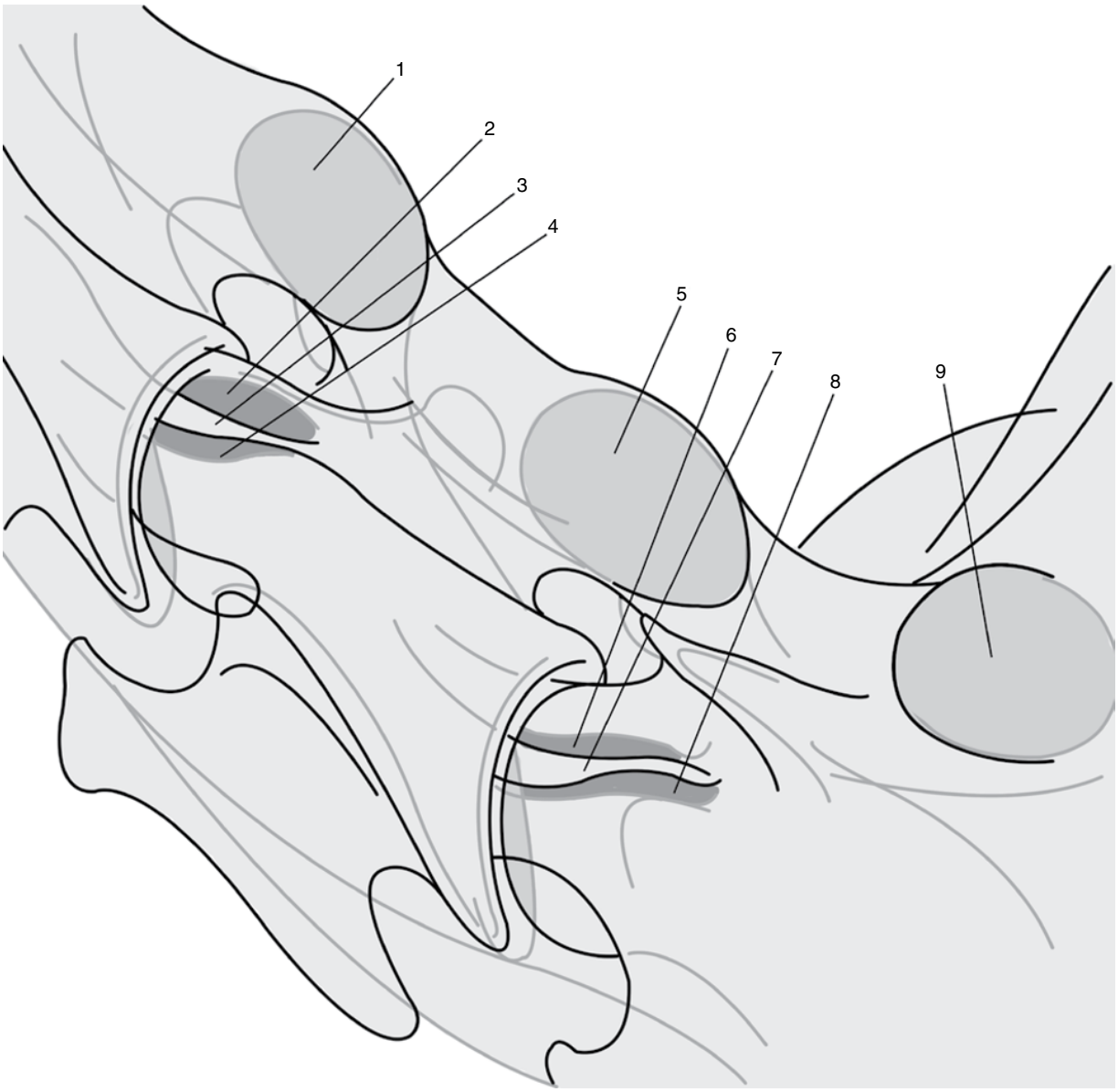


Figure 3.88. (*Continued*) Laterodorsal to lateroventral oblique (La45D-LaVO) projections of the cervical vertebral facets. (A) Cranial to mid-cervical spine. (B) Mid-caudal cervical spine. These projections are used to free the articular facets from superimposition with each other with the goal of having more discernible facet borders and detecting unilateral or bilateral changes.

a. Cranial to mid-cervical spine.
   1. Superimposed articular facets of C2 and C3.
   2. Caudal articular facet of C2.
   3. Joint space between articular facets of caudal C2 and cranial C3.
   4. Cranial articular facet of C3.
   5. Superimposed articular facets of C3–C4.
   6. Caudal articular facet of C3.
   7. Joint space between articular facets of caudal C3 and cranial C4.
   8. Cranial articular facet of C4.
   9. Superimposed articular facets of C4 and C5.
   10. Caudal articular facet of C4.
   11. Joint space between articular facets of caudal C4 and cranial C5.
   12. Cranial articular facet of C5.
   13. Superimposed articular facets of C5 and C6.

b. Caudal cervical spine.
   1. Superimposed articular facets of C5 and C6.
   2. Caudal articular facet of C5.
   3. Joint space between articular facets of caudal C5 and cranial C6.
   4. Caudal articular facet of C6.
   5. Superimposed articular facets of C6 and C7.
   6. Caudal articular facet of C6.
   7. Joint space between articular facets of caudal C6 and cranial C7.
   8. Cranial articular facet of C7.
   9. Superimposed articular facets of C7 and T1.

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ULTRASOUND

W. Rich Redding

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INTRODUCTION

Over the last several decades, ultrasound technology has dramatically improved. The current technology found in linear array ultrasound systems has progressed rapidly and are now very well suited for musculoskeletal examinations. Many of these high-end systems have 14–18-MHz linear tendon probes and 8–10-MHz microconvex probes with variable focusing capabilities with multiple frequencies available in each probe. In addition, mainframe ultrasound platforms have been reduced to the size of notebook-sized computers as well as advancements made in the miniaturization of electronics have reduced the quality differences between portable and stationary technologies. Ultrasonography is now considered the imaging modality of choice to evaluate soft tissue injuries in the horse.

Ultrasonography is a two-dimensional real-time imaging technique that utilizes the transfer and propagation of sound waves into soft tissue.[4,55,61,70,73,74,80] Ultrasound is defined as sound above the audible range. Ultrasound waves behave as classic sound waves that operate at frequencies spanning 1–22 MHz. These sound waves are mechanical waves that require some sort of medium to allow the waves to form and travel. The propagating medium determines how fast the sound wave travels, how easily the waves can be formed, and how well the traveling waves can remain together. Ultrasound machines produce sound waves of longitudinal orientation in which the elements of the medium are compressed and rarefied. The distance between the start of one cycle of compression and rarefaction and the next is considered the wavelength, and most wavelengths are 1 mm or less. Propagation speed of the ultrasound wave is determined by the density and stiffness of a given tissue with bone propagating at higher speeds, while fluid-filled structures propagate at medium speeds and air propagating at the lowest speeds. Because air has molecules that are relatively far apart, sound travels relatively slowly (approximately 330 m/s) in air. In soft tissue, the molecules are closer together allowing sound to travel faster with an average propagation velocity in soft tissues of around 1540 m/s.

Ultrasound waves lose energy to the medium in the form of heat through a process termed absorption. Absorption increases directly with distance and frequency. A transducer produces short bursts of specific frequency sound waves that are transmitted into the patient and reflected back when it interacts with different tissues and tissue interfaces. The transducer then detects the reflected sound waves, and these waves are converted to electrical energy. A computer plots the time the sound waves traveled along with the amplitude of the reflected sound waves. Echoes are produced at tissue interfaces of different acoustic impedance. Acoustic impedance is a measure of how easily waves can be formed and is dependent on sound velocity and tissue density. The greater the differences in acoustic impedance of the reflecting interfaces, the greater the intensity of the returning echo. Ultrasound waves are constantly encountering changes in soft tissue that can affect propagation of the sound wave, which cause scatter and a weakening of the return echoes. The brightness of the dot on the monitor screen correlates to the amplitude of the returning echo. Terms to describe the appearance of an image relate to the tissue's echo intensity or echogenicity. The echogenicity of a structure or the degree to which a structure reflects sound waves determines the brightness of objects on ultrasound. The strength of the reflective sound is displayed using a gray scale, where black indicates that no sound is reflected and white indicates that a large amount of the sound is reflected. The amount of reflected ultrasound received by the probe depends on the angle of incidence (the angle that the sound wave interacts with a structure) of the ultrasound beam transmitted by the probe. When the ultrasound beam is not perpendicular to a structure or portion of a structure (such as a tendon), a portion of the reflected ultrasound will be off angle and will not return to the transducer. As a consequence, a darker (hypoechoic) area will be seen in the image. Thus, during scanning, it is essential to maintain the ultrasound beam as perpendicular as possible to the structure being imaged. The appearance of darker (hypoechoic) areas in the image that result from the ultrasound beam not being perpendicular to the structure in question is designated anisotropism or off-normal incidence. This is especially common when imaging tendons and ligaments. Making sure that the ultrasound beam is perpendicular to the fibers of these structures can be accomplished by repositioning the probe, moving it closer to the tendon or ligament edges, and by slowly moving the probe in different angles while scanning. All this information is displayed as a cross-sectional image developed by an entirely different set of physical parameters of structures (objects) than those measured by other imaging modalities.

The piezoelectric crystals in the scan head determine the frequency of the sound wave. These crystals are man-made and designed to vibrate at specific frequencies and produce a consistent wavelength sound beam. These crystals also receive sound waves coming back from the tissues and convert them to electrical energy. The wavelength dictates the resolution and the energy contained by this sound beam. High-frequency transducers have smaller crystals, the sound pulses are closer together, and the wavelengths are shorter. The shorter the wavelength, the better the axial resolution, which is a measure of the ability to show two interfaces as separate along the axis of the beam. Axial resolution is determined by the wavelength (pulse length), and the wavelength is determined by the frequency. Lateral resolution is the minimum distance that two dots can be distinguished from one another in a plane perpendicular to the sound wave.

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Lateral resolution is best in the focal zone and is dependent on the width of the sound beam. The focal zones optimize the beam and can be positioned by the operator at any depth/level of the image. Deeper to the focal point, the ultrasound beam exponentially diverges, and therefore the lateral resolution becomes progressively inferior. Improving lateral resolution requires focusing the beam to the narrowest width possible. Axial resolution is usually superior to lateral resolution. Images should be obtained with the highest frequency probe possible to obtain the best resolution of the structure of interest. However, sound is attenuated at 1 dB/cm depth per megahertz (MHz). Higher frequencies are therefore attenuated at higher rates, which reduce the penetration of the sound wave. Lower frequencies are attenuated at lower rates, which allow them to penetrate deeper into tissue. The depth of a specific structure is then calculated based on the amount of time it takes for the transmitted ultrasound beam to be reflected and received back by the probe. The ultrasound travel time is calculated assuming that sound travels at the same velocity through all tissues. For any given image, sound can travel through fat, soft tissue, muscle, fluid, blood, and skin. These different tissues have different acoustic impedances (the speed that sound travels through the different tissues), and the ultrasound image will reflect each tissue type as a different shade of gray on the screen.

Over the past several decades, the transducer construction has evolved considerably in design, function, and capability from a single-element resonance crystal to a broadband transducer array of hundreds of individual elements. Flat-face linear (tendon) and convex (micro- and macro)variable focus probes are the most popular probes for musculoskeletal imaging. Transducers are typically described by the arrangement of the crystals in the probe. The specific arrangement creates the shape of the sound beam. A linear transducer has the piezoelectric crystals organized in a linear fashion and emits sound in succession. Linear transducers are more durable, have inherently better resolution, and have a flat, narrow face with approximately 4 cm of scanning surface. These probes give superior images at tissue depths of 2 cm or less due to less distortion and less artifact in the near field. Linear transducers can now be made to produce a sound beam that is wedge shaped through a process called virtual convexing. These transducers can provide a high-resolution image, but this technology compromises the ability to image rapidly moving structures (like the heart). Standoff pads are available that improve contact with the skin increasing the footprint and moves the superficial structures into the near-field focal zone and away from the near-field artifact.

Early ultrasound systems utilized a sector transducer that transmitted a wedge-shaped sound beam that was created by mechanically rotating the crystals. These probes were quite fragile, and the crystals could be easily dislodged by dropping the probe. They have been replaced by curvilinear transducers that are similar in design to the linear transducer except that the scan head is slightly curved. These are called convex and come in microconvex and macroconvex based on the size of the transducer curved surface. Microconvex probes have smaller field of view and higher frequencies (higher resolution with lower depth of penetration), while macroconvex probes have larger field of views and lower frequencies (lower resolution and deeper penetration). The microconvex probe is utilized when the skin is contoured (concave) and difficult to seat a flat-face transducer. The divergent beam allows the examiner to image from a smaller skin contact point. These convex probes can be more difficult to use because it is easier to inadvertently change the beam angle especially when doing longitudinal assessments of fiber alignment. A macroconvex curvilinear transducer is indicated for deeper musculoskeletal examinations and for general abdominal imaging, primarily focusing on the gastrointestinal tract. Many of these probes have multiple frequencies available that allows the examiner to change the frequency without needing to change the probe. Structures within 5–7 cm of the skin should be evaluated with transducers of a minimum of 7.5–10 MHz or higher. Structures within 7–14 cm should be evaluated with 5-MHz transducers. Anything deeper than 14 cm will require lower frequencies such as 2.5–3.5 MHz. A phased-array transducer has a relatively small rectangular, flat surface. In these transducers, the piezoelectric crystals are arranged in a rectangular shape and send out all the sound waves simultaneously. This allows imaging of rapidly moving structures (like the heart or vessels when using color flow Doppler), and it produces higher-contrast (black and white) images but with less resolution compared with the linear and curvilinear transducers. The flat surface of these probes makes it ideal for imaging between the ribs.

Imaging software for available diagnostic ultrasound is constantly evolving, and recent introductions such as harmonic imaging, compound imaging, and extended field of view are now quite common. Harmonic imaging can be used to produce a clearer image by reducing artifacts. This requires a specialized probe, but it generates an image from a fundamental frequency that is sent from the transducer. When this frequency interacts with an organ, it creates a resonance frequency that is twice the fundamental frequency. The benefit of this is that the resonance frequency that is used to generate the image is relatively high, giving better resolution, and only travels through the body once, so it is less prone to artifacts. Compound imaging is the capability to use ultrasound waves sent from multiple angles to evaluate the same region. This is intended to reduce artifacts and give a much more detailed view of a structure, although it is susceptible to blurring due to motion. Extended field of view allows a complete tendon to be imaged or provides the capability to image a structure or lesion that is larger than the field of view obtained by a given transducer. Three-dimensional imaging and contrast medium ultrasound imaging are also available, but at this time it has very limited use in the equine patient.

ULTRASOUND TO EVALUATE TENDONS AND LIGAMENTS

Ultrasonography has significantly advanced the diagnosis and management of a variety of musculoskeletal injuries in performance horses.[21,24,34,41,72–74,80] Ultrasonographic imaging of the musculoskeletal system will be discussed in this chapter, but the abdominal, thoracic, cardiac, and urogenital systems are also routinely

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evaluated with ultrasound. A working knowledge of the normal anatomy is critical for accurate tendon and ligament ultrasound examinations as well as those involving joints, sheaths, and bursae.[31,46,85,90,99] Ultrasonography is routinely utilized for defining morphological change in the superficial digital flexor tendon (SDFT) and deep digital flexor tendon (DDFT), the suspensory ligament (SL), the accessory ligament of the DDF (ICL), and the distal sesamoidean ligaments (DSLs) of the pastern region. The subcutaneous tissue, paratendinous tissue, and vessels should be assessed as well. While the objective is to evaluate the morphological characteristics of the soft tissues of the musculoskeletal system, the contour of the cortical bone in this region should also be evaluated. Most importantly, diagnostic ultrasound is the most useful and practical tool to monitor the repair of these structures and guide the rehabilitation of tendinous and ligamentous structures. Many other soft tissue structures can also be evaluated including muscle, musculotendinous junctions, tendon sheaths, and bursae associated with the tendons and ligaments. Joint injury (which will be discussed later in this section) is very effectively examined with radiography and ultrasonography. These imaging tools are considered complementary and provide more information about a region/joint than either tool used alone. Joint examination should include evaluation of the periarticular structures such as the collateral ligaments and extensor/flexor tendons, joint capsule, and joint fluid accumulations. Real-time imaging capability of ultrasonography allows the use of interventional techniques (like needle insertion for injection or aspiration/biopsy), which can provide additional clinical information.[10,16,60,93] Ultrasonography is also effective at evaluating a variety of other musculoskeletal problems such as fractures of long bones, osteitis/osteomyelitis, foreign body penetration, and implant infection, as well as being utilized intraoperatively to assist with some surgical procedures.[11,81,104]

PATIENT PREPARATION AND SCAN PROTOCOL

Confirmation that lameness is associated with a specific structure or area is critical. Localization of lameness should include a clinical examination with detailed palpation of the limb with diagnostic nerve blocks performed when necessary. In most instances, injection of diagnostic anesthesia into an area will not interfere with the ultrasonographic examination. Occasionally gas bubbles in the injectate may inhibit sound transmission and necessitate performing the exam on a subsequent day. Tranquilization may be necessary and can assist with uncooperative or anxious patients. Patient preparation is very important, and both limbs should be clipped and prepped as strain-induced tendon and ligament injury can occur bilaterally with one limb being more severely affected than the other. Shaving is frequently required to give a higher-resolution image. A scrub with a detergent is generally necessary to remove dirt and debris. Many clinicians do a 5-minute sterile prep with antiseptic solution/detergent followed by an alcohol rinse. Liberal use of ultrasound gel is important although excessive gel can cause a lateral image artifact that may compromise image quality. When clipping and shaving is not possible, then the limb should be thoroughly washed with warm water and detergent. Application of alcohol to the hair coat may enhance the sound transmission. To acquire the best image, a scan head with a frequency of at least 7.5 MHz but frequencies of 10–18 MHz is preferable. Low frame rates should be used to give higher-line density and improved resolution. Most superficial structures can be visualized at a scan depth of 2–4 cm often with the use of a standoff pad. The use of a standoff pad is helpful for the evaluation of superficial structures, such as the palmar or plantar annular ligament and the SDFT in the pastern region. A standoff pad is a gelatin-based pad that is placed between the probe and the skin surface. By increasing the distance between the probe and the skin surface, the near-field artifacts that are seen in the higher portion (near field) of the image are now at the level of the standoff pad instead of being over the structure of interest. In many cases, the footprint of the transducer is limited and restricts the ability to examine the medial and lateral margins of the tendons and ligaments. Utilizing a standoff may increase the footprint and improve the examination of the structures in the same field of view. However, manipulation of the probe to a more abaxial position with the probe angled more medially and laterally may be required to evaluate the peripheral margin(s) of a structure.

The ultrasound examination should be performed in a systematic manner with each structure evaluated from proximal to distal to ensure a complete and thorough tendon/ligament evaluation. The examiner should develop a systematic approach to screening the limb such as the technique described by Genovese and Rantanen.[40,41,72–74] This approach provides a survey of all structures including veins, arteries, subcutaneous tissue, paratendinous tissue, and bone contour at specific levels in the metacarpus/metatarsus. A standardized scanning protocol has numerous advantages, but most importantly it provides a means for clinicians to effectively screen the limb and as a tool to accurately communicate about their findings. This imaging protocol is based on the metacarpus length being approximately 24 cm in length or roughly three hand widths of a person's hand or 8-cm/hand breadth. The metatarsus is longer than the metacarpus and measures approximately 32 cm or roughly four hand widths in length. These zones are numbered 1–3 in the front limb and 1–4 in the hindlimb. Each of these zones are further subdivided into two equal zones named A and B (each being 4 cm) such that the front limb has zones 1A, 1B, 2A, 2B, 3A, and 3B. The area associated with the proximal sesamoid bones of the fetlock is considered zone 3C (or 4C in the hindlimb). Some authors utilize a simple numerical scheme with the front limb having levels 1–7 and the hindlimb having levels 1–9. These levels are the same zones mentioned above but without the letter designations (Figures 3.89–3.106).

To more completely assess the architecture of the SL branches, it is necessary to incline the transducer more medial to lateral or lateral to medial as the examiner progresses distally until they attach to the proximal aspect of their respective proximal sesamoid bone. The imaging protocol for the pastern is based on zones

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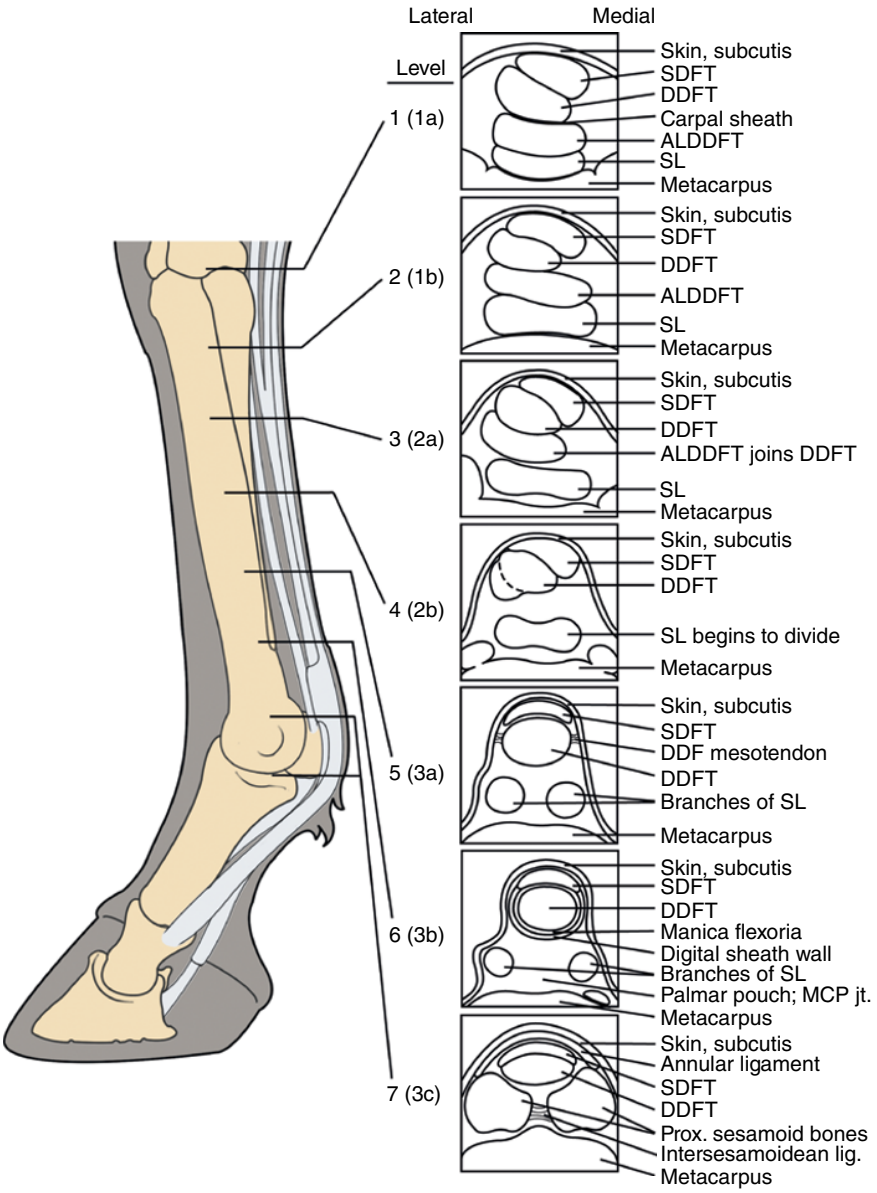
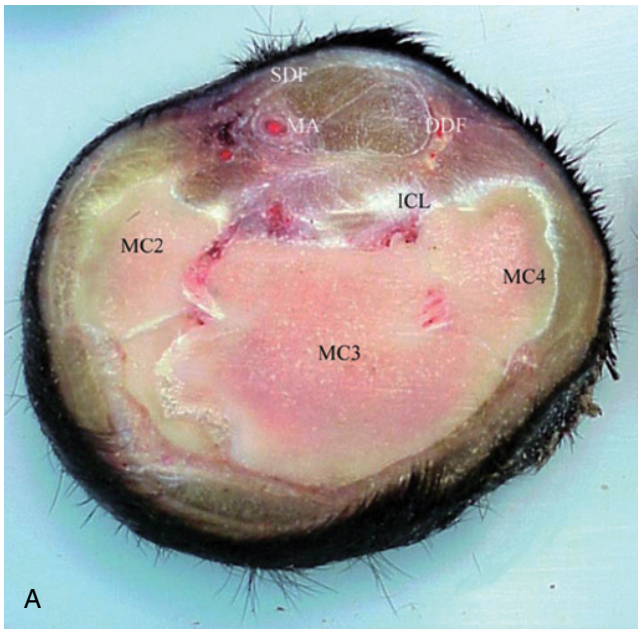
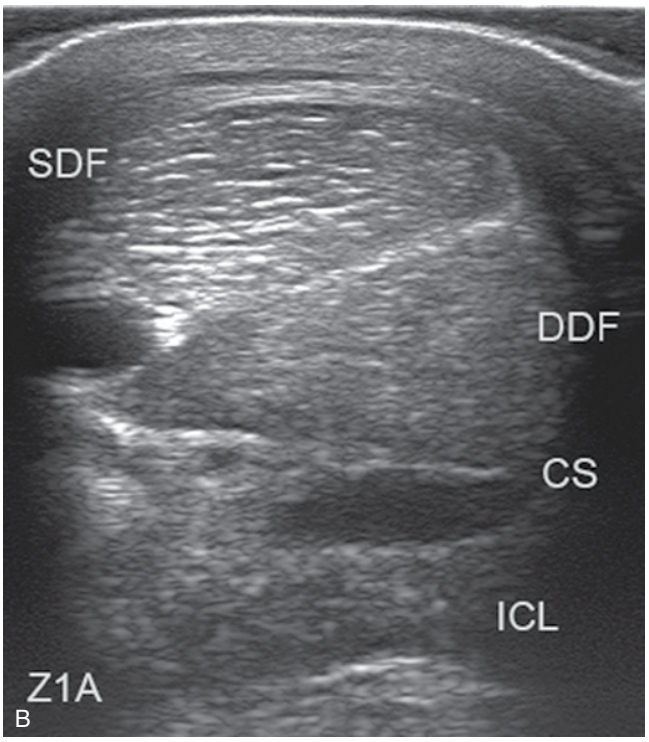


Figure 3.89. Ultrasonographic imaging of the metacarpus is based on the length being approximately 24 cm in length or roughly three hand widths of a person's hand (8 cm/hand breadth). These zones are numbered 1–3 in the forelimb. These zones are further subdivided into two equal zones named a and b (each being approximately 4 cm) such that the forelimb has zones 1a, 1b, 2a, 2b, 3a, and 3b. The area associated with the proximal sesamoid bones (PSB) of the fetlock is considered zone 3c. Some authors use a simple numerical scheme, with the forelimb having levels 1–7. The structures identified from the transducer to the palmar metacarpus are the SDFT, DDFT, ICL, and SL.

related to the proximal and middle pastern bones. The proximal pastern or P1 has 3 2-cm zones of associated with it and named P1A, P1B, and P1C. The middle pastern bone or P2 is shorter and has just 2 2-cm zones each associated with it and named P2A and P2B.[40] The overall length of the pastern and height of the collateral cartilages of the distal phalanx can significantly affect the ability to image the DDFT in zone P2A. To evaluate the origin of the oblique sesamoidean ligament (OSLs) on the distal aspect of the proximal sesamoid bone, it is necessary to incline the transducer more dorsally such that a medial to lateral or lateral to medial orientation is obtained. The pastern is more difficult to examine than the metacarpus because positioning the probe between the distal aspect of the sesamoid bones (and the ergot) and the proximal aspect of the collateral cartilages can be difficult. Placing the foot on a block of wood with the limb placed more caudal than the opposite limb increases the extension of the fetlock joint and places the axis of the pastern in a more upright position (Figure 3.107). In general, the DSLs and branches of the SDFT are smaller and inclined at different angles as they course to their insertions, which require that different scan planes will be used to evaluate each of these structures.

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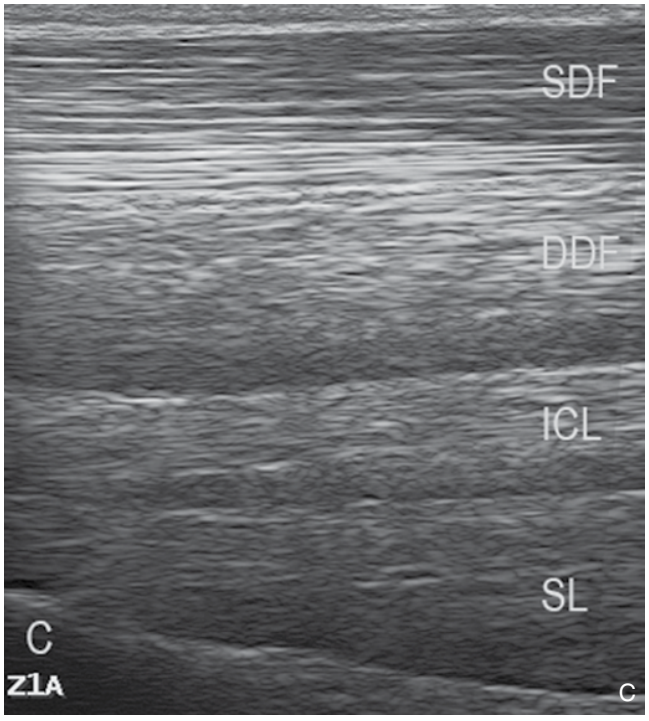
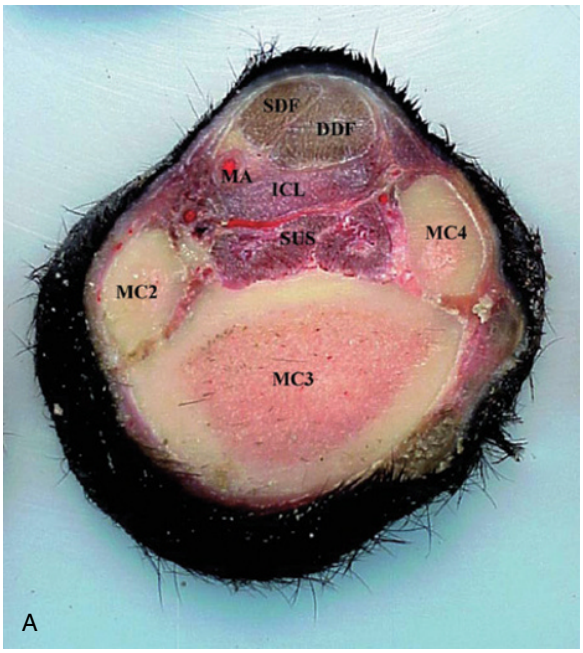
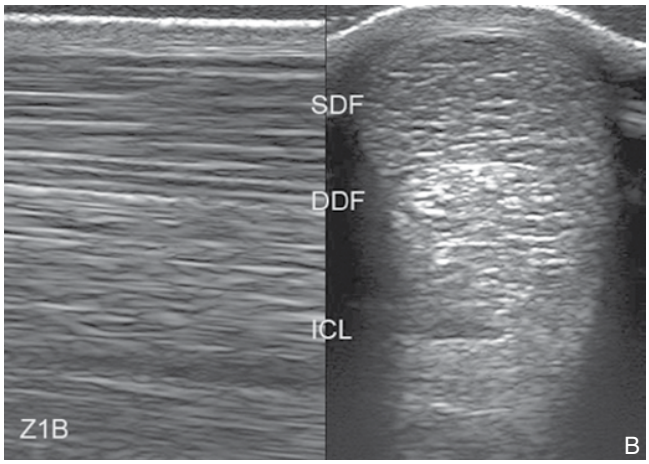
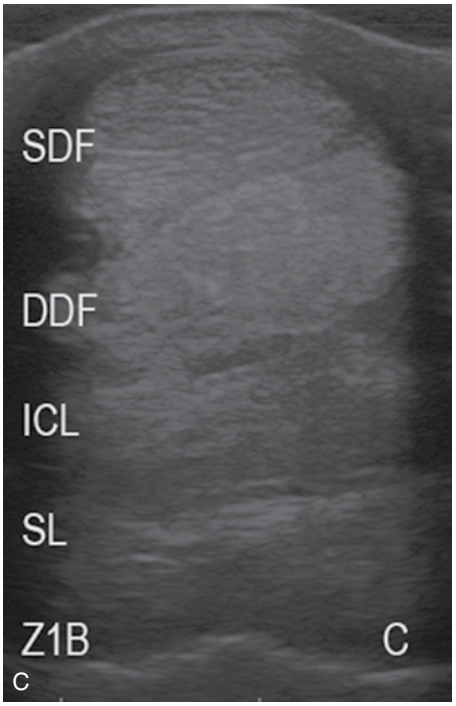


Figure 3.90. (A) In Z1A the SDFT is round but with a flat border dorsally where it is adjacent to the DDFT (A and B). The DDFT is rounded in shape. The ICL is located dorsal to the DDFT with the carpal sheath visualized as an anechoic triangular structure between these two structures (B). The SL is located dorsal to the ICL but may only have a few fibers apparent in proximal Z1A (C). The SL becomes more apparent, progressing distally in this zone. (C) This ultrasound image is taken at the proximal aspect of Z1A to correspond to the gross section slice plane that is close to the carpometacarpal joint. At this level, the ICL is the only structure apparent dorsal to the DDFT. The median artery and large metacarpal vein are prominent on the medial aspect of the DDFT. *Source:* US images courtesy of Dr. Caitlyn Horne.

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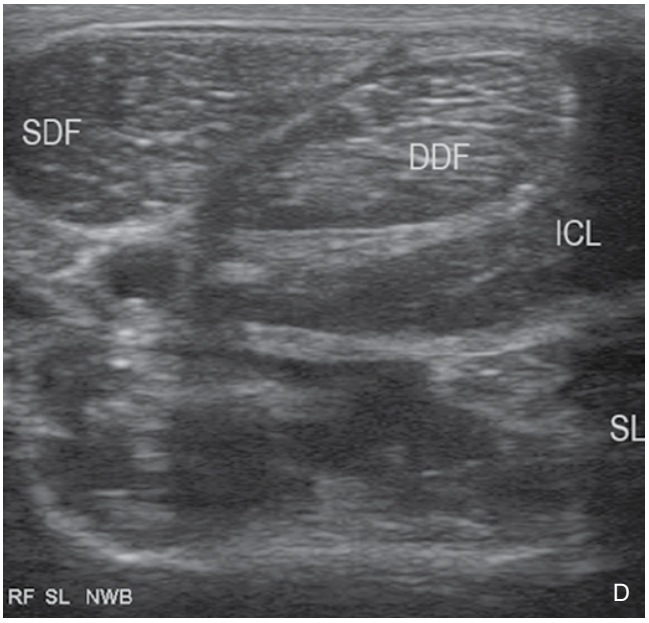
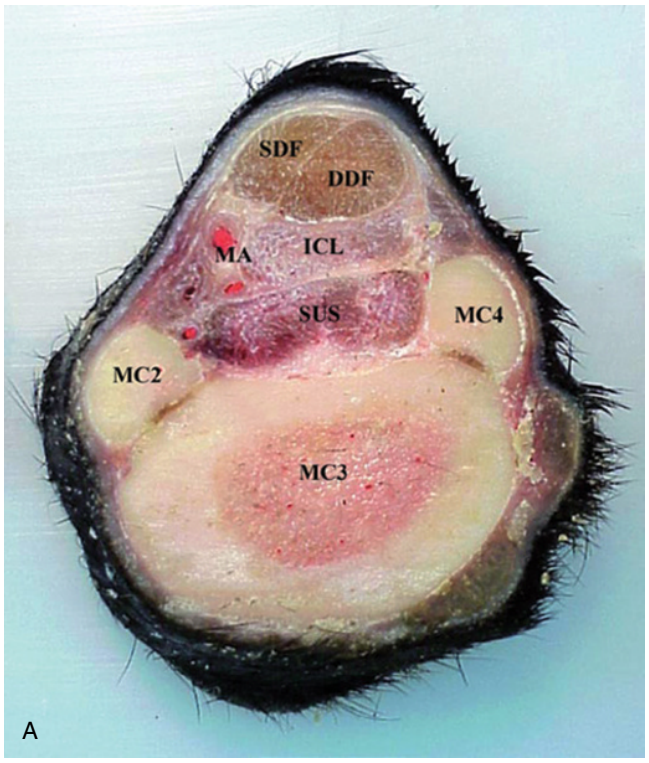
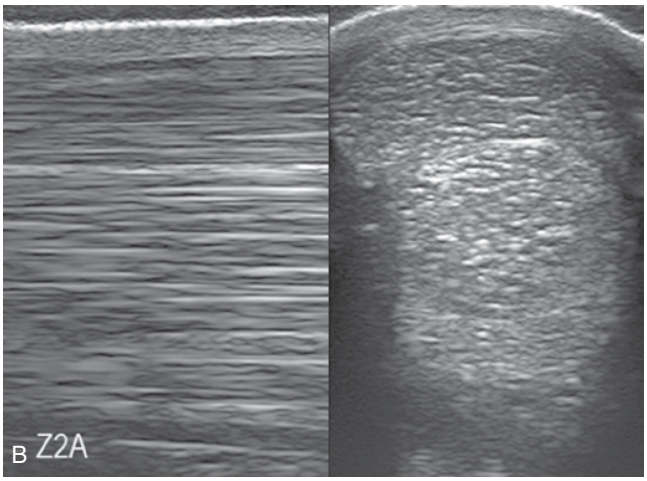
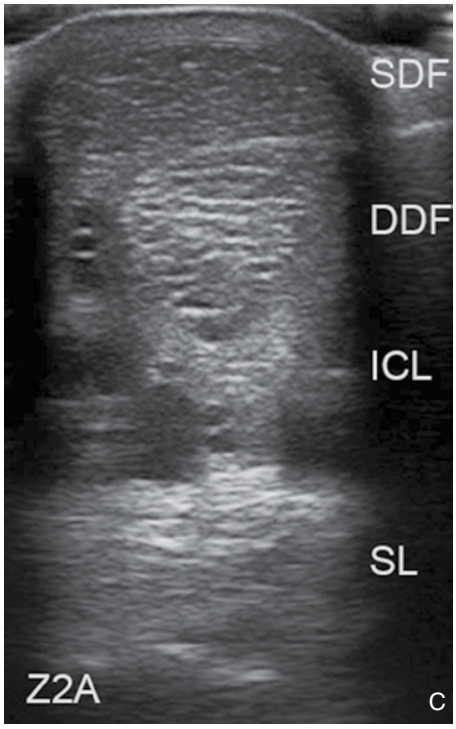


Figure 3.91. In Z1B the palmar metacarpal structures appear much as they did in zone 1A. The SDFT appears to flatten palmar to dorsal, and the DDFT becomes more rounded. The ICL becomes more inclined toward the DDFT. Fibers of the SL become more pronounced, and this structure widens medial to lateral (A). The SDF and DDF tendons should be evaluated with the focal zones placed at the appropriate level and often with a standoff pad in place (B). However, ultrasound imaging of the suspensory ligament in this area is often difficult due to a limited footprint (the SDF and DDF are not as wide as the SL) and the creation of edge artifacts and acoustic enhancement artifacts from more superficial structures (tendons and vessels) that can make evaluation of the SL difficult (C). Angled contrast ultrasound (ACUST) utilizes off-angled and non-weight-bearing imaging of the proximal suspensory to allow visualization of the muscle and fat bundles within the origin of the SL not normally evident in standard transverse ultrasound imaging of this area (D). *Source*: US images courtesy of Dr. Caitlyn Horne.

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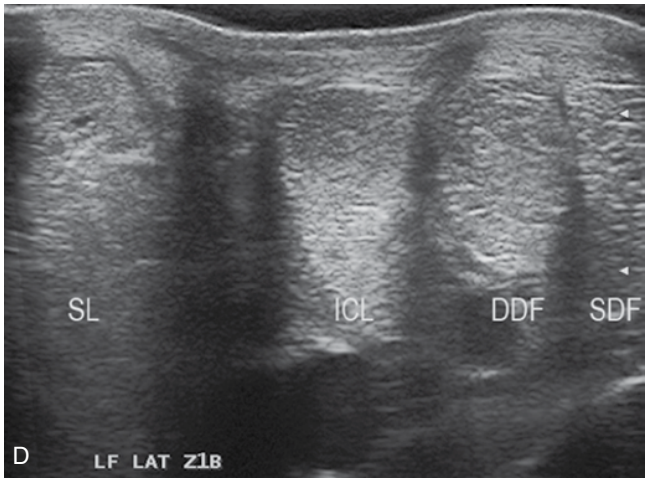


Figure 3.92. In Z2A the ICL narrows palmar to dorsal, and the SL increases in area, becoming more discreet and separated from the palmar cortex of MCIII (cannon bone). The large metacarpal vessels can be seen medially and laterally (A). These vessels also create edge and acoustic enhancement artifacts, compromising interpretation of the SL architecture. (B and C) These ultrasound images demonstrate these artifacts and also highlight the importance of focal zone placement. The focal zone in (B) is placed on the SDFT/DDFT junction with the deeper structures out of focus and poorly imaged, and the focal zone in (C) is placed at the level of the SL. Alternatively, an abaxial orientation (in a more lateral to medial or medial to lateral direction) of the transducer can sometimes allow a better interpretation of the medial and/or lateral border of the suspensory ligament body as evidenced in image (D). *Source:* US images courtesy of Dr. Caitlyn Horne.

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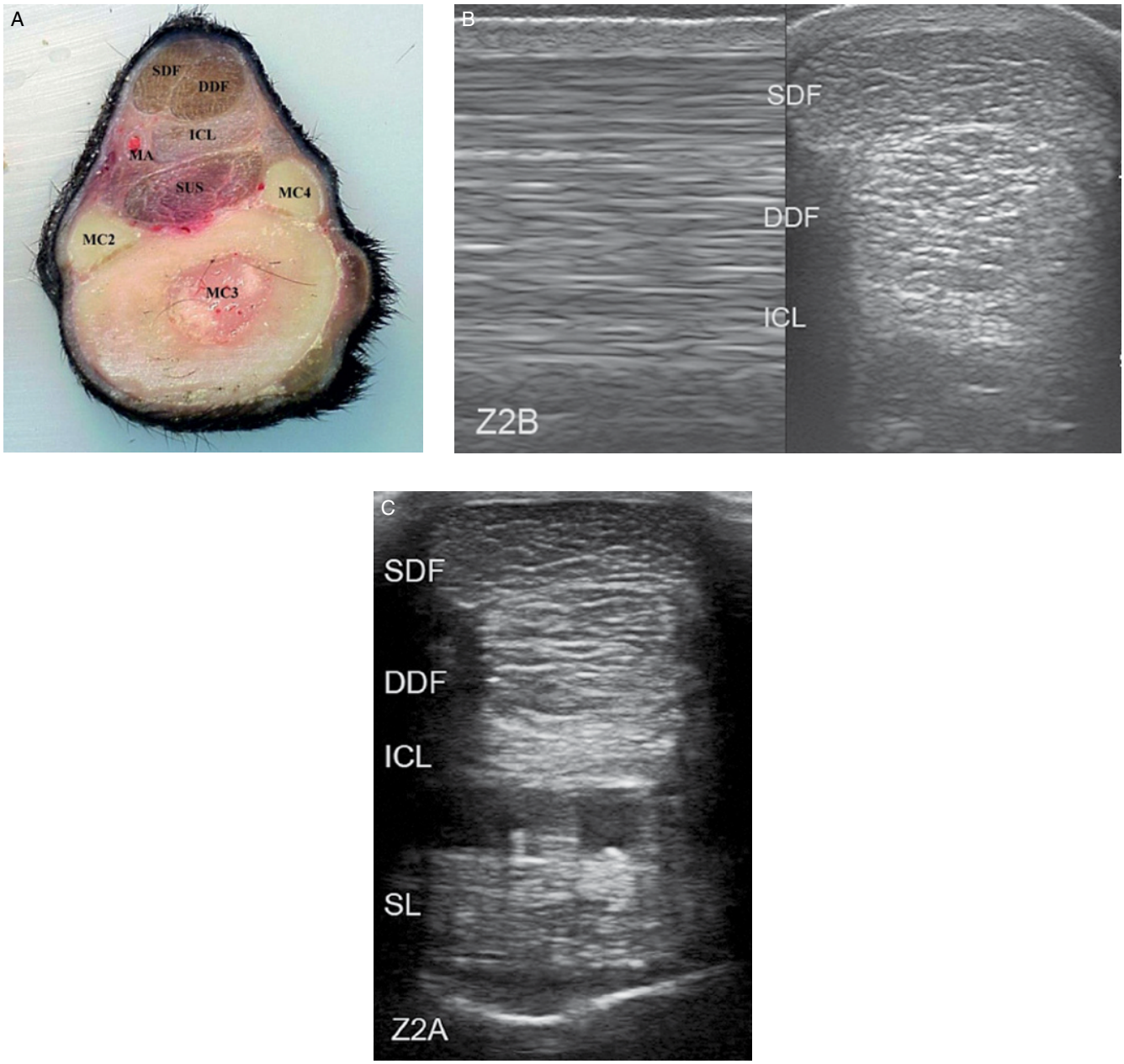
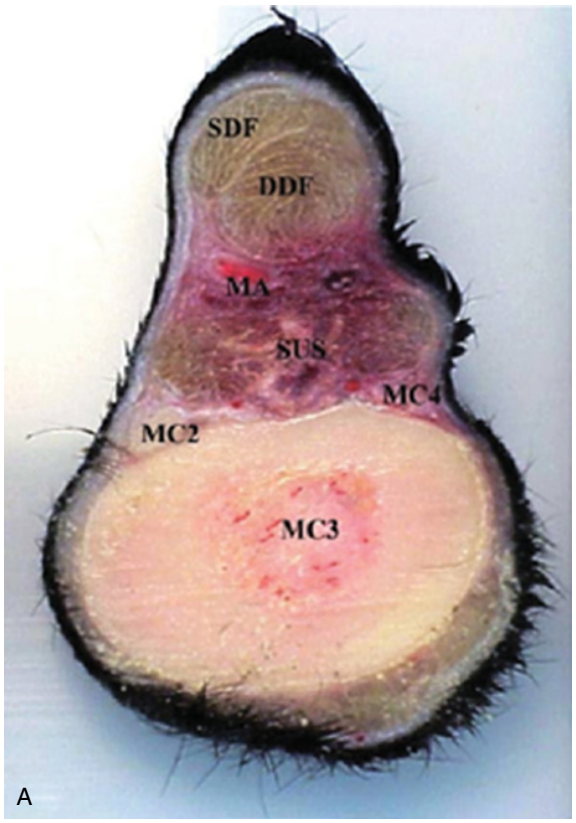
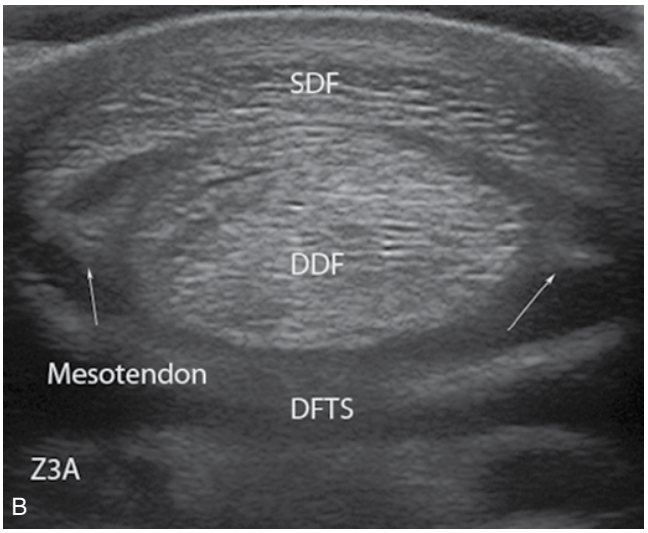


Figure 3.93. (A) In Z2B the SDFT becomes flatter, and the ICL remains distinct from but closely associated with the DDFT. The metacarpal vessels converge to midline between the ICL and the SL. (B) This ultrasound image with the focal zone placed superficially demonstrates the fibers of the SDFT/DDFT interface, while (C) shows focal zone at the level of the SL and the convergence of the large metacarpal vessels palmar to the SL. *Source*: US images courtesy of Dr. Caitlyn Horne.

Obtaining a good-quality images in tissues of varying acoustic impedances is quite dependent on the ability of the ultrasonographer to appropriately adjust the machine and settings (transducer, frequency, depth, focal zones, and gain). The ultrasound probe is placed on the palmar surface of the limb with the beam oriented perpendicular to the longitudinal axis, as well as the transverse axis of the tendons or ligaments of interest with the limb in a weight-bearing position. Each structure should be examined on cross-sectional and on longitudinal orientation with appropriate placement of the focal zone(s) on the structure of interest at each location. The size, shape, margins, and echogenicity of the structures should be evaluated, and a measurement system, using distance (cm) or zones, should be used to obtain images at regular intervals. Comparison to the contralateral limb is important in all cases, even those with obvious injury in the primary limb. In the metacarpus each level should have two transverse scans performed. The first scan should have a standoff placed on the transducer with the focal zone(s) and transducer angle directed initially at the superficial digital flexor (SDFT) and deep digital

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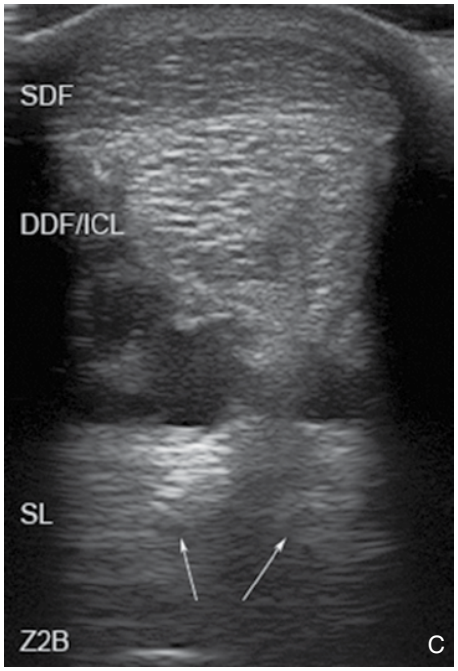
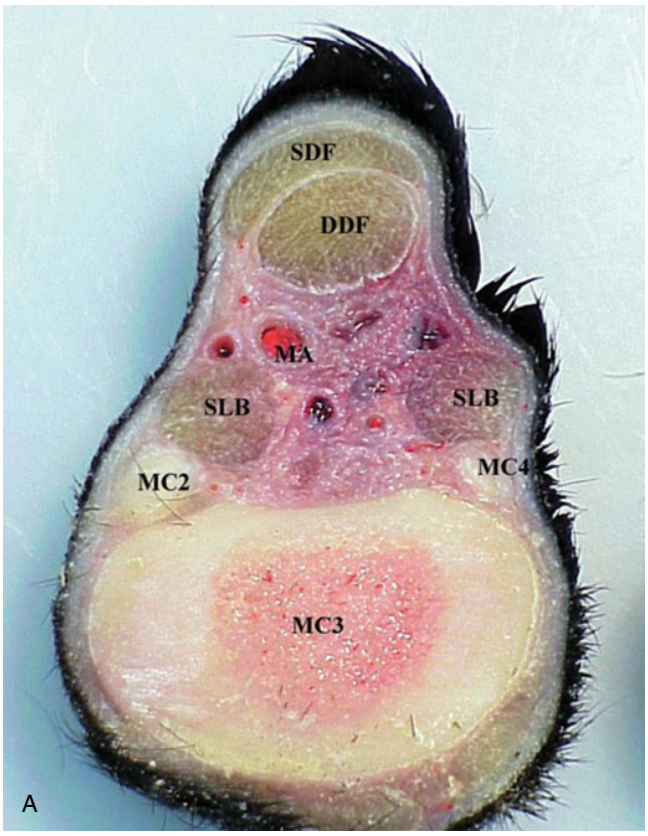


Figure 3.94. (A). In the proximal aspect of zone 3A, the SDFT thins and begins to expand medial to lateral and appears crescent shaped. The DDFT and ICL are beginning to blend together and can appear as one structure. In the proximal aspect of the DFTS, the DDF tendon receives mesotendinous attachments to the medial and lateral borders of which the medial mesotendon provides the predominant arterial blood supply to the DDF within the sheath (arrows) (B). In the proximal aspect of this zone, the SL has not divided and continues to appear as one structure. There may appear to be a hypoechoic area within the central aspect of the SL just prior to the formation of the branches, but this should not be confused with a lesion (C). *Source*: US images courtesy of Dr. Caitlyn Horne.

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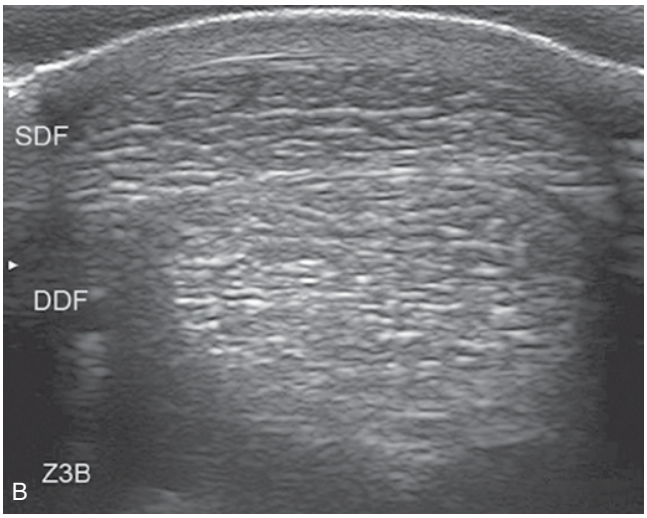


Figure 3.95. (A) The distal aspect of Z3A demonstrates the SDFT as elongated in a medial to lateral direction and the division (splitting) of the SL into branches (SLBs). (B) This ultrasound image with the focal zone(s) directed at the SDFT and DDFT demonstrates the widening of the SDFT and blending of the fibers of the DDFT and the ICL. *Source:* US images courtesy of Dr. Caitlyn Horne.

flexor (DDFT). The second scan should be acquired with or without a standoff with focal zone(s) and transducer angle directed at the ICL and SL (Figure 3.108). In some instances it may be helpful to utilize a microconvex transducer for this scan because the divergent beam can improve assessment of the medial and lateral borders of the SL. The transverse scan plane should be positioned such that the structures on the left side of the horse are placed to the left side of the screen. Some clinicians like to place the medial side of the limb on the left side of the screen and the lateral side of the limb of the right side of the screen.[80] In addition the sagittal scan plan should position structures of the proximal aspect of the limb to the left of the screen and structures to the distal aspect of the limb to the right side of the screen. Some clinicians place these structures opposite with the proximal aspect of the limb to the right of the screen.[80] Whatever protocol is utilized it should be done consistently with appropriate labeling on the recorded image.

Careful attention should be paid to all tendinous/ligamentous structures as multiple structures are often involved. The examiner should also be aware that an injury might extend proximally into the carpal sheath or distally into the digital sheath/pastern area. If an abnormality exists then the lesion should be mapped and measured in centimeters from a reference point such as the accessory carpal bone in the front limb and the point of the hock or tarsometatarsal (TMT) joint (head of the lateral splint) in the hindlimb. Measurement of the lesion should extend from the proximal aspect of the normal to the affected interface of the structure to the distal aspect of the lesion. The lesion should be evaluated on both cross-sectional and longitudinal scan planes. The lesions' maximum size should be determined and mapped. Echogenicity and fiber alignment should be subjectively evaluated throughout the abnormal tissue. Musculoskeletal scanning requires a technique that creates the maximum echogenicity in the structure being examined to prevent decreased echogenicity caused by variations in beam angle (anisotrophy) that could be mistaken for a lesion. Each image should be labeled with the date, owner's name, patient name, the limb being examined, and the location of the lesion(s). A description of the scan plane should be included in every image and every image recorded as part

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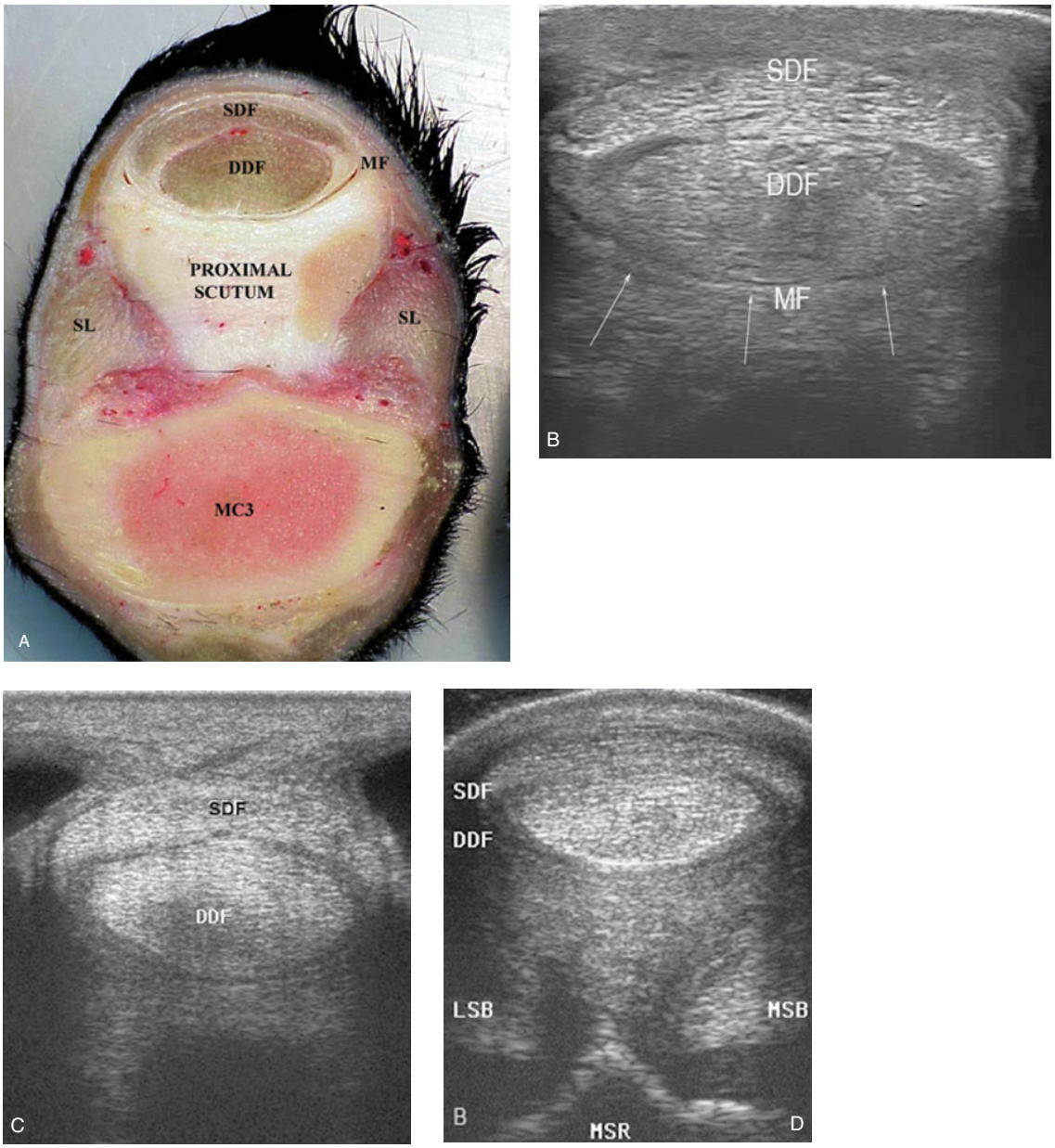
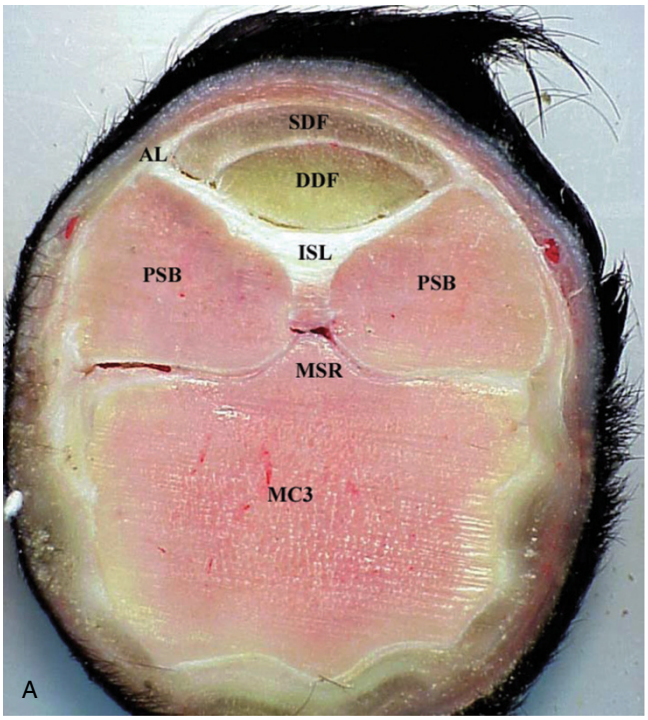


Figure 3.96. (A and B) Zone 3B is somewhat longer than the other zones, and several important landmarks can be seen in this area. The SDFT and DDFT widen in a medial to lateral direction. These structures can be difficult to image together, so each must be examined independently. The SDF tendon forms a ring around the DDF tendon called the manica flexoria. (B) This ultrasound image demonstrates a moderate amount of effusion within the DFTS and allows the natural axial connection of the DFTS to the palmar border of the SDFT proximal to the metacarpophalangeal (MCP) joint to be visualized (C). This axial attachment should not be considered an adhesion/abnormality. At the junction of Z3A and 3B after the SL divides into medial and lateral branches, the proximal scutum and palmar aspect of the fetlock joint just proximal to the PSBs can be seen (D). The proximal aspect of the midsagittal ridge of the palmar metacarpus can be seen through the proximal scutum. *Source*: US images courtesy of Dr. Caitlyn Horne.)

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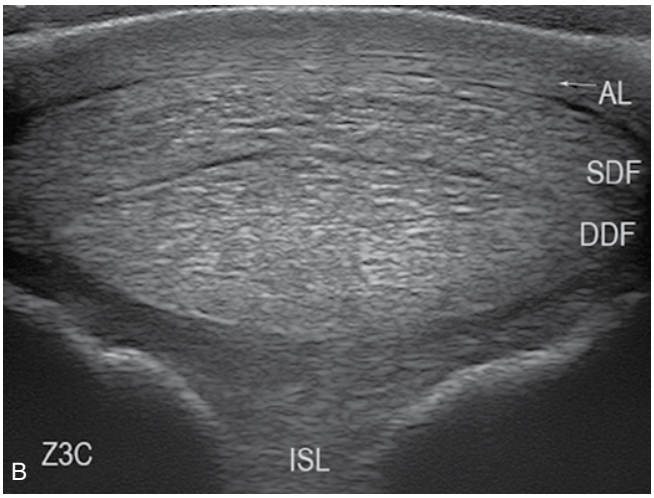


Figure 3.97. (A) Zone 3C begins at the level of the PSBs and extends through the fetlock canal. The primary annular ligament (PAL) extends from the palmarolateral to palmaromedial border of the PSBs (abaxial border of each PSB). (B) This ultrasound image demonstrates the PAL wrapping around the SDFT and DDFT to hold these tendons within the fetlock canal. It may be necessary to move the transducer in the medial to lateral plane and slightly rotate transducer to more effectively demonstrate the fibers of the PAL. A more abaxial orientation of the transducer should also be utilized to image the attachment of the PAL to the PSBs. *Source*: US images courtesy of Dr. Caitlyn Horne.

of the medical record. Documentation of the lesion location, lesion echogenicity, and extent or degree of change should be included on the image. Once the image has been labeled, it should be stored and maintained as part of the permanent medical record. Images can be stored as thermal prints (which degrade over time), as video recording, or as digital images for comparison at later examinations. Most current machines available provide software that allows the image to be traced, which provides a cross-sectional area (CSA) of the tendon/ligament and lesion. As mentioned earlier this is critical for the rehabilitation process.

Tendons and ligaments appear similarly as moderately echogenic structures with relatively well-defined margins due to their parallel fascicular arrangement. It is this arrangement of fibers, aligned to resist tensile forces, that creates the intense specular reflections (echoes) seen when the sound beam is perpendicular to the direction of the fascicles. The examiner must pay particular attention to the course of the tendon or ligament being imaged and maintain the transducer at 90° to the structure. The structures of the metacarpus/metatarsus and the pastern change orientation as they incline distally toward their insertion. To perform a complete examination each structure should be evaluated independently with careful attention being paid to correct beam angle and focal zone placement. The horse should be weight-bearing on the limb and the tendons and ligaments loaded during the ultrasound examination. Imaging while the horse is not bearing weight can create changes in shape and size of these structures. Not bearing full weight on the limb also can cause relaxation artifacts to occur within the tendons and ligaments being examined. Relaxation artifacts appear as hypoechoic areas within the normally bright specular reflections seen during routine examination of tendons and ligaments that can compromise the accuracy of the study. Relaxation artifacts can also occur in some abnormal conditions. For example, complete disruption of the suspensory apparatus relieves the tension in the straight sesamoidean ligament (SSL), resulting in relaxation artifacts in the SSL. This is also apparent when evaluating lacerations or rupture of a tendon/ligament that relaxes the tensile forces in the affected structures and can create relaxation artifacts most often proximal but also distal to the site.

There are some indications for off-weighted examination of some structures. Proximal annular ligament syndrome of the digital flexor tendon sheath frequently has a proliferative tenosynovitis that can obscure the borders of the SDF and DDF diminishing the ability of ultrasonography to define these structures as separate.

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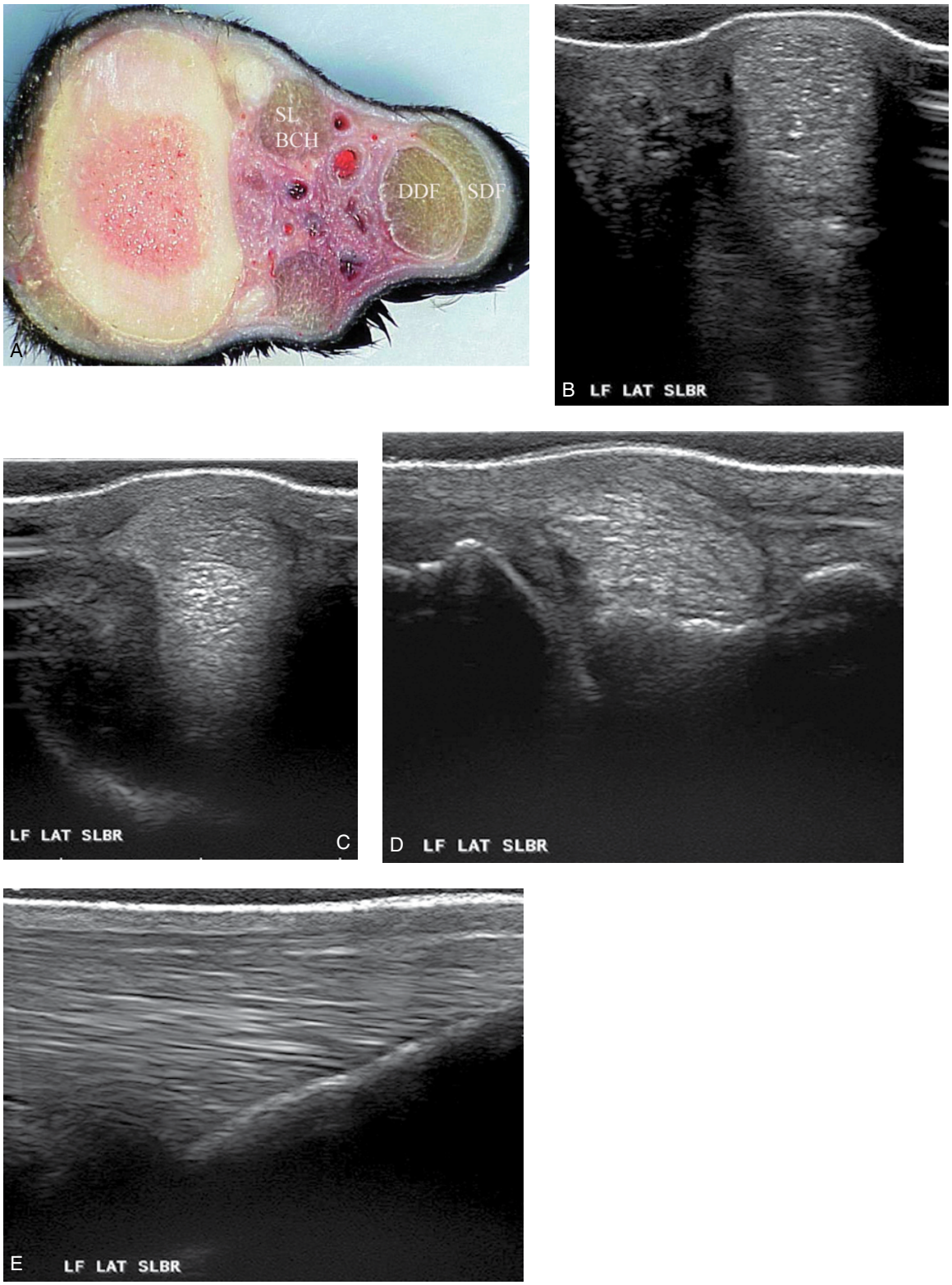


Figure 3.98. (A–E) The branches of the suspensory ligament (SLBs) are best imaged with the transducer placed more abaxially with a medial to lateral and/or lateral to medial orientation. These structures should be evaluated at the level where they begin to divide in distal 3A until their attachments to the PSBs. (B) Initially the SLBs have an oval shape. (C) Further distally the branch becomes somewhat D shaped. (D) The branch appears to rotate in a dorsal to palmar direction, becoming oval shaped just prior to their attachment onto the PSBs. (E) The SLBs should also be evaluated in the longitudinal plane from their beginning to their attachments to the PSBs. *Source*: US images courtesy of Dr. Caitlyn Horne.

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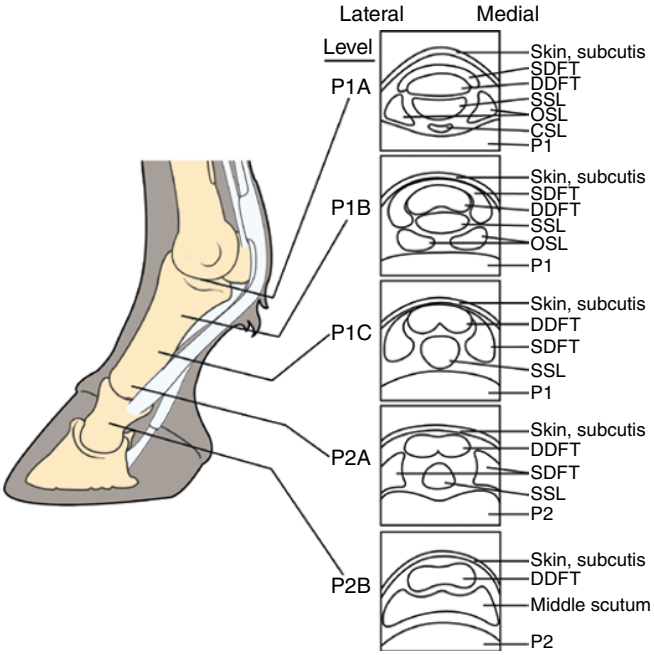
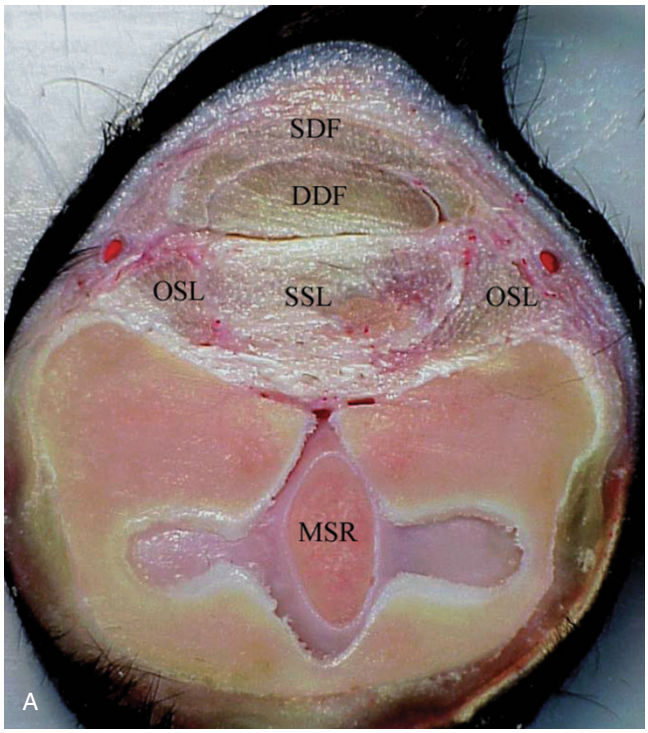


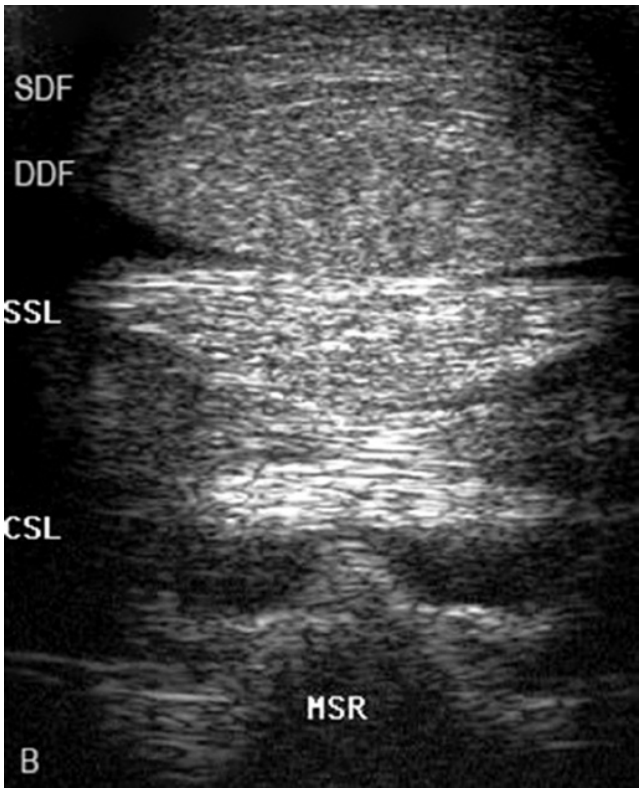
Figure 3.99. The SDFT and DDFT continue into the pastern. The DSLs (SSL, paired OSLs, and paired CSLs) originate on the PSBs and course distally onto insert on the distal aspects of P1 and P2. The SSL originates from the axial region of the PSBs and crosses the pastern joint onto insert on the palmaroproximal aspect of P2. The OSLs originate from the abaxial region of the PSBs and insert on the roughened triangular area of P1. The cruciate sesamoidean ligaments cross from the PSB to the contralateral aspect of palmaroproximal P1. The SDFT encircles the DDFT as they course out of the fetlock canal. The fibers of the SDFT incline abaxially, moving distally in the pastern to form teardrop-shaped branches that insert on the abaxial area of palmaroproximal P2. The DDFT continues distally and becomes bilobed in the midpastern area. The DDFT widens in a medial to lateral direction as it courses into the foot to insert on the distal phalanx (P3).

Off-weighted scan of this area is indicated to determine if adhesions may have developed between the flexor tendons within the digital flexor tendon sheath. Lifting the limb off the ground and placing the fetlock joint through a full range of motion while performing an ultrasonographic examination in longitudinal scan plane may be necessary to demonstrate a lack of independent movement of the tendons. A lack of independent movement suggests the formation of adhesions between these two structures. In addition, horses with proximal annular ligament syndrome may have a compartmentalization syndrome, which can compress the tendons obscuring significant pathology in the standing animal. Lifting the limb off the ground and placing through a range of motion while imaging in transverse scan plane may allow the architecture of the SDF and DDF to be assessed more completely.

Another indication for examining the limb while off-weighted is to perform angled contrast ultrasound technique (ACUST), which was reported to allow a more complete examination of the proximal SL.[24] When used ACUST is performed in conjunction with and following the examination with the standard ultrasound technique. This technique can provide more information about an injury than just the standard US examination. ACUST is performed with the limb off-weighted and with the probe manipulated off incidence. The ultrasound beam is first positioned perpendicular to the tendon or ligament to create maximum echogenicity, and this image is saved and then compared with an oblique angle of incidence image at the same level. The least amount of angle to produce a decrease in echogenicity of the tendon/ligament fibers should be used. Relaxation of the flexor tendons allows them to become hypoechoic and brings the suspensory closer to the transducer and within the focal zones of the transducer. ACUST allows the periphery of a structure to be seen more clearly, and this can be used to compare the shape of a structure to the normal side and to ensure that the examinations are being performed at similar levels of the tendon/ligament. Anisotropic properties of tendon and ligament fibers differ from that of adipose tissue and muscle that are found proximally in the SL. The echogenicity of muscle is much less dependent on beam angle compared with tendon and ligament fibers. The echogenicity of adipose tissue is not beam angle dependent. Injuries to the proximal suspensory can distort the fat/muscle bundles and their echogenicity as well as the perimeter of the proximal suspensory ligament (PSL). The principles of ACUST are now also utilized to evaluate a number of different tendinous/ligamentous structures. The appearance of an acute injury of a tendon/ligament is probably best determined with standard on angle technique, but as the tendon begins to repair, ACUST can help to delineate the development of scar tissue within a structure. Scar is more echogenic than normal collagenous tissue when examined off angle because the collagen fibers in scar tissue are initially randomly oriented and progressively remodel over time and with load. Normal collagen reflects sound away from the probe when off angle (appearing hypoechoic), making scar more conspicuous. ACUST can be used to characterize regions of pathologic change within a structure from normal anatomical variation. This technique can also allow better definition of the perimeter of a structure particularly when surrounded by peritendinous/periligamentous fibrosis. As an added benefit ACUST allows the examination of a structure without tension, making some lesions to become more conspicuous especially when adjacent fluid can enter regions of fiber disruption.

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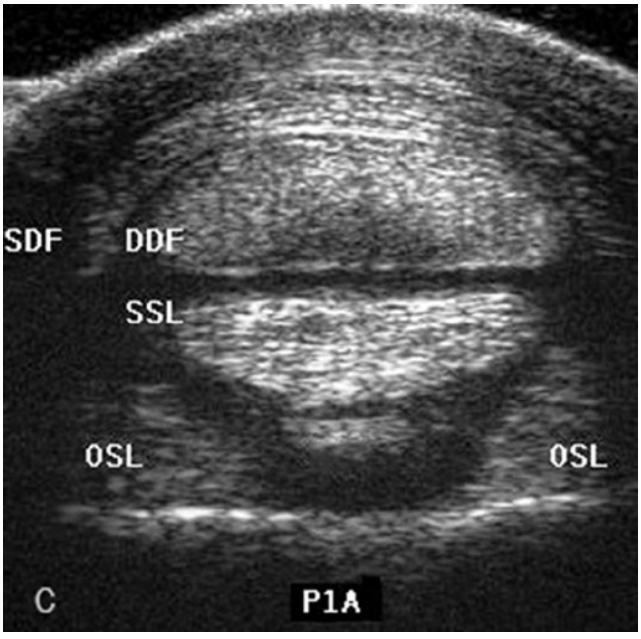
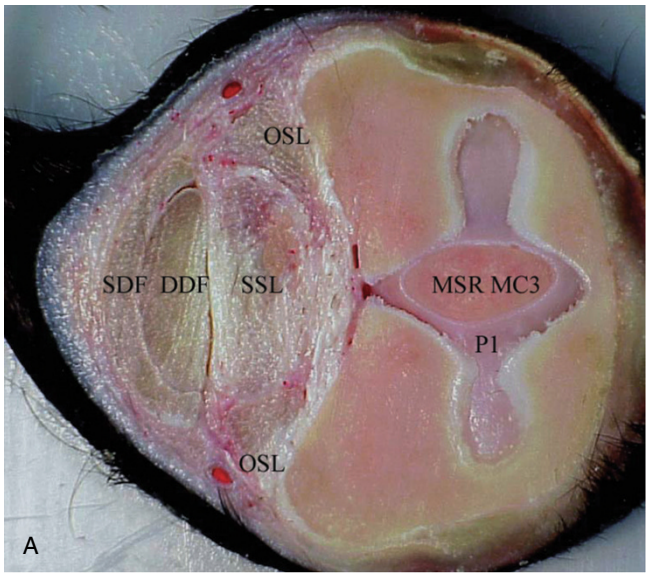
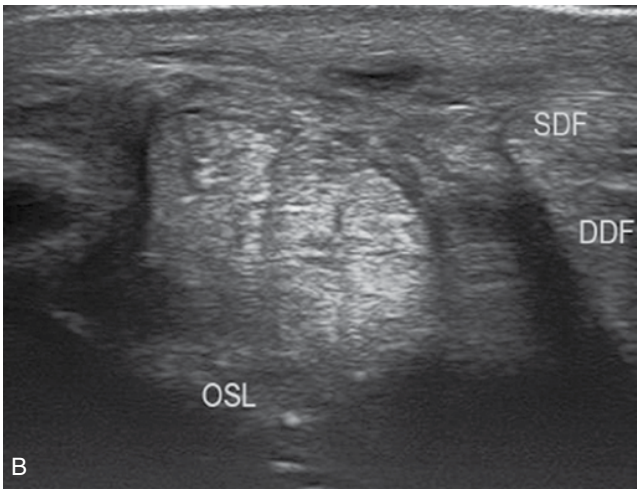


Figure 3.100. (A) In ZP1A the contour of the palmar border of the proximal P1 is a V-shaped hyperechoic line. From the midline position the OSLs are positioned on the abaxial surfaces of P1 (palmar tuberosities). The SDFT encircles the palmar/plantar border of the oval-shaped DDFT with a uniform palmar to dorsal dimension. The SSL is triangular in shape at its origin from the intersesamoidean ligament (ISL) and PSBs. (B) Angling the transducer proximad allows the distal aspect of the midsagittal ridge to be seen (B). The DDFT is oval at this level, while the SSL is triangular at its origin; the cruciate sesamoidean ligament (CSL) can be seen dorsal to the SSL (and the midsagittal ridge). (C) At the level proximal of P1, the DDFT and the SSL are easily imaged, but the OSL will require a more abaxial position of the transducer. *Source*: US images courtesy of Dr. Caitlyn Horne.

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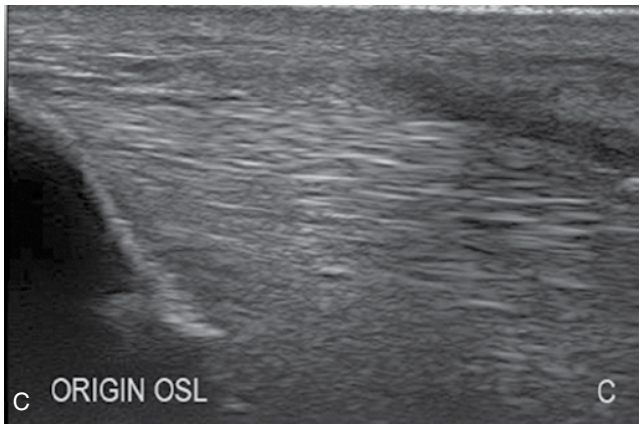
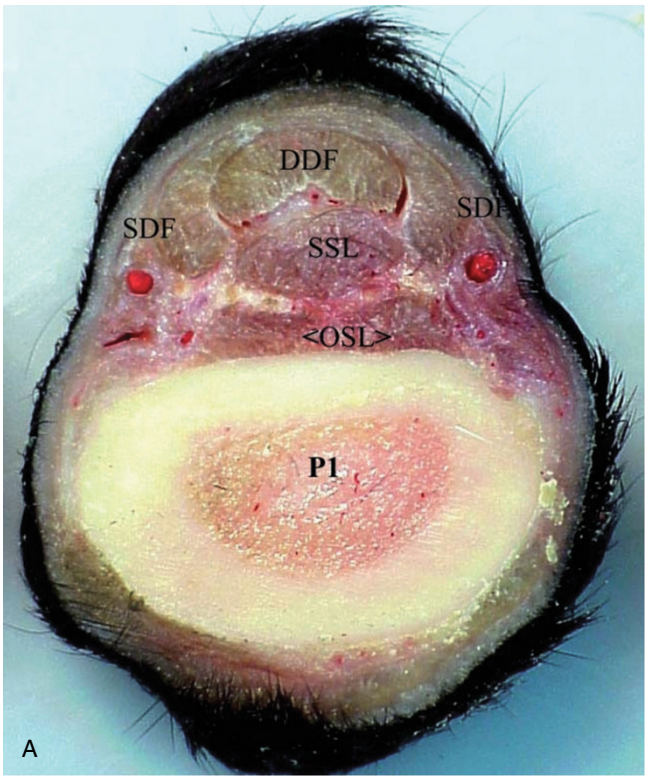


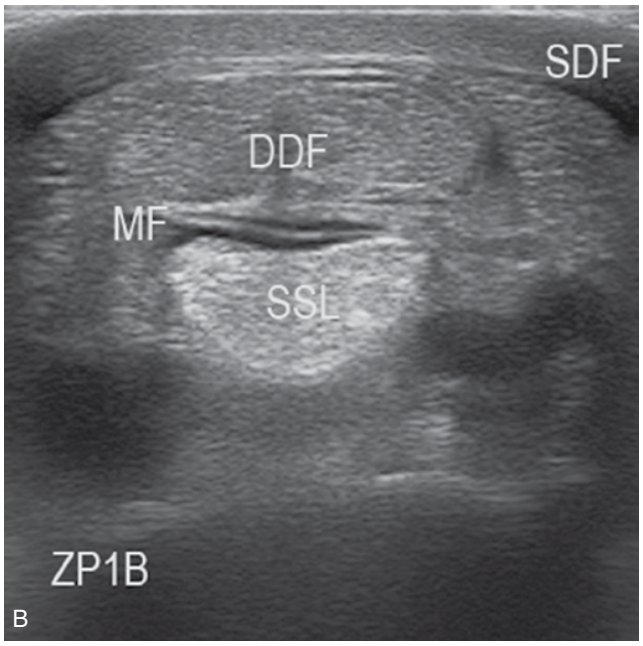
Figure 3.101. (A) To best image the origin of the OSLs, the transducer should be placed more abaxially and directed toward the base of the PSBs (similarly to the position for the SL branch examination). In transverse section the OSLs appear somewhat rounded at this level. (B) The transducer should also be placed longitudinally to image the origin of the OSLs to their respective PSBs. The longitudinal examination should image the insertion of the OSL onto the proximal sesamoid bone (C). *Source*: US images courtesy of Dr. Caitlyn Horne.

ULTRASONOGRAPHIC ASSESSMENT OF TENDON/LIGAMENT PATHOLOGY

Tendon and ligament injuries are recognized ultrasonographically by changes in size, shape, architecture, position (with respect to surrounding anatomy), and fiber alignment. Acute tendon injury manifests ultrasonographically as enlargement often with hypoechogenicity of the tendon/ligament architecture on cross section and the appearance of a striated or fibrillar pattern in the longitudinal images. These changes are often due to disruption of tendon matrix, the occurrence of intratendinous hemorrhage, and the intensity of the inflammatory response. Depending on the severity of injury, there may be changes in the overall shape, distortion of the margins/outline of the structure, and a change in the normal anatomical position in relation to other structures in that area. The most consistently diagnosed tendon and ligament injuries include enlargement and hypoechogenicity (indicating inflammation, edema, fibroplasia, and/or tearing) and enthesopathies. CSA measurements are considered a very sensitive indicator of inflammation, and the best way to assess increases in size is from the transverse images (Figure 3.109). Subtle enlargements of a structure may require comparison with the opposite limb. Any enlargement suggests structural thickening, and the rest of the examination should attempt to determine if the change is a result of acute, subacute, or chronic injury. Most current ultrasound machines have the capability to trace the CSA of the frozen image on the screen. Some machines have installed software that allows stored images to be processed after recovery and storage. Post-processing of the image to determine a CSA can be accomplished with digitizing software provided the images are stored as DICOMs; however this can be quiet time consuming (Dicom Works$_R$). Lesion CSA can be compared with the tendon/ligament CSA measurement to calculate the proportion of tendon involved. An accurate

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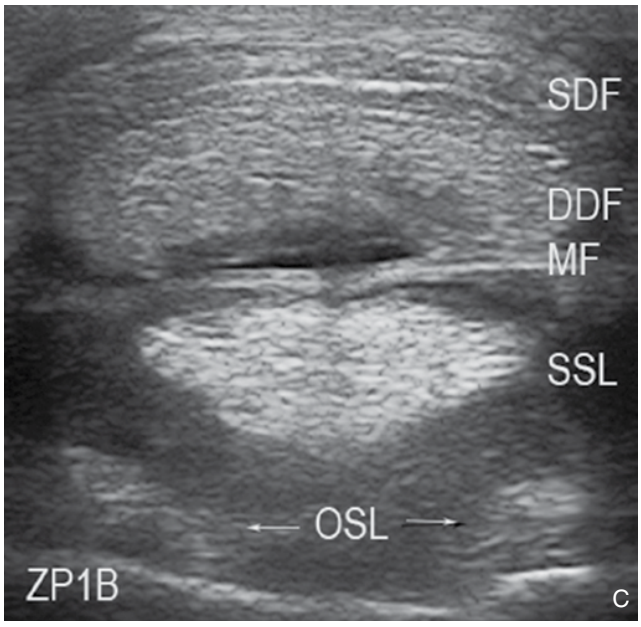
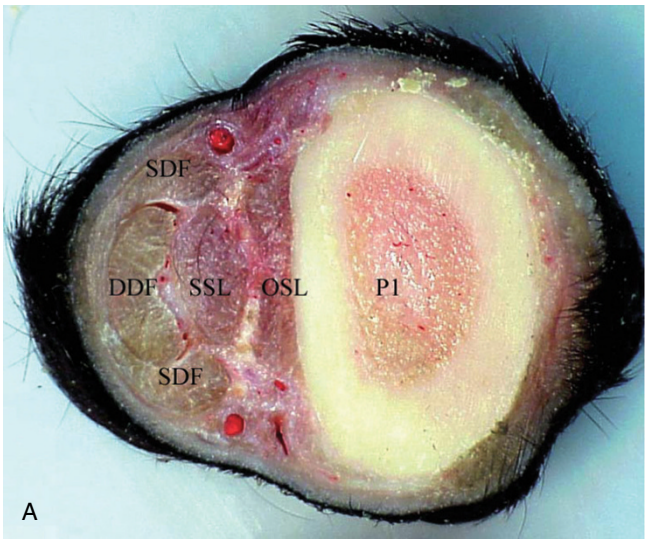


Figure 3.102. (A) Zone P1B is considered the middle of the proximal phalanx (P1). The bone contour of P1 is flat at this level. The fibers of the SDFT incline abaxially to the medial and lateral aspect of the DDFT as they begin to divide into branches. The DDFT develops a central depression on its dorsal surface and becomes bilobed in appearance. The SSL becomes more rounded in appearance. (B) The ultrasound image shows that the SSL appears hyperechoic relative to other structures. When the SSL is in focus, the DDFT is hypoechoic, necessitating that each structure be imaged independently. The SDFT branches cannot be adequately imaged with this transducer orientation and will require a more abaxial location of the transducer (Figure 3.103). As the SDF begins to form branches, it forms a distal ring of the manica flexoria dorsal to the DDF, and this should not be mistaken for an abnormality. (B) The OSLs incline more axially on P1 ultimately blending together to form a rectangular structure (C). *Source*: US image courtesy of Dr. Caitlyn Horne.

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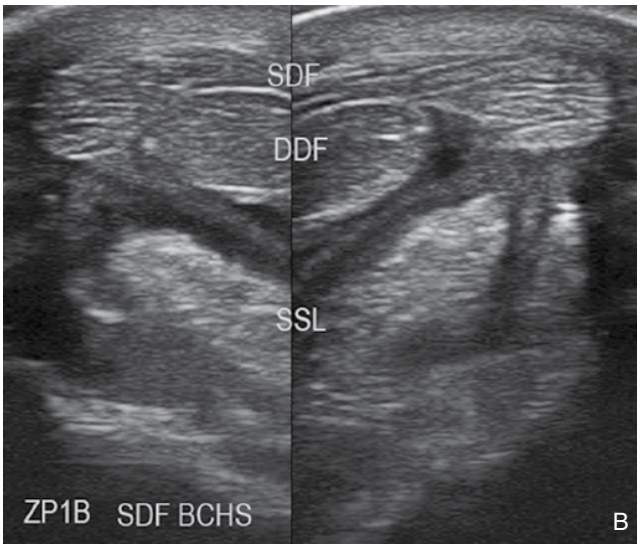
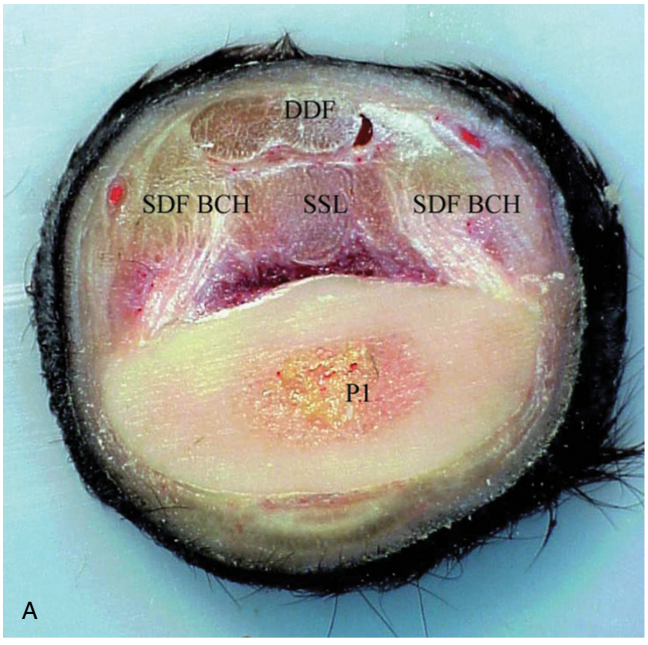


Figure 3.103. (A and B) To examine the SDFT branches, the transducer must be placed more abaxially, which allows the individual SDFT branches to be imaged as they incline to their insertion onto the medial and lateral aspects of proximal P2. *Source*: US image courtesy of Dr. Caitlyn Horne.



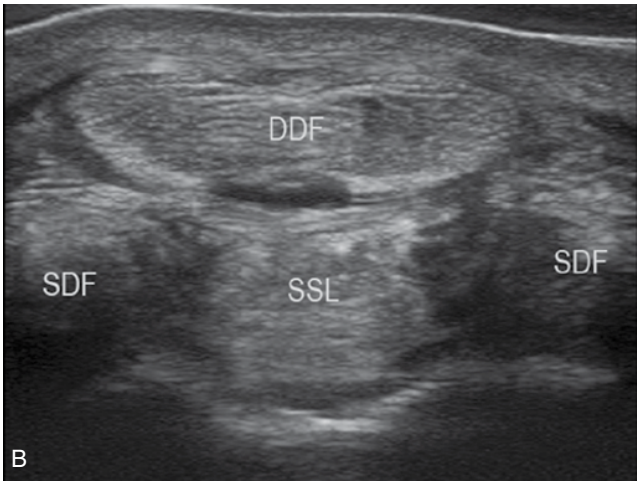
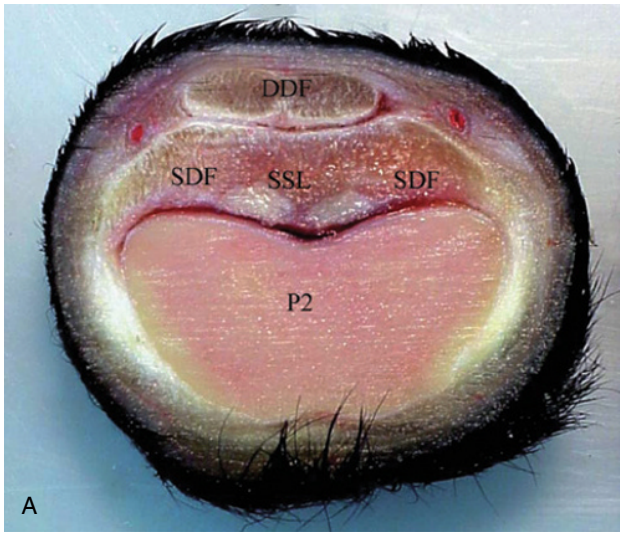


Figure 3.104. (A) In ZP1C the DDFT and the SSL are the only structures easily imaged when the transducer is on midline in a palmar position. The DDFT is bilobed and now expands in a medial to lateral direction. The SSL is rounded just prior to insertion onto palmar P2 and the middle scutum. (B) The SDFT branches are more abaxial and difficult to image with this transducer orientation. *Source*: US image courtesy of Dr. Caitlyn Horne.

CSA must be obtained as this measurement is often used in the rehabilitation of tendon and ligaments. CSA may increase for 2–3 weeks after an injury due to persistent inflammation. A repeat scan at 2 weeks will provide a more accurate assessment of maximum lesion and tendon CSA to compare back during rehabilitation. Normal values for Standardbred and Thoroughbred horses have been published.[80] If normal values for CSA for a structure are not available, they can be obtained from the opposite limb at the same level. As the tendon/ligament remodels the CSA will progressively diminish during the rehabilitation process. Increases in CSA during the rehabilitation period must be carefully assessed and often indicate that a reduction in the level of activity is warranted.

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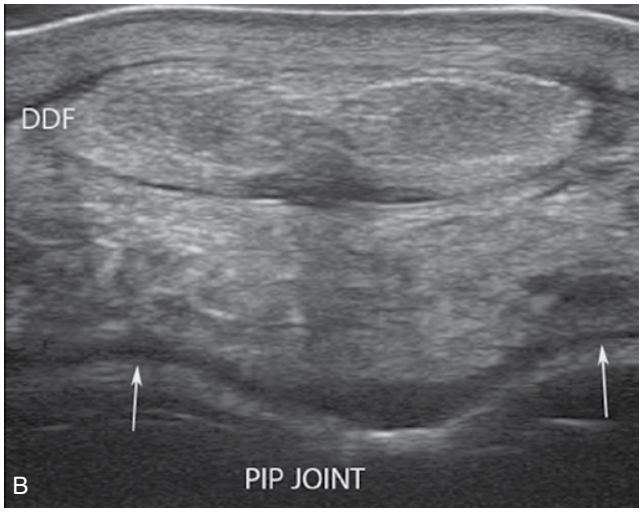
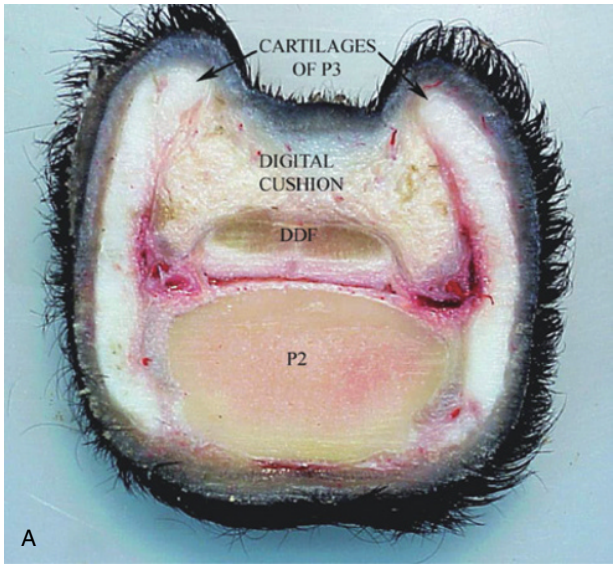


Figure 3.105. (A) Zones P2A and P2B are associated with the middle phalanx (P2). Zone P2A begins when the ultrasound examination visualizes the structures of the pastern crossing the proximal interphalangeal joint. The SSL, medial, and lateral SDFT branches and the axial and abaxial ligaments of the pastern joint blend together to form the cartilaginous attachment onto P2, called the middle scutum. (B) There is a normal hypoechoic area within the SSL just prior to insertion onto P2. The palmar/plantar aspect of the pastern joint including the articular cartilage can be seen (arrows). *Source*: US image courtesy of Dr. Caitlyn Horne.



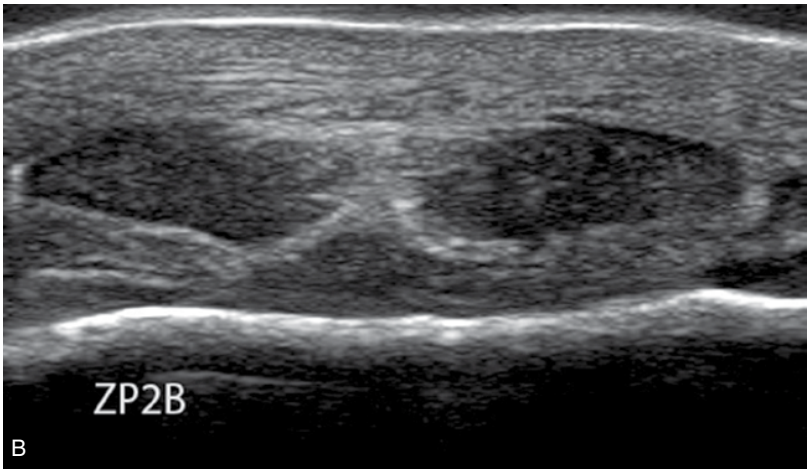
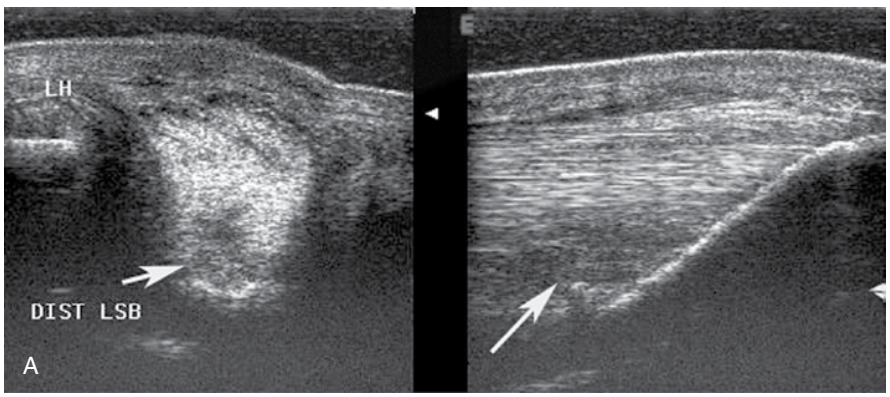


Figure 3.106. (A) Ultrasound imaging of Z2PB in the distopalmar aspect of P2 can be difficult due to the presence of the collateral cartilages of the distal phalanx. (B) The collateral cartilages interfere with proper placement of the transducer distal enough to assess the DDFT with a perpendicular orientation of the sound beam to the fiber pattern of the tendon. The shape of the DDF and some areas of scar (random disorganized fibrous material) can be assessed with this orientation. *Source*: US image courtesy of Dr. Caitlyn Horne.

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Figure 3.107. Placing the limb on a small block with the leg more caudal allows a more comfortable examination of the structures of the pastern region. Notice the standoff pad placed over the probe to move the near-field artifact away from the superficial structures.



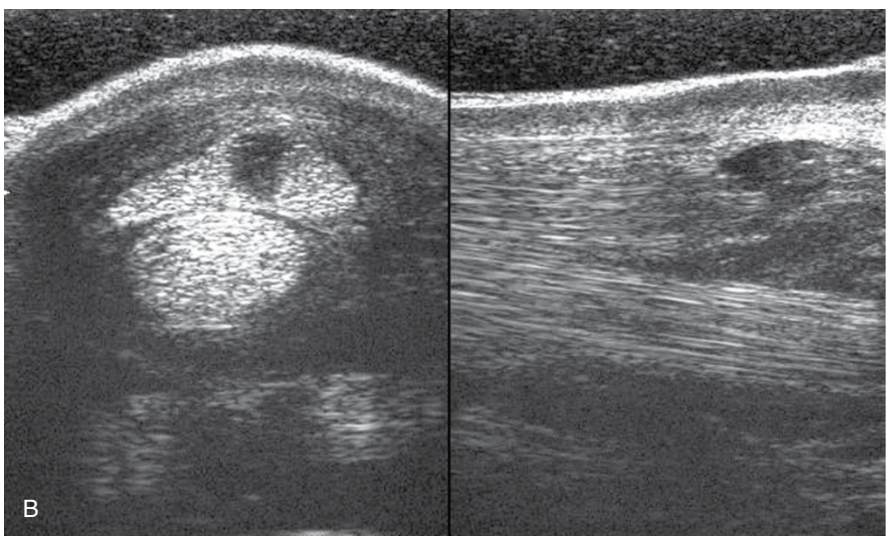


Figure 3.108. (A) Longitudinal and cross-sectional ultrasound image of a suspensory ligament attachment injury (arrows). Notice the loss of fiber pattern and the calcification at the attachment to the proximal sesamoid bone on transverse and longitudinal sections. (B) Longitudinal and cross-sectional ultrasound image of the SDFT with a core-type defect of the central aspect of the tendon.

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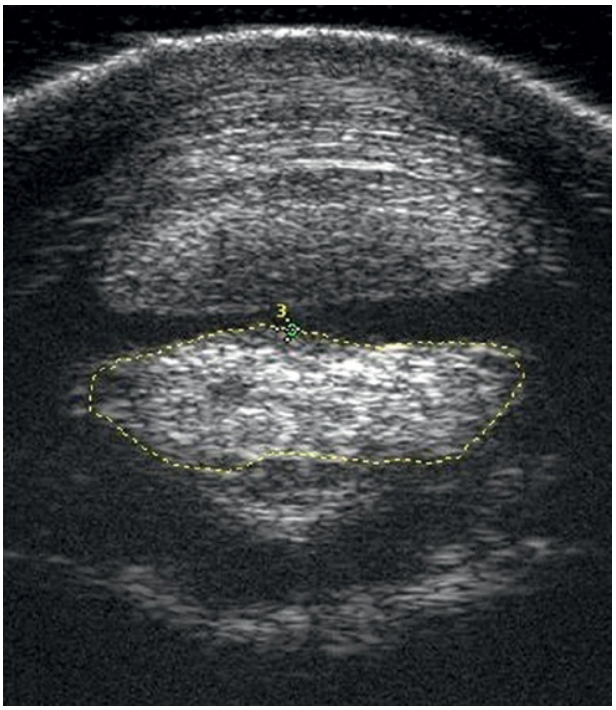


Figure 3.109. This is a straight sesamoidean ligament (SSL) injury. There is enlargement with an irregular outline associated with the heterogeneous appearance of the ligament. Tracing of the cross-sectional area is helpful to follow the rehabilitation process. Tendon and ligament damage is represented by changes in size, shape, architecture, position (with respect to surrounding anatomy), and fiber alignment.

The tendons and ligaments of the distal limb (and to certain extent proximal limb as well) have been found to have consistent but unique shapes at each level of the examination.[22,31,40,41,46,57,72–74,77,82,85,90,99] It is normal to have these structures change shape as they course distally in the limb. Therefore, if the examiner perceives a structure to have an abnormal shape, then it should be compared with the same structure in the opposite limb at the exact same level. It is also helpful in those horses with a change in shape to assess their position with respect to the surrounding anatomy. Architecture (or texture) is a subjective assessment of the ultrasound image attempting to describe morphological change or damage. Terms used to describe the architecture of an image relate to the tissue's ultrasonographic intensity. Architectural change is described as a change in echogenicity or the whiteness/brightness of a structure. Echogenicity is a function of each structure's particular density based on several things including cellular composition, fiber alignment, and blood supply. Alterations in echogenicity are subjective interpretations and have been described with the terms isoechoic, anechoic, hypoechoic, and hyperechoic. Isoechoic implies a normal echogenicity, while hypoechoic and hyperechoic implies less than and more than isoechoic, respectively. Anechoic implies the structure (or lesion) is mostly black. Fluid is often considered anechoic. In general, the denser the structure is, the more echoes it returns, and the whiter the structure appears.

Fibers have a parallel alignment in most normal tendon and ligamentous structures. The fibers of tendinous tissue are more uniformly distributed, while the fibers in ligamentous tissue are more multidirectional. This parallel fiber bundle alignment is best assessed on longitudinal images. Injury to and inflammation of tendons and ligaments can disrupt fiber bundle alignment. Subtle changes in fiber alignment are best seen on the longitudinal plane images. More severe fiber bundle alignment changes can begin to be appreciated on transverse images. The longitudinal image must be obtained through the affected tissue as seen on transverse images. Damage seen on cross section should be confirmed on longitudinal orientation. Fiber disruption seen as echolucent areas surrounding the fibers is compatible with hemorrhage and edema seen with acute injuries. Nonparallel or random fiber alignment without echolucent fluid content is compatible with chronic injury. Tendon injury can be focal or generalized such that the distribution of fiber damage can be quite variable. Fibroblasts migrate into the damaged area and begin to deposit collagen and form granulation tissue. This collagen is laid down randomly, and cross-links are produced between the fibers. This random disorganized tissue appears hypoechoic on ultrasound and can persist for some time post-injury. Rehabilitation with increasing levels of exercise precipitates remodeling of the collagen and a return of the echogenicity and alignment toward normal.

LIMITATIONS OF ULTRASONOGRAPHY

The quality of the image is directly related to the operator, the equipment, and the anatomical area being examined. This imaging tool is influenced by the skill of the operator more than any other imaging technique. The operator is responsible for positioning and steering the sound beam as well as determining the equipment settings during image acquisition. Artifacts are easily produced and can create inaccuracies in the image that can significantly compromise interpretation. It is also recognized that ligaments have normal anatomical variation in their fiber orientation and this can cause inconsistencies in their echogenicity on ultrasound examination (such as in the collateral ligaments of the distal interphalangeal joint). Artifacts most often involve operator error and an assortment of sound–tissue interactions that may or may not be controllable. One common but easily correctible artifact is created by inadequate skin preparation. Inadequate skin prep leads to poor transmission of sound and a corresponding dark image. The limb should be clipped and prepped to maximize skin contact and sound transmission. High-frequency transducers produce better images but often require shaving the area to be examined with a razor. Improving skin–transducer contact is critical to obtain the best images possible. Another common artifact frequently created by the operator occurs when the ultrasound beam is off incidence to tendinous/ligamentous structures and tissue interfaces. Off incidence artifact

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occurs when the ultrasound beam is not at 90° to the fibers of the target structure that reflect the returning echoes away from the transducer. This creates a hypoechoic area that mimics a lesion(s) within the targeted structure. Improper gain and power settings and inappropriate focal zone position can also lead to suboptimal images. Near-gain and power settings that are set too high reduce the ability to differentiate the tissues. Gain settings should be adjusted to produce a uniform gray scale across the entire image. Focal zones are variable in number and position and should be adjusted to the level of the specific structure(s) to optimize the image quality.

Artifacts that are created by sound–tissue interactions include acoustic enhancement, refractive scattering (edge artifacts), reverberation, and acoustic shadowing. Acoustic enhancement occurs when sound passes through a fluid structure. Fluid attenuates sound less than soft tissue. Enhancement results from relatively increased amplitude of deeper echoes by an overlying structure of low attenuation. This creates the appearance of increased echogenicity of the tissue deep to the fluid-filled structure (far-field enhancement). The most common structures that can cause enhancement artifacts in the musculoskeletal examinations are veins and arteries. Refractive artifacts are generated from the edge of a curved surface and are created because part of the sound beam is refracted off the curved structure and does not return to the transducer. Shadow artifacts are displayed distal the lateral margins of curved or cystic structures. Edge artifacts are consistent and particularly troublesome when examining specific anatomical locations. The SDF, DDF, and ICL are superimposed over the SL in the metacarpus and metatarsus, and the size differential (SL is wider than the SDF/DDF/ICL) between these structures allows refractive or edge artifact to occur in the SL. In addition, the presence of vessels over the SL creates enhancement artifacts in the image. The proximal plantar metatarsal region is another good example of how these artifacts can affect the quality of the ultrasonographic examination of the origin of the SL (Figure 3.110). At this location there are large oval/rounded structures of different echogenicity (SDF, DDF, ICL, and origin of the SL) all with a slightly different fiber orientation and acoustic impedances. The operator must find an acoustic window that minimizes the interference of the splint bones as well as prevents or limits the development of refraction and enhancement artifacts from the overlying tendons and vessels. Dilatation of the vessels in this area due to the inflammatory response of the damaged SL makes scanning this area even more problematic and significantly compromises interpretation of the SL morphology. Scanning at a more plantaromedial to dorsolateral oblique orientation may be necessary to improve image quality, but the experience of the operator scanning areas such as the proximal plantar metatarsus will improve the diagnostic accuracy of the ultrasound exam, but the image may still be suboptimal.

Injuries to the tendons inside of the digital sheath can cause considerable synovial reaction, which may be associated with annular ligament constriction syndrome[28,33] (Figure 3.111). Because the SDF and DDF tendons and the macular flexoria exist inside the fetlock

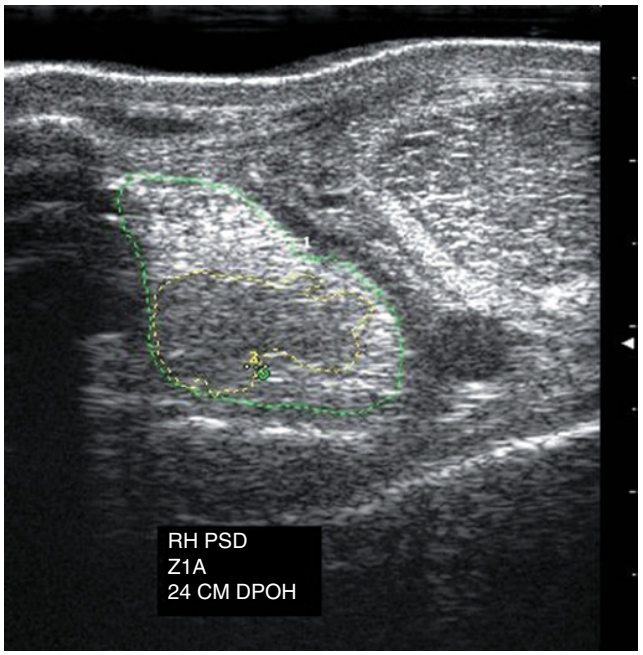


Figure 3.110. Transverse scan of the proximal plantar metatarsal area with the inherent artifacts that occur in this area due to edge shadowing, acoustic enhancement, and acoustic shadowing that occur because of the bony anatomy and overlying SDF and DDF and vessels. This lesion was found to be a false positive when the horse had a normal MRI examination of this area. The operator must find an acoustic window that minimizes the interference of the splint bones as well as prevents or limits the development of refraction and enhancement artifacts from the overlying tendons and vessels.

canal with the restricting annular ligament, it may be difficult to visualize certain types of injuries with ultrasonography. Recent introduction and use of tenoscopy or endoscopy to explore the sheaths and bursae of the equine limb have allowed comparison of ultrasonography and tenoscopy for identifying tendon/ligament injury.[37,75,76,92,104] Tenosynovitis of the DFTS may result from tears in the DDFT or SDFT, manica flexoria, or other structures that communicate with the synovial fluid and thus can generate an intense synovial response in a sheath or bursa. However, these lesions can be identified confidently only by tenoscopy, which also permits appropriate lesion management.[92] Longitudinal tears of the DDFT should also be suspected if ultrasonographic changes are present lateral or medial to the border of the DDFT, but tenoscopic examination of the tendon sheath is essential to establish an accurate diagnosis and an effective treatment.[104]

Ultrasound remains a practical, inexpensive, and readily accessible imaging technique for soft tissue injuries of the horse. However, with the recent introduction of magnetic resonance imaging (MRI) to equine musculoskeletal imaging, soft tissue and bone injuries can be evaluated in detail not capable with any other imaging technique. MRI is now considered the gold standard to assess lameness originating from the carpus and tarsus distally (especially of the foot within the horny hoof capsule), but it is not always practical or necessary to make a diagnosis. Lesion(s) seen with standard ultrasonographic imaging are not typically candidates for

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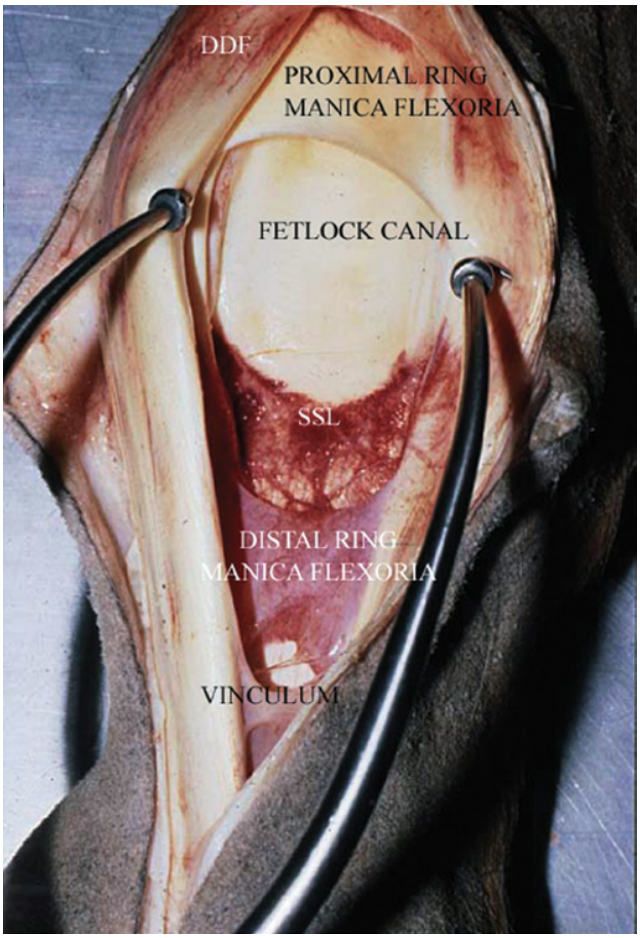


Figure 3.111. The structures within the digital flexor tendon sheath (DFTS) are exposed via a longitudinal incision through the annular ligament and the SDFT at the level of the fetlock joint. The unique anatomy at this level begins with the mesotendinous attachments (not seen in this section) to the DDFT at the medial and lateral borders in the proximal sheath. Progressing distally the SDFT produces the proximal ring of the manica flexoria encircling the DDFT. Just distal to the manica flexoria, the SDFT and DDFT are bound within the fetlock canal by the primary annular ligament (PAL), which has been incised in this section. Just distal to the fetlock canal, the SDFT makes another ring (smaller) around the DDFT called the distal ring of the manica flexoria. Just distal to this ring, the vincula attach to the midline of the DDFT on its dorsal border.

MRI examination. While ultrasonography and MRI remain the optimal choices for soft tissue imaging, it remains unclear in all cases which is the most effective imaging tool. In humans there are many studies that have been performed that compare the diagnostic accuracy and utility of ultrasound compared with MRI for a variety of orthopedic problems. These studies are lacking in horses and need to be performed. A limited number of studies have been performed and have looked at the collateral ligaments of the distal interphalangeal joint and the proximal plantar region.[6,35,36] In those studies a negative ultrasound study does not rule out an abnormality and positive ultrasonographic needs to be regarded with caution. It is hoped that further experience comparing ultrasound with MRI along with the completion of more comparative studies between MRI and ultrasound will identify specific indications for each imaging modality and improve our capabilities as ultrasonographers.

ULTRASOUND TO EVALUATE JOINT INJURY

Joint injury, osteochondrosis, and degenerative joint disease are significant causes of lameness in the horse.[68,69] An accurate diagnosis of the cause of joint pain can prove critical for selecting the most appropriate treatment(s) and rehabilitation. Intra-articular diagnostic anesthesia should be utilized to localize a lameness to a particular joint. Radiology, ultrasonography, thermography, nuclear imaging, CT, and MRI all have a place in lameness diagnostics, and each may be warranted in selected cases.[50,62] However, many of the joint problems seen in the horse can be effectively imaged with a combination of radiographs and ultrasound. Good-quality baseline images are critical to provide a diagnosis but also will be helpful in reevaluation of the joint in the future. The radiographic exam is most effective at evaluating the bony structure of the joints of the equine limb. In some joints, there can be poor correlation between clinical and radiographic findings, or the radiographic study may be inconclusive.[55,62] In these types of cases the cause of lameness is presumed to be soft tissue injury, and often empirical treatment is instituted. A complete ultrasonographic exam is indicated in these cases to evaluate the periarticular tendons and ligaments, the joint capsule, and the joint fluid, as well as to provide valuable information about the cartilage and subchondral bone surface.[2,17–20,58,78,79] Ultrasonography has the added advantage of providing immediate, detailed information through a range of motion (in extension and flexion). However, ultrasound provides a relatively limited field of view when compared with other imaging techniques making global survey of a joint to be time consuming. When used in combination, radiography and ultrasonography are complementary and can supply more information about an orthopedic problem than when each modality is used alone.

The clinician must understand the advantages and limitations of each imaging modality as well as the appearance of the normal anatomy and pathophysiology characteristic with each technique. When utilizing diagnostic ultrasound to evaluate joints, it is necessary to become familiar with the appearance of many of these structures in transverse, longitudinal (sagittal), and frontal planes. Normal anatomical variations exist, and it is important that the clinician recognizes these variations as normal to prevent developing an inaccurate diagnosis. The opposite limb can and should be used for comparison when possible recognizing that disease can occur at the same location in the opposite limb.

In general, the stability of a joint is provided by the congruent contours of the joint surfaces of the bony column, the collateral (and in some joints the intra-articular) ligaments, the integrity of the joint capsule, and the surrounding tendons and ligaments. In the proximal limb, various muscle groups and tendons that cross the joints provide additional stability. Most joints of the appendicular skeleton in the horse are designed to work in the sagittal plane with flexion and extension being the

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predominant range of motion. This necessitates a joint to have a redundant joint capsule at the dorsal and palmar/plantar surfaces. In addition, tendon and ligaments that pass over joints with a wide range of motion typically have sheaths or bursae to allow these structures to effectively glide across the joint surface throughout their full range of motion. In addition, many tendons that cross a joint have retinacular bands and/or annular ligaments to maintain the position of these structures through the range of motion of that joint. Development of fluid or effusion within the joint in any of these structures needs to be accurately defined to effectively manage the cause of the effusion.

INDICATIONS FOR ULTRASONOGRAPHY OF JOINTS

The use of diagnostic ultrasound is an excellent adjunct to the radiographic examination of a joint. While the soft tissue structures associated with the joint can be carefully evaluated, ultrasound examination can prove superior at detecting bony changes such as the early periarticular changes (osteophyte) manifested in osteoarthritis than radiographic examination. Ultrasonography can be utilized in the examinations of a joint to assess the cartilage, subchondral bone surface, and some of the intra-articular soft tissues of the joint. A complete ultrasonographic examination of joints requires examination of the joint in a weight-bearing position as well as imaging the joint in flexion (or through a range of motion). In joints that commonly manifest articular cartilage damage such as the fetlocks and carpi, the major weight-bearing regions of the joint that are not accessible with ultrasound when the limb is weighted should be examined during joint flexion. Hyperextension injuries that impact the dorsal articular surfaces of the joint (such as the medial and lateral proximodorsal proximal phalanx in the fetlock) can also be more easily identified with the joint flexed.

Joints with radiographically obvious lesions should not be excluded from an ultrasound examination because coexisting soft tissue-type problems may alter the prognosis. Some bony abnormalities evident on radiographic exam are related to soft tissue attachment sites and require an ultrasound examination to more carefully assess the extent of soft tissue injury (i.e. apical sesamoid fracture should be assessed for the degree of SL involvement to provide a more accurate prognosis). Some intra-articular fragments may occur due to avulsion of soft tissue attachments (i.e. lateral or medial malleolar fragments with collateral ligament attachments), and ultrasound can help determine the full extent of injury to these structures. Ultrasound can also assist with the surgical approach to the fragment by documenting the position as intra- or extra-articular and fixed or mobile. Ultrasonography of a joint injury is definitely indicated when the radiographic study is inconclusive especially when there is synovial effusion, joint capsule thickening, or focal swelling of any of the soft tissue structures associated with the joint (osteochondrosis, intra-articular ligament pathology, periarticular tendon or ligaments, etc.). In addition, there are some regions of the limb where diagnostic radiographs can be difficult to obtain (such as the pelvis and coxofemoral joint). Ultrasonography has proven very helpful to assess both the soft tissues and bony contours of this joint.

The diagnostic workup of a joint may utilize ultrasound to guide interventional procedures to improve the clinical information in the workup of a joint. Visualization of needle placement into the joint can document the placement of local anesthetic or medication into the appropriate structure (joints, sheaths, or bursae) particularly in areas where it can be difficult to ensure accurate needle placement (navicular bursa, articular facets of the cervical spine, shoulder joint, bicipital bursa, coxofemoral joint, etc.).[10,16,56,60,93] Documenting needle placement and seeing that the injection is within the synovial space (of joints, sheaths or bursae) are important to reduce false-positive and false-negative results. Collection of fluid samples can be directed by ultrasonography improving the recovery rate in situations like septic arthritis. Ultrasound of a joint may guide the surgical approach to and/or the intraoperative retrieval of bony fragments (i.e. lateral malleolar fractures of the distal tibia).

Lacerations and puncture wounds to the distal limb can traumatize tissue and introduce contaminates such as hair or debris into a synovial structure. Such foreign material can act as a nidus for infection and inflammation inducing immediate and delayed immune responses. Wounds or punctures that are close to a joint should be carefully evaluated for communication with and possible contamination of the joint. The ultrasonographic appearance of hair shafts within synovial structures has recently been reported in horses. Ultrasonographic evidence of intrasynovial contamination should direct the clinician to be more aggressive at removing the offending material.

EQUIPMENT AND TECHNIQUE

The same equipment utilized for tendon and ligament examinations is ideal for joint evaluations. The joint should be prepared as for a tendon or ligament examination with the joint being clipped (and possibly shaved, depending on the operator's preference); cleansed to remove dirt, hair, and other surface debris; and liberally coated with a conducting gel. Most joint examinations will require clipping and prepping the entirety of the joint surface. The majority of the structures evaluated during joint ultrasonography are superficial in location well within the focal zones of most high-frequency transducer. Flat-face linear transducers of 10–18 MHz are often used because of superior near-field resolution and broad field of view. It is helpful in evaluating most of the structures of the joint at a scan depth of 2–4 cm. Standoff pads move the superficial structures out of the near-field artifact and into the near focal zone of the probe. Standoff pads also improve the footprint by conforming to the skin surface. Lower-frequency transducers (5 or 7.5 MHz) may be necessary to image deeper aspects of the joint such as the caudal aspect of the stifle. In the proximal limb the joints are farther from the skin due to muscle coverage. The overlying muscle mass provides a good window for the

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sound transmission to the deeper aspects of the joint. However lower-frequency probes may need to be used, which have poorer resolution and somewhat compromised image quality. Microconvex or macroconvex probes have the added advantage of a divergent beam that allows image acquisition from a smaller skin contact point.

Joint ultrasound requires a specific knowledge of the anatomy of the joint. Unlike the palmar/plantar metacarpal exam where the transverse images are typically acquired first, it is helpful to begin the examination of many joints in the longitudinal scan plane. Identifying the articular surfaces of a joint provides orientation for the rest of the examination. Global assessment of a joint can be time consuming requiring multiple scan plane. The extensor and flexor surfaces (and the corresponding soft tissues such as the extensor/flexor tendons that cross these areas) and the collateral ligaments and the articular surfaces (weight-bearing and non-weight-bearing areas) of the joint all should be examined. For example, examination of the fetlock, which is considered a simple joint, is divided into quadrants. The quadrants of the fetlock are dorsal, palmar/plantar, medial/lateral, and a dorsomedial/dorsolateral scan plane.[23,27] In more complex joints additional scan planes may be indicated due to additional structures associated with the joint (i.e. the stifle has the patellar ligaments, long digital extensor tendon, popliteal tendon, etc.). This emphasizes that the examiner must have developed experience in the use of ultrasound and knowledge of the anatomy of the joint being examined.[3,9,12,13,15,16,25–27,32,42,44,64,66,71,83,84,86,88,89,93,95–98,100,103]

All tendinous or ligamentous structures should be evaluated in both transverse and longitudinal scan planes. A description of the scan plane used to acquire the image should be documented on the stored image. Scan planes should be labeled similar to the nomenclature utilized for radiographic projections. These oblique projections are described by the direction that the central ray of the primary beam penetrates the body part of interest, from the point of entrance to the point of exit. Soft tissue structures should be examined first followed by the cartilage and subchondral bone surfaces. Much of the cartilage and subchondral bone surface in the non-weight-bearing areas can be imaged in the standing horse. However, dynamic examination of the cartilage and subchondral bones surfaces of the weight-bearing areas of the joint requires lifting the limb, placing the probe on the dorsal joint surface all the while attempting to place the probe 90° to the subchondral surface while flexing and extending the joint. While this maneuver requires considerable practice, it can be expedited by having someone hold the limb (or placing the limb in a stand) while the examiner positions the transducer while manipulating the limb. Maximally flexing the leg is necessary to access the major weight-bearing areas of a joint. Unfortunately, flexion of an inflamed joint will likely be resented making it more difficult to evaluate the weight-bearing surface.

The joint examined and the area of interest within the joint are, in many cases, directed by the signalment (and by any prior radiographic findings). For example, racehorses have a high incidence of problems involving the dorsal articular surfaces of the high-motion joints, particularly the fetlock and carpus; younger horses may have developmental orthopedic disease that, when involving a joint, frequently involves the trochlear ridges of the stifle, the distal intermediate ridge of the tibia, or the trochlear ridges of the talus; Warmbloods often have a similar distribution of osteochondrosis lesions, and they also tend to develop periarticular changes in the coffin, pastern, and fetlock joints. In each case, it is important to perform as complete an examination of the joint as possible because many joint conditions involve multiple structures and/or multiple areas of the joint. For example, osteoarthritis tends to cause cartilage degeneration and thinning in the loaded areas of the articular surface. Along with these cartilage changes, periarticular remodeling occurs in the form of osteophyte and/or enthesophyte formation. Joint capsule thickening, with or without metaplasia, is also a common feature of osteoarthritis. Many of the osteochondral fractures that occur in the joints of performance horses occur on the dorsal surface due to hyperextension of the joint. But hyperextension may also cause damage to the palmar/plantar soft tissues (flexor tendons, SL, and distal sesamoidean ligaments), which can significantly alter the prognosis.

Dynamic examination of the joint as described for the examination of the weight-bearing cartilage allows the evaluation of redundant parts of the joint capsule in high-motion joints. For example, the dorsal surface of the fetlock joint has redundancy of the joint capsule that is relaxed when the horse is bearing weight on the limb. Flexion of the fetlock tenses the dorsal aspect of the joint capsule, allowing better evaluation of this part of the joint. Tearing of the dorsal joint capsule is more accurately imaged if the structure is under tension while the joint is in flexion. Flexion and extension of the joint during ultrasonographic examination can also be helpful in demonstrating mobility of an osteochondral fragment and in evaluating fluid movement within the joint.

The same joint on the contralateral limb should serve as a comparison when evaluating articular and periarticular structures. However, there are two precautions: (1) the examiner must make certain to use the same orientation of the transducer and image the contralateral joint at exactly the same location when making comparisons, and (2) some joint conditions, in particular osteochondrosis in young horses and osteochondral fragmentation in racehorses, can be bilateral.

The major obstacle to the effective use of diagnostic ultrasound in the examination of joints is the need to understand the unique anatomy of each joint and appreciate how the various tissue types are influenced by sound–tissue interactions. It is also important to understand the information and misinformation (artifacts) created during any ultrasound evaluation (see limitations of ultrasound earlier in this chapter). There are many texts that describe the anatomy of specific joints in the horse[31,46,85,90] and the use of diagnostic ultrasound in the examination of joints.[2,17,18,20,21,38,39,58,68,69,78,79,101,102] It is recommended that these textbooks be available as reference materials to assist with the examination. Descriptions of the ultrasonographic examination of the specific joints are beyond the scope of this chapter and will not be discussed. However, a discussion of some general features unique to the use of ultrasound in the examination of joints follows.

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ULTRASONOGRAPHIC APPEARANCE OF PERIARTICULAR STRUCTURES

Collateral Ligaments

The joints of the appendicular skeleton in the horse are designed to work predominantly in the sagittal plane with flexion and extension as the primary range of motion. A major component of the stability of a joint is provided by the periarticular soft tissues but in particular the collateral ligaments. The collateral ligaments are designed to impart stability to the joint throughout its entire range of motion. This unique task is accomplished either with paired structures (i.e. multiple bundles of the fetlock, tarsal joints) or by unpaired structures (i.e. single bundle of coffin joint). Knowing the anatomic arrangement of the collateral ligaments of each specific joint becomes extremely important as the ultrasonographic appearance of these ligaments is determined by the fiber orientation. Most of the collateral ligaments have a uniform fascicular orientation and, therefore, a homogeneous ultrasonographic appearance. Some have a mixed fascicular arrangement, which gives them a heterogeneous appearance. The lateral collateral ligaments of the stifle, hock, and elbow and the collateral ligaments of the fetlock and carpus have spiral or crossed fibers.[23] This mixed arrangement of fascicles within the CLs is one of the arrangements that allow the collateral ligaments to function in both extension and flexion (Figure 3.112). In the tarsocrural joint, the superficial collateral ligaments are under tension while the limb is extended and relaxed, whereas while the limb is flexed, the short collateral ligaments are tensed only when the tarsus is flexed.[99] Therefore, the short collateral ligaments should be examined while the leg is flexed. (See tarsus and tibia in Chapter 5 for more information.)

Tendons and Ligaments

As discussed previously tendons and ligaments appear as moderately echogenic structures with relatively well-defined margins, and injury is recognized with ultrasound by changes in size, shape, architecture, position, and fiber alignment. Ultrasonographic examination of the tendons at the level of the joint is no different except from a couple of perspectives. First, the examiner must pay particular attention to the course of the tendon or ligament as it courses over a joint because there can be quite remarkable changes in direction. This becomes important in that the transducer must constantly be adjusted to maintain a perpendicular orientation to the structure to prevent beam angle artifact. Second, while injury is recognized as in other areas, the periarticular portions of tendons have sheaths (and occasionally bursae) associated with them. Injury to these tendons/ligaments may also manifest as effusion within the sheaths or bursae. To confuse the matter even more, the effusion may occur without structural damage to the tendon or ligament. Therefore, effusion of the sheaths and bursae associated with these structures requires careful evaluation to define the cause and to differentiate the fluid accumulation as separate from joint effusion.

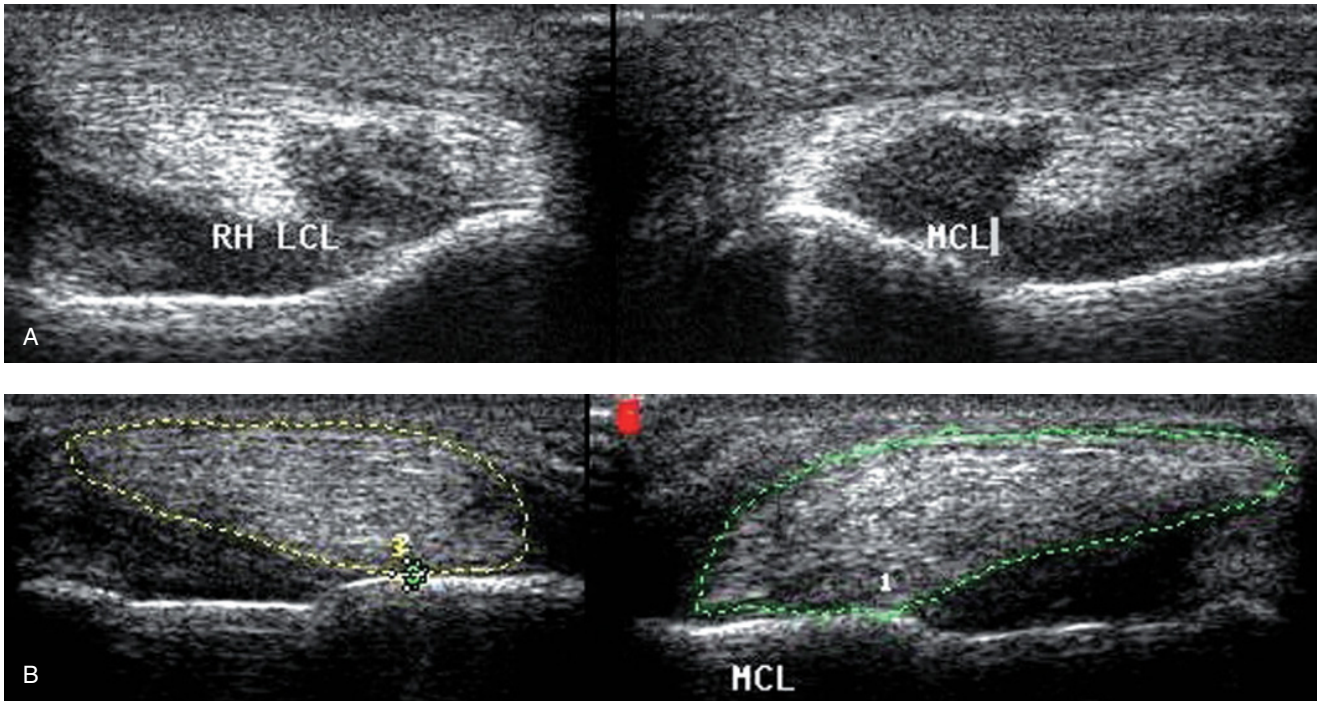


Figure 3.112. A mixed arrangement of fascicles within the CLs of many joints is one of the unique arrangements of ligaments that allow a collateral ligament to function in both extension and flexion. This mixed arrangement of fibers can produce a heterogeneous appearance of the collateral ligament on ultrasound examination. (A) This is a transverse ultrasound image of the CLs of the fetlock joint that appears to have a hypoechoic lesion within the dorsal aspect of the medial CL. However, the lateral CL has the same appearance and similar cross-sectional area, suggesting that this is a variation of normal in this horse. (B) This transverse image of an MCP CL demonstrates a thickened CSA with a heterogeneous fiber pattern, consistent with chronic desmitis.

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ULTRASONIC APPEARANCE OF THE JOINT

Joint Capsule, Synovium, and Synovial Fluid

The joint capsule is primarily connective tissue with low cell density. The joint capsule is continuous with the periosteum or perichondrium, but it does not insert directly at the perimeter of the articular cartilage (Figure 3.113). There tends to be redundancy of the joint capsule in the high-motion joints, such as the fetlock, carpus, tarsus, and stifle. In fact, this redundancy can cause relaxation artifacts if the redundant aspect of the joint is examined in the weight-bearing position rather than during flexion. Inflammatory conditions within the joint can cause capsular changes that include thickening (initially due to hemorrhage and edema, and later fibrosis), calcification, and insertional capsulopathies (enthesophytes). Osteoarthritis in high-motion joints typically involves periarticular changes that begin with congestion and thickening of the joint capsule. The synovial membrane becomes hyperplastic, and in more chronic cases, synoviocyte metaplasia may lead to the formation of synovial chondromas, which are seen as nodules of cartilage. These nodules may undergo endochondral ossification, resulting in ovoid radiodense bodies within the joint capsule.

Synovitis is an early response to joint injury and usually progresses to capsulitis; however, thickening of the thin synovial membrane is difficult to appreciate ultrasonographically. Synovial effusion is helpful in assessing synovial membrane proliferation and thickening. In cases of severe synovitis, synovial fluid that is fibrinous, cellular, or hemorrhagic can cloud evaluation of the joint capsule and synovium.

There is usually minimal synovial fluid in a normal joint, so the joint capsule and synovium are normally in close apposition with the articular cartilage. Synovial fluid accumulation occurs with many abnormal joint conditions, so thorough evaluation of the various articular structures is indicated when synovial effusion is present. The ultrasonographic properties of the synovial fluid can help assess the nature of the fluid (Figure 3.114). Effusion associated with acute synovitis typically is anechoic, with occasional fibrinous accumulations. Hemorrhage or sepsis increases the cellularity and fibrin deposition within the joint and appears as echogenic fluid. Ultrasonography can aid in aspiration of synovial fluid by identifying areas of fluid accumulation. Utilization of this technique, particularly in the deeper joints such as the hip and the shoulder, can minimize trauma to the joint and reduce the potential for blood contamination of the fluid sample.

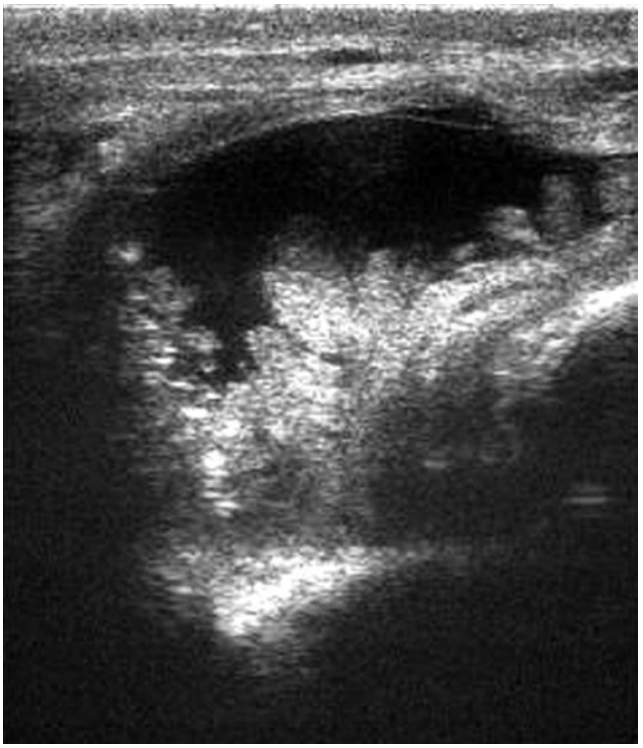


Figure 3.114. Synovial fluid accumulation occurs with many joint, sheath, or bursal conditions. A thorough evaluation of the various structures of the joint or synovial sheath is indicated when effusion is present. This image demonstrates a proliferative synovial membrane from a chronic synovial effusion that was due to a previously undiagnosed osteochondrosis lesion of the medial malleolus of the tibia.

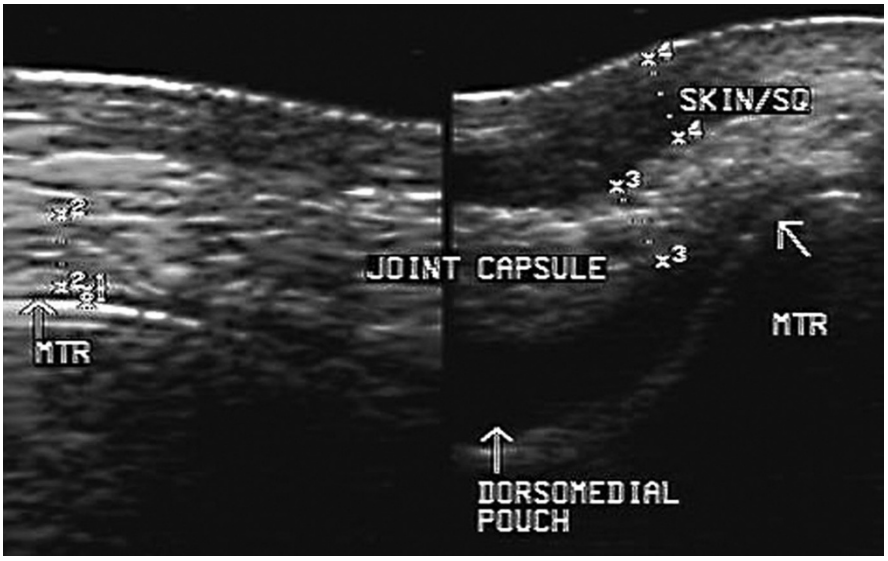


Figure 3.113. Ultrasound image of the dorsomedial aspect of the tarsocrural joint. There is mild edema of the subcutaneous tissues and synovial effusion within the joint, allowing visualization of the joint capsule. Fluid accumulation within the joint can allow delineation of the synovium.

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Articular Cartilage and Subchondral Bone Surface

With ultrasonography, reflections of the sound beam occur at interfaces between tissues of different material densities. Articular cartilage is predominantly water producing a sonolucent (anechoic to hypoechoic) image between the echogenic joint capsule/synovium and subchondral bone; however, the distinction between joint fluid and cartilage cannot always be appreciated. The ultrasound beam cannot pass through the bone, so the subchondral bone interface is seen as a dense, hyperechoic line that follows the normal contour of the joint surface. Normally, there are distinct soft tissue–cartilage and cartilage–bone interfaces. When synovial fluid is seen between the synovial membrane and the cartilage surface, the interface between the fluid and the cartilage may create a thin, echogenic line.[17]

To accurately define the thickness and to detect changes in surface and internal characteristics of cartilage, the sound beam must be perpendicular to the cartilage surface. Normal articular cartilage thickness in horses remains to be well defined in all but a few joints. A recent study in healthy adult horses showed that the mean cartilage thickness on the lateral and medial trochlear ridges of the talus and on the distal intermediate ridge of the tibia was 0.57, 0.58, and 0.70 mm, respectively.[97] The trochlear ridges of the talus were chosen in that study because of the high incidence of osteochondrosis and the potential for cartilage thickening at these sites. Fetlock cartilage thickness was found to be thicker proximally (0.8 mm) more than distally (0.4 mm).[23] In humans, cartilage thickness varies somewhat between joints and even within a particular joint. For example, in the human knee the cartilage thickness on the femoral condyles normally ranges from 1.2 to 1.9 mm, with cartilage thickness on the medial condyle being less than that on the intercondylar notch and the lateral condyle.[2] From the limited number of equine studies and based on clinical impression, there is also variation in cartilage thickness both between and within joints in horses. Compare, for example, the difference in articular cartilage thickness between the lateral and medial trochlear ridges of the femur in the normal femoropatellar joint. The cartilage is thinner on the medial trochlear ridge; this is normal and should not be mistaken for a pathologic process.

Normal Variations

Articular cartilage has a fairly uniform thickness and a smooth contour. There are however a couple of areas where cartilage thickness and contour vary normally. Synovial fossae occur in some joints being bilaterally symmetrical and most often located in a non-weight-bearing part of the articular surface (e.g. humeroradial, tarsocrural, and coxofemoral joints). If an area is found that is of concern, the same site in the opposite limb should be evaluated for comparison (bearing in mind that some developmental lesions often occur bilaterally). The articular cartilage at the perimeter of a concave epiphysis and the cartilage at the center of a convex epiphysis are thickest.[55]

Immature Cartilage and Bone

Epiphyseal cartilage ossifies from a secondary ossification center, and the carpal and tarsal bones ossify by endochondral ossification. Ossification radiates out toward the articular surface so that articular cartilage of immature joints can appear thickened and the interface with the subchondral bone irregular. A more distinct cartilage–subchondral bone interface develops with maturity. Thus, measurements of articular/epiphyseal cartilage thickness in foals may vary greatly with age and possibly with the joint involved. In neonates, articular/epiphyseal cartilage thickness on the medial trochlear ridge of the femur can be as much as 25 mm; the articular cartilage is about 1 mm thick, and the remaining thickness is unossified epiphyseal cartilage, although the differentiation between the two types of cartilage is not appreciable ultrasonographically.[31] The thickness of articular cartilage can be quite variable depending on the stage of development and the amount of calcification that exists. As epiphyseal ossification continues, the thickness of cartilage covering the epiphyseal bone decreases.[38,39]

Osteochondral Lesions

Ultrasonography complements radiography in the early diagnosis of osteochondrosis in growing animals. In young horses, articular cartilage lesions associated with osteochondrosis can cause significant joint effusion, and in many cases, there are thickened cartilage or osteochondral fragments either *in situ* or free-floating within the joint. Areas of thickened cartilage or osteochondral fragment(s) are often apparent over an irregular subchondral bone surface (Figure 3.115). This irregular surface is thought to be associated with a continuum of changes that occur in the development of osteochondrosis lesions starting with a linear fissure or fold formation in the thickened articular cartilage that can progress to an undermined cartilage flap with lysis of the underlying bone.[68,69] Developmental abnormalities in the articular cartilage and subchondral bone can occur bilaterally, so use of the contralateral limb for comparison may be misleading.

Clinical signs often precede radiographic evidence of osteochondrosis in young horses. Radiographic assessment of osteochondrosis lesions commonly underestimates the extent of the articular lesion when compared with what is seen arthroscopically. Ultrasonography has proven to be a very useful imaging technique to evaluate the cartilage/subchondral surface and is indicated in any young horse with joint effusion suspected of having osteochondrosis. Serial examination of affected joints can be useful when deciding whether surgery is necessary. Progression of an osteochondrosis lesion is a good indicator for surgical debridement. When serial examination demonstrates a static osteochondrosis lesion or even progression to a normal cartilage–subchondral bone surface, surgery may not be indicated.

Osteoarthritis

Osteoarthritis manifests as thinning and erosion of the articular cartilage and progresses to osteophyte formation and periarticular soft tissue changes such as enthesis new bone at the joint capsule attachments. In the horse early cartilage thinning has been difficult to identify ultrasonographically. More advanced stages of the disease may be easier to demonstrate in the high-motion joints

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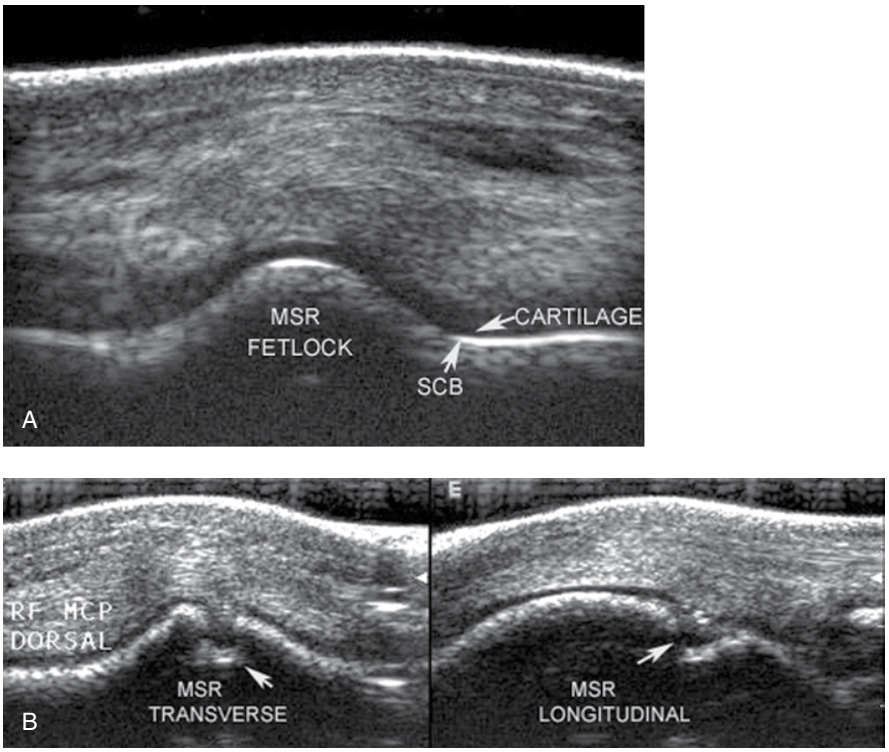


Figure 3.115. Osteochondrosis in the developing animal manifests articular cartilage lesions that are often associated with significant joint effusion, and in many cases there are cartilage or osteochondral fragments either *in situ* or free-floating within the joint. (A) This is a transverse ultrasound image of the dorsal aspect of a normal MCP joint. The anechoic cartilage is seen overlying the hyperechoic subchondral bone surface. Notice the smooth subchondral surface of the normal joint. (B) This transverse and longitudinal ultrasound image demonstrates an irregular subchondral surface of the sagittal ridge of this MCP joint, consistent with osteochondrosis.

particularly when the joint capsule is seen to be in close contact with the subchondral bone surface when pressure is applied with the transducer. The advanced stages of the disease often manifest significant soft tissue changes such as enthesis new bone and periarticular lipping. In the low-motion joints, such as the distal hock joints and the pastern, ultrasonographic evidence of osteoarthritis generally is limited to periarticular changes as access to the cartilage surface is limited. A study in humans showed that ultrasound was useful in identifying early osteoarthritis of the knee.[58] Changes included a decrease in cartilage thickness and blurring of cartilage margins that made cartilage measurements less precise. In general, ratings of clarity and sharpness correlated better with clinical status than absolute thickness of the articular cartilage in that study. Further experience with ultrasonographic examination with higher frequency transducer will hopefully allow similar criteria to be established for horses. Subchondral bone sclerosis is also a feature of osteoarthritis, but cannot be detected with ultrasonography because the sound beam does not penetrate the subchondral bone surface. However, except in foals, the subchondral bone surface should be uniform.

Subchondral Bone Cystic Lesions

Horses with lameness that improves after intra-articular anesthesia of a joint should have a radiographic and ultrasonographic examination performed.[101] Ultrasonography should be utilized to evaluate the intra-articular environment. Flexing the joint as best as possible and placing the probe perpendicular to the subchondral bone surface facilitate evaluation of the weight-bearing area of the joint. The joint most commonly examined for subchondral cyst formation is the medial femoral condyle of the stifle (Figure 3.116). Cartilage lesions with underlying subchondral bone defects are readily identified and their size and position documented with ultrasonography. Subchondral cystic lesions are also readily identified and help characterize the size of the cyst opening and depth of the cyst. Some clinicians have reported using ultrasound to guide the placement of a needle percutaneously into the cyst cavity. Treatment is directed at placing various medications directly in the cystic cavity (corticosteroids, bone marrow, stem cells, etc.).[102] As in other joint injuries, radiographic and ultrasonographic examination is complementary and should be performed together. Identifying a specific injury within these joints may preclude the use intra-articular injection of anti-inflammatories and direct the clinician to explore the joint arthroscopically.

Menisci

The equine stifle has two menisci that are composed of specialized fibrocartilage. A frontal ultrasound scan (lateral to medial or medial to lateral longitudinal orientation) of the femorotibial joints produces the best images of the menisci and collateral ligaments of the stifle. The menisci have a triangular shape and a homogeneous

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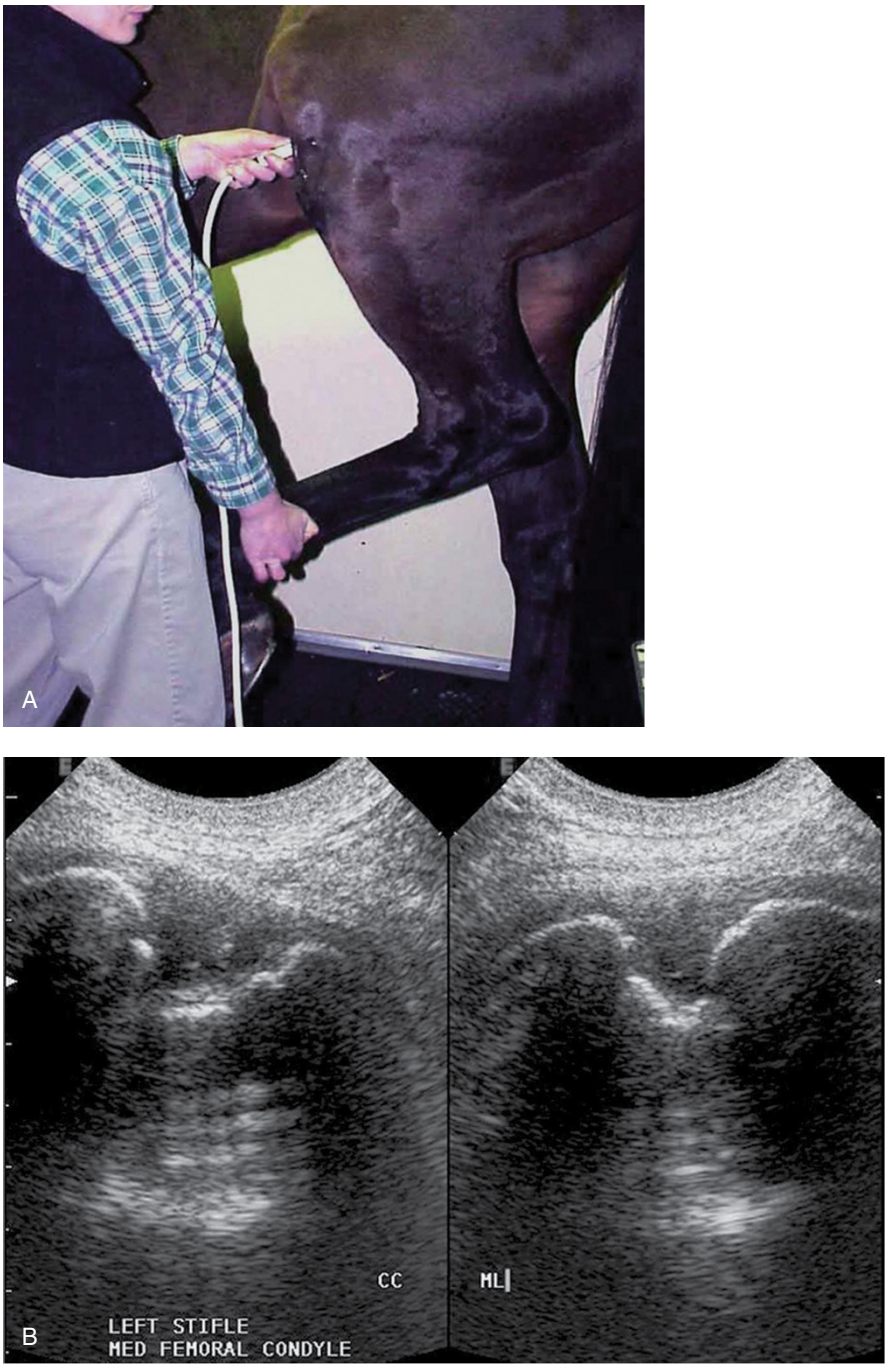


Figure 3.116. (A) The weight-bearing area of the medial condyle is evaluated by flexing the stifle and placing the probe between the medial and middle patellar ligaments just above the tibial crest. (B) The ultrasound image demonstrates a large subchondral defect in the medial femoral condyle of the femur, consistent with a subchondral cystic lesion.

appearance when the transducer is perpendicular to their abaxial border. A complete description of the ultrasonographic examination of the stifle is presented in various texts and should be studied before the examiner attempts to evaluate the stifle.[17,20] Meniscal evaluation is enhanced by fluid accumulation within the joint. A complete assessment of the entire meniscus is essential and should include the cranial and caudal horns. Cranial lateral and cranial medial horns are visible from between the lateral and middle patellar ligaments and between the medial and middle patellar ligaments in the standing horse. The cranial and caudal horns of the meniscus have a similar ultrasonographic appearance when imaged from the cranial and caudal aspects of the joint and with the stifle flexed. Documentation of caudal horn involvement with ultrasonography indicates that exploratory arthroscopy should also involve a caudal approach because damage to the caudal horn of the meniscus often is not appreciated via the normal cranial arthroscopic approach to the femorotibial joint. Damage

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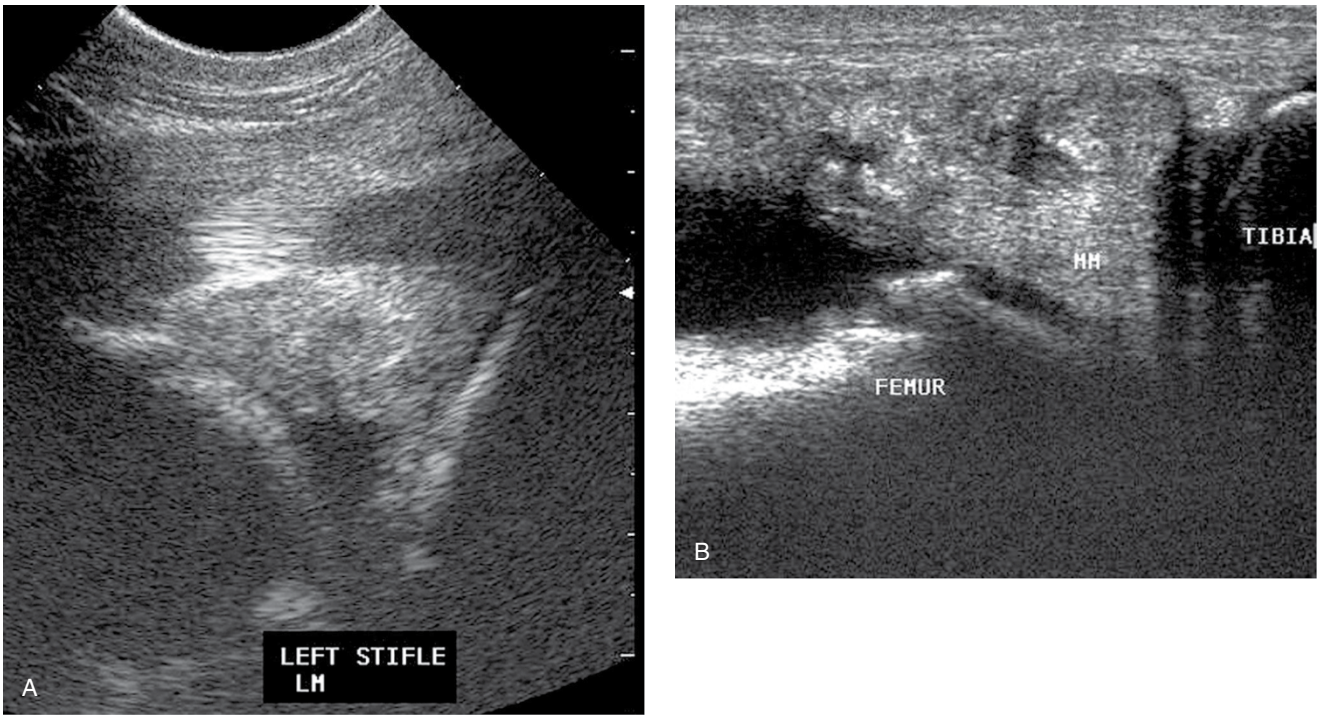


Figure 3.117. Damage to the medial meniscus is manifested ultrasonographically by a change in size, shape, echogenicity, or position relative to the femoral condyles and proximal tibia. (A) This ultrasound image of a damaged meniscus demonstrates an axial injury of the meniscus. (B) This image represents a more severe injury of the meniscus with prolapse of the meniscus.

to the meniscus is manifested ultrasonographically by a change in size, shape, echogenicity, or position relative to the femoral condyles and proximal tibia (Figure 3.117). Hyperechoic areas casting shadows are indicative of calcification and suggest chronic damage. Loss of the normal triangular shape is indicative of tearing. Linear hypoechoic images indicative of tears may be seen crossing horizontally through the medial meniscus (MM) and are best identified with the stifle in slight flexion. Large hypoechoic areas in cross section can be associated with chronic lameness and compatible with edema, fiber disruption, and degenerative processes. Extrusion of part of the meniscus from its normal position can occur if the damage is severe. Collapse of the joint space and joint effusion may also be appreciated when both the collateral ligaments and menisci are involved. When synovial distension is extensive and the synovial membrane is thickened, a hypoechoic space can be identified between the MM and the superficial structures (fascia and medial collateral ligament [MCL]). This gap between the MM and the MCL is abnormal, because these two structures normal are adhered to each other.

Identifying Incomplete Ossification

Ultrasonography can be useful in determining the stage of endochondral ossification in young foals. It is particularly useful for identifying incomplete ossification of the cuboidal bones in the carpus or tarsus of neonates. Unlike radiography, ultrasonography provides the information immediately in a field situation, allowing appropriate treatment or management changes to be instituted without delay.

CONCLUSIONS

Ultrasonography is vastly underutilized in the clinical assessment of joint problems in horses. A complete radiographic study of a joint can demonstrate osseous lesions, but it gives little information about the articular cartilage and soft tissue structures of the joint, which are important sites of pathology in most types of joint disease. These tissues are readily imaged using ultrasonography (within the anatomical limitations of the particular joint being imaged). Arthroscopy allows direct visualization of lesions found on radiography and assessment of the articular cartilage surfaces and other intra-articular structures. However, arthroscopy requires general anesthesia and it is invasive and expensive. Ultrasonography is noninvasive, rapidly performed, widely available, well tolerated by the patient, and inexpensive.

Ultrasonography offers many advantages in the clinical assessment and management of joint problems in horses. Ultrasonography can potentially detect lesions not evident radiographically allowing treatment to be instituted and/or management changes made that would slow or arrest lesion progression and prolong the useful life of the horse. In particular, ultrasonography may identify soft tissue and cartilage defects over radiographically normal bone that may not otherwise be identified only at surgery or postmortem examination. Although the initial ultrasound exam may not always provide a specific diagnosis, repeat ultrasound examination may reveal early changes indicative of a potentially serious joint problem. In humans, ultrasonography is purportedly a more sensitive indicator of early osteoarthritis than is radiography.[58] It allows monitoring of lesion progression or

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resolution over time, which can aid surgical decision-making, direct management, and rehabilitation strategies and increase the understanding of joint disease in horses.

Ultrasonography can enhance the equine practitioner's ability to accurately diagnose and manage joint-related problems in performance horses. However, it is only one tool at the clinician's disposal; clinical acumen and selective use of other imaging modalities are needed to accurately diagnose many types of joint disease. Currently, the gold standard for assessing soft tissue injuries in humans is MRI. However, in human medicine, ultrasonography remains a very useful imaging modality for evaluating the popliteal space, knee, patellar tendon, shoulder (especially the rotator cuff), and neonatal hip. Extensive use of ultrasonography in the examination of equine joints has already demonstrated the sensitivity of this modality in defining articular lesions not apparent radiographically. In time, there will undoubtedly be certain joint conditions in horses that will most readily be identified with ultrasonography.

OTHER INDICATIONS FOR ULTRASONOGRAPHY OF THE MUSCULOSKELETAL SYSTEM

Evaluation of Bone

Bone appears as a bright hyperechoic line with a strong acoustic shadow. This is due to the high acoustic impedance when compared with the soft tissues. The bone surface appears to be of uniform thickness. Ultrasonography has proven useful in the diagnosis of fractures particularly in areas that are not readily accessible to radiographic examination such as the pelvis, femur, scapula, humerus, and spine. Ultrasonography is often utilized in conjunction with nuclear scintigraphy to focus on areas of bone injury not apparent on radiographs. Fractures can be seen as an anechoic to hypoechoic line that is visible in the cortical bone. Often there is displacement (distraction) of the bone edges evident along the cortical margin.

Osteitis and osteomyelitis can also be evaluated with diagnostic ultrasound and appears as a fluid interface at the bone surface.[81] Acute trauma may have hemorrhage at the bone surface that can appear similarly to osteitis and may indicate more detailed radiographs to rule out a fracture. A repeat scan should be performed in 4–5 days to document resolution or resorption of the hemorrhage of the fluid interface. Progression to osteitis is demonstrated by the persistence of fluid, which can vary from hypoechoic to anechoic and may contain hyperechoic echoes consistent with gas in the fluid. Hypoechoic tracts that begin at or just under the skin surface can occasionally be seen tracking to the bone surface. The bone surface may begin to demonstrate a raised area of periosteal new bone production consistent with involucrum formation at the margins of the sequestrum or bone fragment(s). These periosteal changes can be seen earlier with ultrasound than radiographs. Sequestra appear as hyperechoic structures that cast acoustic shadows (Figure 3.118). Sequestra typically remain adjacent to parent bone and are surrounded by hypoechoic to anechoic fluid. Occasionally the sequestra can be seen displaced from the involucrum and lying in the tract leading to the skin surface. Areas with a predisposition to form sequestra include the metacarpal/metatarsal bones, spine of the scapula, and

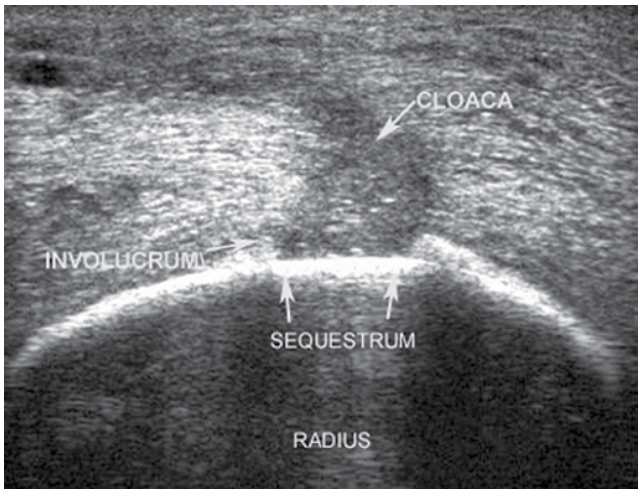


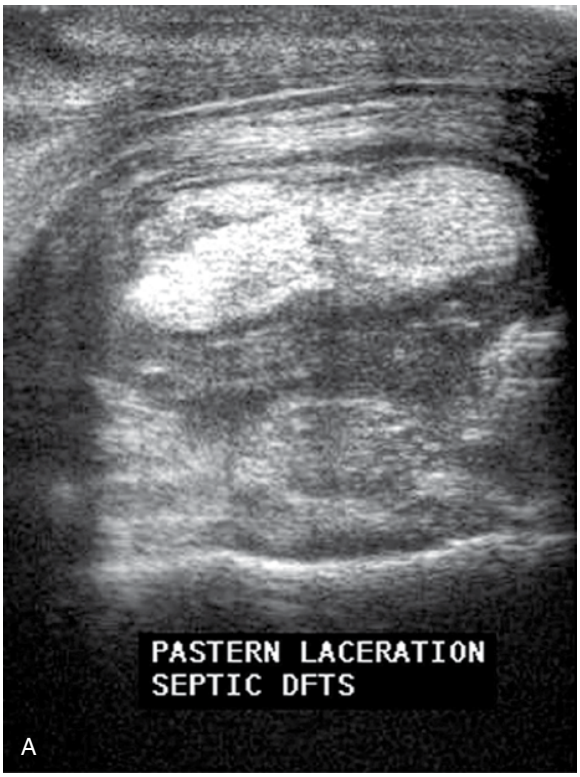
Figure 3.118. This horse had a draining tract of the medial radius. Radiographs were suggestive but not conclusive for a sequestrum, but ultrasonography proved to be diagnostic. There are small gas shadows within the middle of the tract (cloaca) that courses from surface of the radius to the skin. New bone is being deposited at the periphery of the sequestrum. The margins of the sequestrum have raised periosteal new bone production consistent with involucrum formation. Sequestra appear as hyperechoic structures that cast acoustic shadows. These periosteal changes can be seen earlier with ultrasound than radiographs.

medial aspect of the radius, but any area with cortical bone that is close to the skin surface may develop a sequestrum. Ultrasonography is also helpful to assess fractures that have been repaired with internal fixation. In the early postoperative period, the repair will appear much like acute trauma cases with hemorrhage and edema surrounding the implants. However, 5–7 days postoperatively this fluid interface should begin to become more organized unless there is increased motion or infection of the repair. If this fluid interface persists and the animal manifests systemic signs such as pain, heat and swelling at the incision site, fever, and/or lameness, then infection of the implants should be suspected.

Evaluation of Punctures and Lacerations

Ultrasonography has proven particularly helpful to define the extent of soft tissue damage incurred during wounding from a puncture or a laceration. Lacerations over the extensor or flexor tendons in the distal extremities require careful examination of the tendons to document involvement and then determine the extent of damage incurred at wounding (Figure 3.119). Also, because these tendinous structures are frequently associated with sheaths and bursa, it is important to determine if these synovial structures are involved. The wound should have a sterile prep applied to the wound margins and the wound bed flushed with a balanced electrolyte solution to clean dirt and debris from the wound. Sterile lubricant can be applied to the wound bed and a sterile glove or sheath placed over the probe. The probe can then be placed into the wound to exam the structures deep within the wound bed. Air introduced into the wound may block sound transmission and compromise the study, which can be performed on another day after keeping the wound under a bandage. Documentation of tendon

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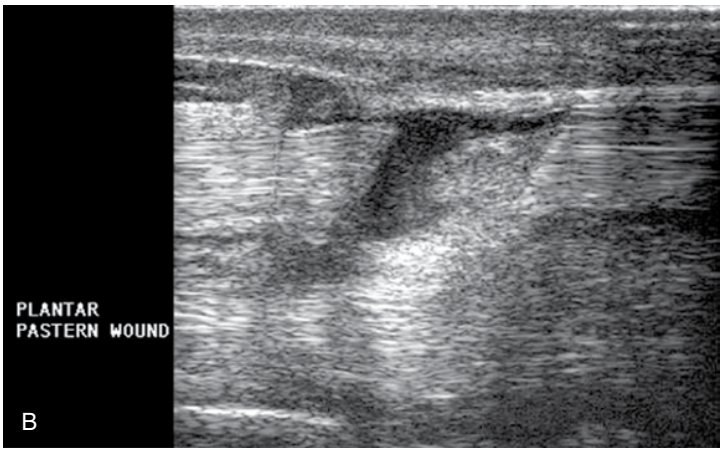


Figure 3.119. Lacerations over the extensor or flexor tendons of the distal extremities require careful examination of the tendons and any associated sheaths/bursae to determine the extent of damage incurred at wounding. (A) Transverse ultrasound image of a DDFT injury in the pastern. There is a proliferative response within the DFTS that suggests an active inflammatory process. Due to the penetrating injury, sepsis is likely. (B) Longitudinal ultrasound image of a laceration that demonstrates a severe DDFT injury. There is complete transaction of the DDFT with retraction of the proximal stump of the tendon.

or sheath involvement significantly changes the management of these types of wounds. Diagnostic ultrasound can be useful to identify synovial distension and assess the character of the synovial fluid. An increase in cellularity and fibrin content in the synovial fluid increases its echogenicity. The presence of gas shadows suggests either an open joint space or the presence of gas-producing organisms in the joint fluid. Treatment must be directed at eliminating any foreign material, reducing bacterial numbers, removing contaminated and devitalized material, and neutralizing and eliminating inflammatory enzymes and other inflammatory products. Puncture wounds involving the tendons and ligaments around the joint can significantly affect the prognosis.

A recent report looked at seven horses that had wounds close to a synovial structure (joint or sheath) and found that it was possible to see hair shafts within the wound and within the synovial lining.[65] These findings support the use of ultrasonography as a valuable diagnostic tool in wounds that may have breached a synovial structure. The presence of hair and/or foreign material in horses with signs of inflammatory or septic synovitis/tenosynovitis often suggests that aggressive measures be taken to eliminate the material.

Evaluation of Muscle

Acute muscle injury occurs due to blunt trauma, violent contraction against resistance, and myositis. Muscle has a heterogeneous appearance with hypoechogenic muscle fibers laced with and surrounded by fascia, connective tissue, and fat. On transverse images normal muscle has a marbled or speckled appearance.[81] Each muscle has a fairly unique appearance that can change between weight-bearing and non-weight-bearing. More severe muscle injuries may have fluid-filled areas with loculation visible suggesting significant muscle fiber tearing and hemorrhage. Minor injuries can be more difficult to define because ultrasonographic changes are less obvious. Minor injuries may simply manifest an enlargement of the muscle belly with very little loss of the normal striated muscle pattern and a decrease in echogenicity. The paired structure on the opposite limb should be used for comparison (Figure 3.120). Ultrasonography can be utilized to monitor the repair process. The hematoma will be slowly resorbed and the area gradually filled in with granulation tissue. The infiltration of fibrocytes and capillaries create a more heterogeneous appearance to the muscle injury over time.

Fibrotic myopathy is a chronic muscle condition that is probably the result of multiple previous acute muscle injuries that healed with exuberant scar. This scar creates a restricted gait where the limb is rapidly slapped to the ground at the end of swing phase (described as goose stepping). Palpable muscle scarring can usually be found within the semimembranosus, semitendinosus, and occasionally the gracilis muscles. The ultrasound appearance of this mass is consistent with excessive fibrosis seen as hyperechogenic areas within the body of the muscle. There may be hyperechogenic

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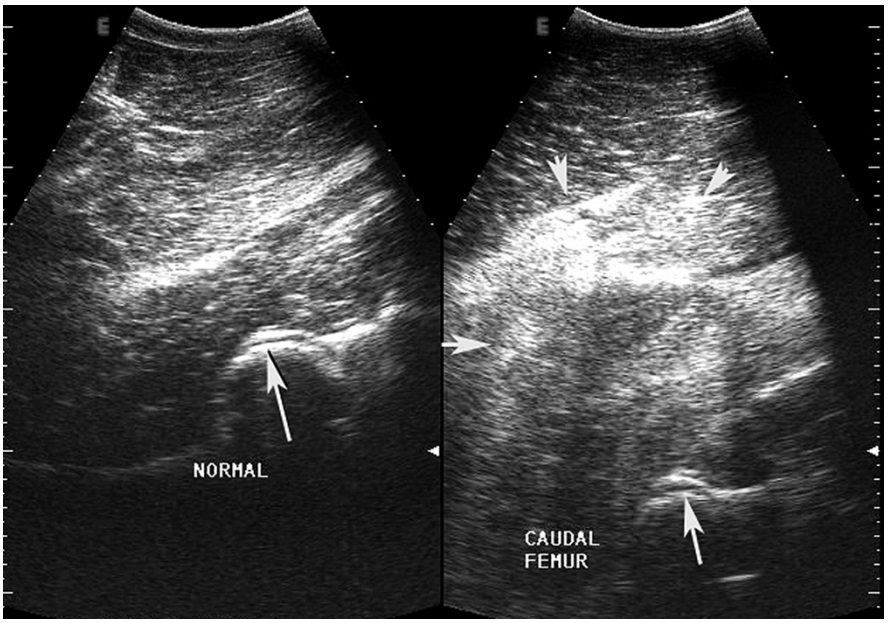


Figure 3.120. This is a longitudinal ultrasound image of the caudal aspect of the distal femur of the stifle. The cartilage covering the caudal condyle is apparent (lower arrows). Proximal is to the left, and distal is to the right. This horse incurred a caudal reciprocal apparatus breakdown, and the gastrocnemius muscle can be seen to have significant damage (smaller arrows surrounding the damaged area).

areas suggestive of mineralization or dystrophic calcification that cast acoustic shadows within or adjacent to the affect tissue.

Evaluation of Foreign Bodies

A number of different materials when introduced into the soft tissues can create a significant foreign body reaction in the horse. The most common foreign bodies include wood, lead (bullets or buckshot), metallic objects (such as wire or fencing materials), glass, plant material, hair, and suture material (Figure 3.121). Wood appears as a linear hyperechoic structure that casts a strong acoustic shadow. The most common wood foreign bodies are associated with fencing materials that splinter after penetrating the skin. It is important to carefully evaluate the wounded area for multiple wood splinters before initiating retrieval as air introduced into the wound either at wounding or during surgery can block ultrasound transmission further limiting the evaluation of tissues deep to it. Bullets and metallic structures can appear to have variable shapes and contours, but like wood these objects can cast strong acoustic shadows. However, plant material and hair appear to have small hyperechoic shadows that may or may not cast acoustic shadows. This hyperechoic material is usually seen within a hypoechoic tract. Metals such as surgical instruments appear similarly and cast strong acoustic shadows that can be utilized to the clinician's advantage when ultrasound-guided retrieval is utilized. Placement of an instrument such as a mosquito forcep around the foreign body can easily be seen until the air/gas is introduced. If retrieval of the foreign body is more complicated, then the area should be mapped out with ultrasonography before retrieval.



Figure 3.121. This horse had a draining tract at the shoulder region. There appears to be a dense foreign body several centimeters deep, consistent with a bullet. The surgical approach to the foreign body was mapped out with ultrasonography intraoperatively.

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NEW DIRECTIONS IN THE USE OF ULTRASOUND

Ultrasound-Guided Injections

Diagnostic ultrasound can enhance the accurate placement of a needle into a specific site. Real-time needle tracking allows the needle to be visualized entering a target area (Figure 3.122). For example, the correct placement of diagnostic analgesia will help improve the accuracy of diagnosing the cause of lameness, and placing medication or regenerative therapy (platelet-rich plasma [PRP], stem cells, and interleukin receptor antagonist protein [IRAP]) directly into a lesion will enhance the successful treatment of that injury. In veterinary medicine there are several reports evaluating the use of diagnostic ultrasound to inject the navicular bursa, cervical articular facets, scapulohumeral joint, coxofemoral joint, and the sacroiliac joint.[5,10,14,16,60,93] Competency in ultrasound-guided injection technique begins with a comprehensive knowledge of the anatomy of the area both grossly and as seen with ultrasound. A reasonable amount of skill is required to handle a transducer focused on the area of interest while driving a needle within the ultrasound beam. Depth of the structure to be injected should determine the angle the needle must take to hit the structure. To increase the degree of difficulty, ultrasound-guided injections are frequently done while maintaining sterile technique (the probe is covered with a sterile ultrasound covers). As a means to improve needle placement and minimize damage to vessels in the area, a Doppler examination (color or power) can help determine a path for the needle free of vessels that may not be obvious on gray scale. As will be discussed in a later section on Doppler examination of soft tissue injury, power Doppler (PD) can provide some insight of where within a structure an injection should be directed to have the most advantageous effect.



Figure 3.122. The image demonstrates the use of ultrasound to guide the placement of a probe (in this picture a probe that utilizes ultrasound energy to remove damaged tissue), instrument, or more routinely a needle into a specific area of tendon/ligament damage.

Sterile technique requires a large area of skin prep with chlorhexidine detergent and alcohol rinse. It is advisable to disinfect the probe and cable with a disinfectant before placement of a sterile probe cover or surgical glove. The examiner should view the area between the probe and the structure to be injected. Previewing the area and knowing the pertinent local anatomy are critical to avoid penetration of key structures (vessels and nerves). It is best to mapped out the approach that will be taken to drive the needle to the target. A decision should be made about utilizing short axis or long axis visualization of the needle. The examiner should use the nondominant hand to manipulate the probe and scan the target keeping 2–3 fingers of the probe hand in contact with the patient's skin. Most often the needle should cross the longitudinal plane of the sound beam, and the examiner then must make short axis slides of the probe to keep the needle in view. Placing the needle in the plane of the sound beam (long axis) is preferred during most procedures because the needle tip and shaft are visualized throughout the entire procedure. Some ultrasound probes have a needle guide that can be attached directly to the transducer for accurate placement of the needle. Alternatively, and probably more commonly, the needle may be placed freehand. Either way, the needle should be visualized throughout the procedure. The shortest pathway to the target should be selected avoiding regional neurovascular structures. Deeper injections require more skill as tracking the needle can be more challenging and requires better technique. Some alternative techniques to improve ultrasound-guided injections include using beam steer, scoring the needle with scalpel, using reflective facet tip needles, and placing the bevel of the needle up. Another option though not preferable is to use a blinded technique whereby a needle is placed into the area of the synovial structures. If no fluid is retrieved, then the needle position is evaluated with ultrasound to confirm the correct placement. If it appears that the needle is position correctly, then fluid can be injected to confirm the correct position within the joint or any indication that repositioning the needle is necessary.

MRI/Ultrasound Fusion: Navigational Ultrasound

MRI/ultrasound fusion imaging or navigational ultrasound imaging is a novel imaging technique developed to combine cross-sectional imaging (CT or MRI) with real-time ultrasound. This technique has been developed and utilized in the human medicine to sample abnormal masses within the liver, kidney, and prostate. The technique was developed to guide the sampling and radiofrequency ablation of masses that were invisible

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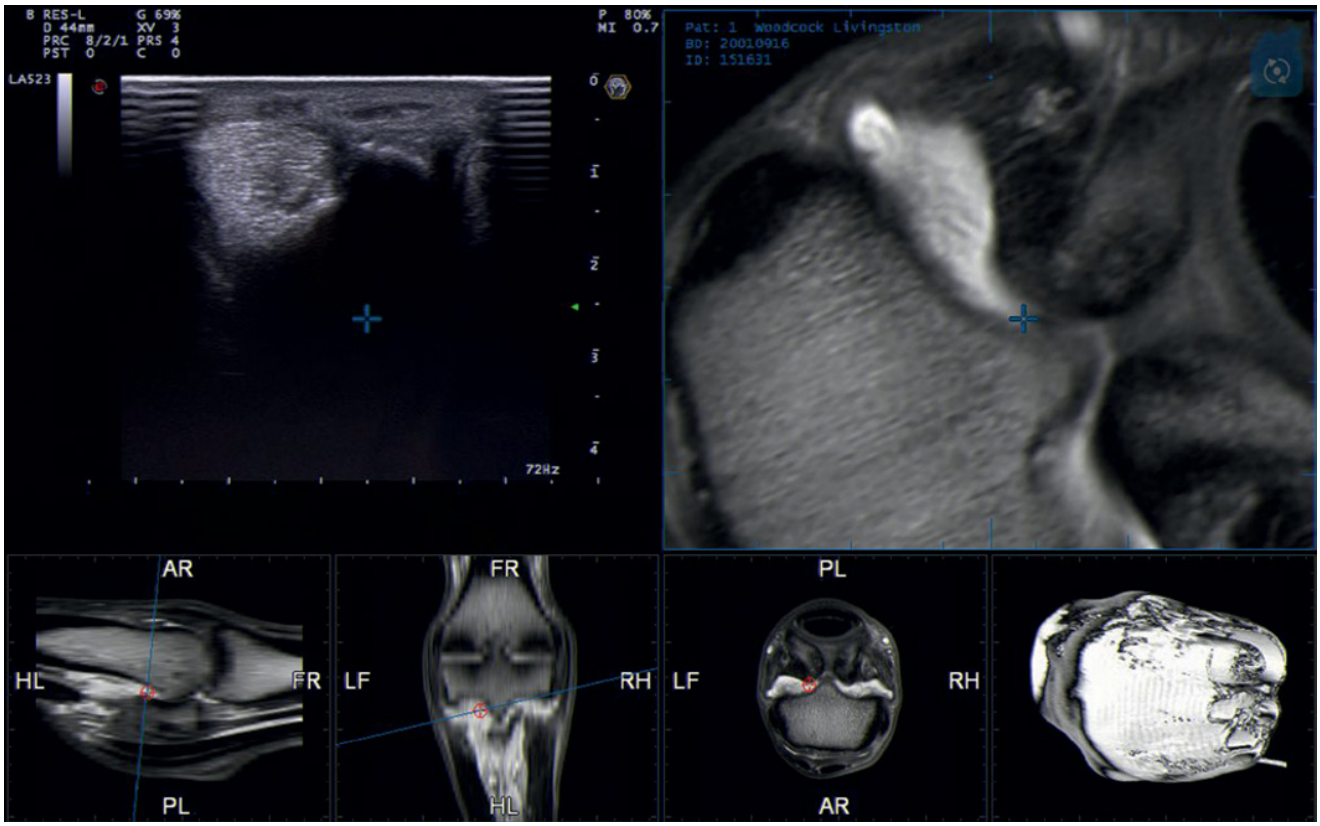


Figure 3.123. Navigational ultrasound fuses the real-time ultrasound capability with the MR sequence of an area to more accurately demonstrate a lesion that is difficult to see with ultrasound.

to ultrasound. It has been shown not only to improve sampling accuracy but also to decrease procedure risk and time.[1,45,47,87,106]

This technology has recently been utilized in horses that have had an MRI (high-field) examination of a region.[54] Candidates for this technology were animals with MRI lesions that were difficult to detect with ultrasound (Figure 3.123). Navigational ultrasound was utilized to combine the accuracy of MRI for lesion detection with use of real-time ultrasound examination to guide interventional procedures into these lesions. Navigational ultrasound has proven to be quite successful at guiding the accurate placement of a needle or instrument (tendon splitting knife, radiofrequency probe, Tenex$_R$ probe) into a lesion. The needle can be placed into sites of active inflammation within a lesion (increased T2 hyperintensities on MRI). The needle placement and injection of regenerative therapies (PRP, IRAP, and fat-derived or mesenchymal stem cells) into a lesion can be visualized. The volume of the injectate can be assessed and leakage minimized. In some other cases the fusion directed the placement of an instrument in the surgical approach to a lesion(s).

To perform a navigational ultrasound, the transverse PD sequence of a study acquired by a Siemens Symphony 1.5 T was imported into a Biosound MyLab Twice in which the navigational software has been installed. A magnetic field is established around the extremity with the placement of a GPS device at the level of the lesion. A magnetic sensor is positioned on the ultrasound transducer and allows the transducer to be tracked within the magnetic field of the GPS system. The MRI sequence is scrolled to visualize a predetermined anatomic location (usually on the immovable bony column) close to the MRI target lesion. This same anatomic site is then identified with the ultrasound on transverse view. The same procedure is performed on longitudinal view. Co-registration of this anatomic site on both transverse and longitudinal planes now blends the ultrasound image and the corresponding image on MRI in whatever orientation the transducer moves within then magnetic field. Once this has been completed, the MRI study tracks along with the ultrasound transducer as it is moved within the magnetic field of the GPS demonstrating the real-time ultrasound image superimposed over the MRI sequence. The software within the ultrasound allows the MRI and ultrasound images to be superimposed and blended, which makes this technique very useful to allow the direct comparison of the pathology seen in these structures with these different imaging technologies.[45,54,106] Navigational ultrasound has proven to be quite helpful to treat those lesions that ultrasound examination doesn't accurately portray the lesion(s) as seen on MRI.

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Elastography to Evaluate Soft Tissue Injuries

Elastography is a new ultrasonographic technique that allows an examiner to evaluate the mechanical properties of soft tissues.[8,51,67] This technique estimates tissue strain, where strain is defined as the fractional change in length of a tissue when an external force is applied. Elastography evaluates tissue motion as compression is applied to the tissue using manual compression with the ultrasound transducer. The tissue displacement causes a measureable displacement of the ultrasound waves. Soft, deformable structures, such as damaged or inflamed tissues, cause greater displacement of sound waves than hard, rigid structures. Elastography can therefore provide information about the behavior of internal soft tissue structures when compressed. Since pathologic tissues often exhibit altered mechanical properties, elastography allows the differentiation between normal and diseased tissues due to the differences in these elastic properties. With elastography, compression (in a transverse or longitudinal plane) is applied with the ultrasound probe, and tissue displacement is measured by comparing the echo signals before and after compression. This is in contrast to conventional grayscale ultrasonography that only evaluates tissue morphology and the differences between acoustic impedance of adjacent structures.

Elastography was initially used in humans to help differentiate benign from malignant nodules in the breast, prostate, thyroid, liver, and lymph tissue and for evaluating hepatic fibrosis.[67] Recently, elastography was used to evaluate this technology in musculoskeletal injuries in humans primarily the Achilles tendon.[29,30] The evaluation of acute injuries with elastography can reveal significantly increased softness in areas of hematoma and fiber disruption. Serial elastographic examinations can detect the tissue becoming progressively stiffer with healing.

An initial study was performed to determine the feasibility, reproducibility, and repeatability of elastography and to establish the normal elastographic appearance of the tendons of the equine metacarpus.[52] A group of sound horses without evidence of musculoskeletal pathology were evaluated with standard grayscale ultrasound, and elastographic evaluations were performed using an ultrasound scanner (MyLab 70 ultrasound system Biosound Esaote Inc. Indianapolis, IN) with a 6–18-MHz linear probe using a previously described technique. Elastographic images of the SDF and DDF tendons and the branches of the SL were evaluated quantitatively and qualitatively.

An extension of that study in normal horse was performed in horses with clinical conditions found with grayscale ultrasound. This study attempted to characterize elastographic findings of clinical cases with soft tissue injuries of the distal limb and to evaluate the differences in the elastographic appearance of acute versus chronic injuries.[53] Horses were evaluated throughout their rehabilitation and assessed for changes in the ultrasonographic and elastographic appearance over time. These findings were correlated with clinical healing. This study found that compression elastography corresponded well with clinical evaluation using grayscale ultrasound (Figure 3.124). Acute lesions were significantly softer than subacute/chronic lesions when

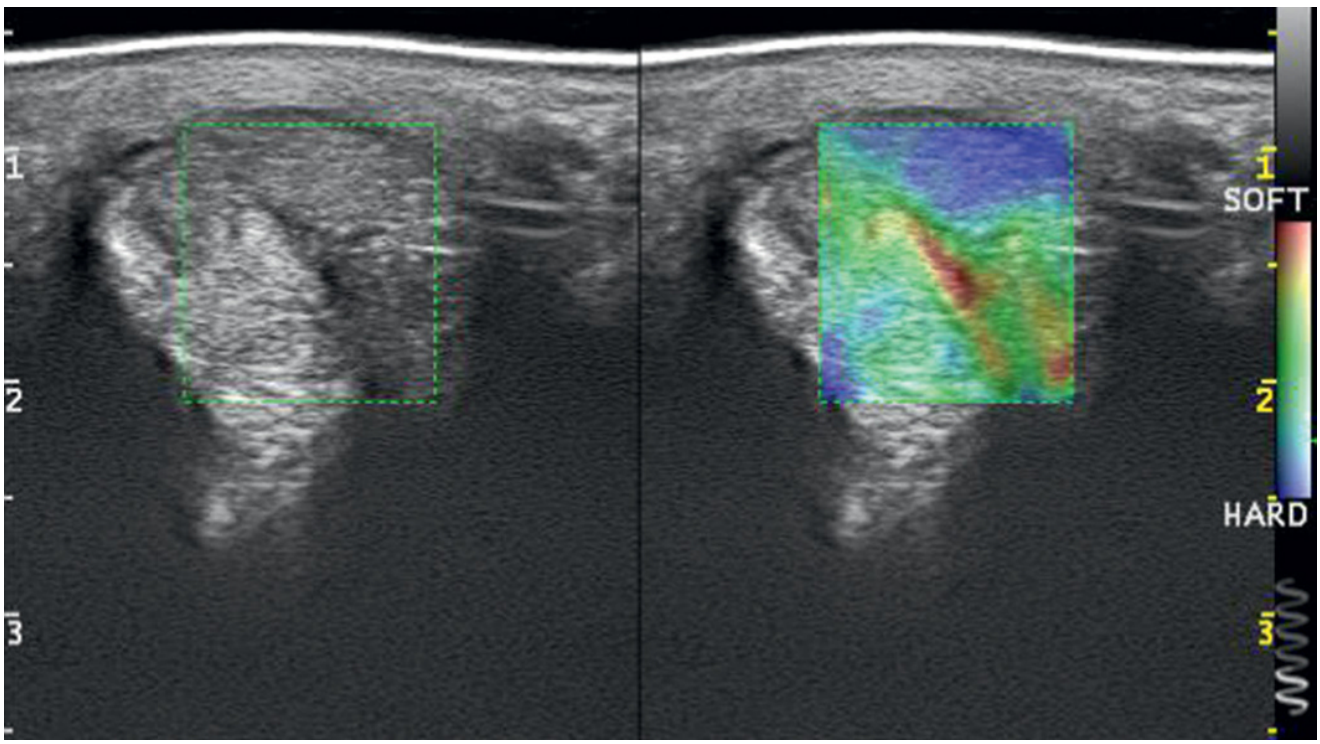


Figure 3.124. Elastography, a new software program available in some advanced ultrasound systems that can measure the strain within that structure and portray the softness of a structure displayed by a difference with color codes.

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evaluated with elastography. These injuries showed a progressive increase in stiffness of acute lesions over time. Most lesions of the suspensory ligament branches (SLBs) nearest to the insertion on the proximal sesamoid bone were detectable with elastography in longitudinal orientation with the limb weight-bearing. Lesions of the SLBs nearest to the insertion were only reliably detectable in transverse orientation when the limb was off-weighted. It should be noted that lesions of the PSL, which caused detectable changes with elastography, were moderate in size and severity, while small lesions of the PSL near the origin were not detectable using elastography or with grayscale ultrasound.

The main limitations of elastography are related to the operator-dependent elastographic images that are obtained. Manual compression and tissue displacement are difficult to standardize. This study found elastography to be a valuable and complementary imaging modality for soft tissue lesions of the equine distal limb. These findings would suggest that incorporating elastography into routine US examinations may be of benefit for characterizing the age of the injury or the stage of healing. Elastography may also prove to be valuable for guiding rehabilitation by monitoring the stiffness of lesions over time.

Color Doppler Ultrasound to Evaluate Tendon and Ligament Injury

Currently, grayscale ultrasonography is the most popular means of diagnosing equine tendon/ligament injuries and is the most practical soft tissue imaging tool available to the equine practitioner.[80] Acute tendon injury manifests ultrasonographically as enlargement often with hypoechogenicity of the tendon/ligament architecture on cross section and the appearance of a striated or fibrillar pattern in the longitudinal images. These changes are due to disruption of tendon matrix, with intratendinous hemorrhage and edema. In more severe injuries there may be changes in the overall shape, distortion of the margins/outline of the structure, and a change in the normal anatomical position in relation to other structures in that area. Typical for any wound, a tendon or ligament injury precipitates an increase in blood flow during the acute inflammatory phase. Depending on the severity of the injury, the inflammatory phase is usually short lived and blends into the subacute fibroblastic phase of tendon healing. Within a few days of injury, there is a pronounced angiogenic response with an increase in the synthesis of collagen during the fibroproliferative tissue. The fibroblastic phase of tendon healing merges with the remodeling phase with a gradual transformation of collagen type III to type I as the scar tissue matures. This corresponds to a gradual reduction in blood flow.[33,43,91] Currently serial grayscale ultrasound imaging is the tool of choice at providing important morphologic information especially during this remodeling phase and has been found to be quite useful to assess and monitor healing of soft tissue injuries. However, few studies have evaluated blood flow in equine tendon and ligament injury.[48,49,59,94,105]

Soft tissue injuries such as acute tendon and ligament lesions rely on vascularization for cell recruitment and to provide humoral mediators, growth factors, and nutrients. Blood flow begins to increase in the early inflammatory stage, peaks in the fibroblastic phase, and gradually subsides during the remodeling phase. Tendons and ligaments receive blood supply from three potential sources. The primary supply is intratendinous coming predominantly from the musculotendinous junction and from its osseous insertion. Within a sheath, tendons have blood vessels entering the tendon via mesotenonous attachments. Outside of the sheath blood vessels can enter the tendon from the paratenon. The relative contribution of the blood supply from these different sources depends on the specific structure and the location within that structure. Some areas within tendinous structures are thought to be at risk of injury due to a reduction in blood flow in specific areas of the tendon (i.e. SDFT in mid-metacarpus DDFT in the fetlock region).[94] For the metacarpal region of the SDFT, the intratendinous supply is considered the most important source of blood supply as necrosis was only achieved by ligation of the blood vessels within the tendon whereas stripping the paratenon had no effect.[48] The blood flow in the SDFT has been recorded between 1 and 2 mL/min/100 g, which is similar to the blood flow of resting skeletal muscle and has been shown to increase two-fold with exercise.[94,105] Injury to the SDF tendon was associated with an even greater increase in blood flow (>300%). A recent study described the microvascular anatomy of the SL of the equine forelimb and found an abundant intraligamentous microvascular supply throughout its entire length. The absence of an obvious hypovascular area suggests that regional variations in healing rates of the SL are probably not associated with the microvasculature anatomy.[53] Alterations in vasculature are emerging as important diagnostic and prognostic findings in tendon injuries in both equine and humans (Figure 3.125). Neovascularization plays an important role in tendon healing with proper blood supply required for adequate healing and the quality of repair related to the amount of neovascularization as seen in Achilles tendons in humans.[63]

In humans color Doppler (CD) is widely used in cardiovascular examinations to evaluate changes in blood flow. It is utilized to visualize both the velocity and the direction of the blood flow that has been proven to be quite helpful in the diagnosis of venous thrombosis, atherosclerotic changes in the carotids, and arterial aneurysms. High-definition CD can allow the examination of very small vessels in the near field, making it possible to visualize vessels with CD that are not visible with grayscale US. One equine study utilized CD to assess vascular flow after surgically created SDF tendinitis were treated with PRP and found increased blood flow out to 23 weeks in the treated limbs.[7] The use of CD in the evaluation of horses with tendon/ligament injury can prove to be problematic due to the creation of artifacts that occur due to probe position. In addition, blood flow in the tendon/ligaments of horses can be different in weight-bearing and non-weight-bearing positions. Consistent use of CD in equine injuries can hopefully provide information to help stage an injury (acute, chronic, and acute on chronic) and to assess quality of repair.

Power Doppler (PD) is an even more sensitive Doppler method to study the flow of red blood cells

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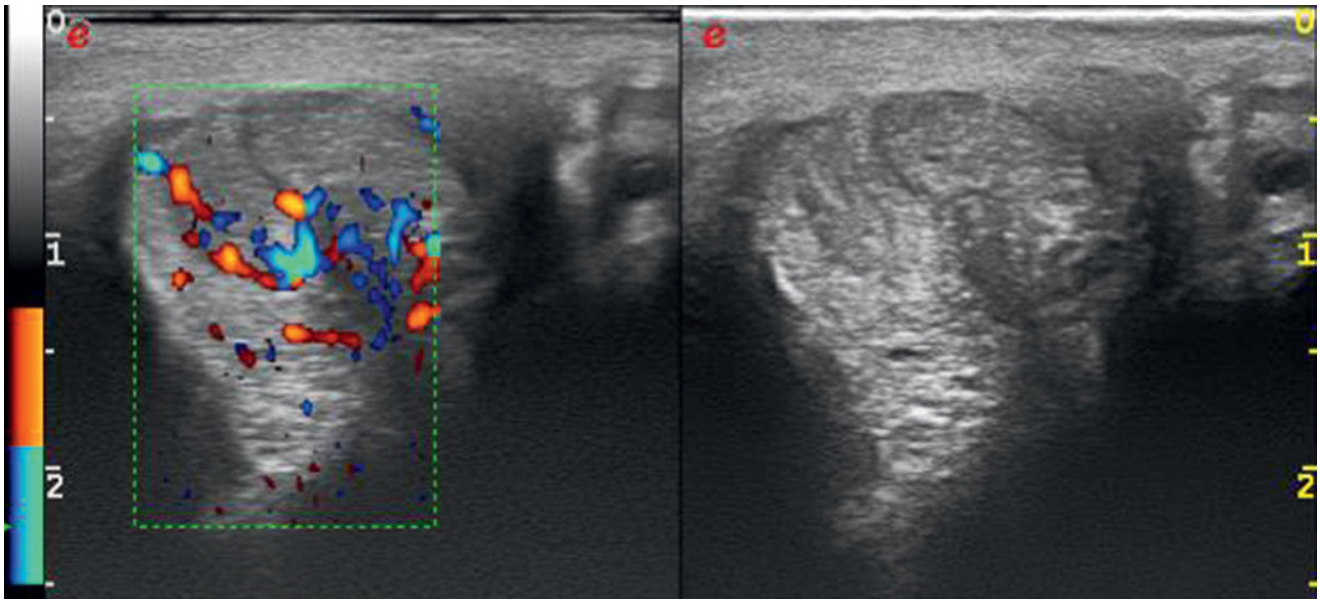


Figure 3.125. Blood flow of a tendon or ligamentous structure can be evaluated by the use of color flow and/or power Doppler. The demonstration of blood flow in a structure can be utilized to help assess the age of a lesion (acute injuries have a more active increase in blood flow than chronic injuries) and can be useful to guide rehabilitation of soft tissue injuries (reduction in blood flow with healing and remodeling).

within the intravascular spaces. PD sonography is a newer technique that displays the strength of the Doppler signal in color, rather than speed or direction information. It has three times the sensitivity of conventional color Doppler for detection of flow and is particularly useful for the evaluation of regional perfusions of small vessels and those with low-velocity flow. PD (unlike CD) is independent of velocity and direction of flow, so there is no possibility of signal aliasing. It is also independent of angle and allows detection of smaller velocities than CD. PD can consistently demonstrate blood flow in smaller vessels (hyperperfusion) that is associated with musculoskeletal inflammatory disease that can provide a useful adjunct to grayscale sonographic examination. The use of PD can increase the specificity of an US examination by helping to differentiate vascular tissue from other types of tissue debris (blood clot, fibrin, and effusion). Increased perfusion is usually evident in the vicinity of tendon injury and within the area of suspected tendinitis.

PD examinations are performed by utilizing a high-frequency linear probe (6–18 MHz) with the Doppler frequency set at 6.3 MHz and a pulse repetition frequency (PRF) maintained at 500 Hz. A region of interest is selected (often a rectangle that centers on the soft tissue structure(s) being evaluated). Most often a standoff pad is not utilized, and care must be taken not to press too firmly that can collapse the vessels. Color gain should be adjusted to a level just below that at which all color noise disappears from the region of interest. Horses are evaluated while standing on the limb, and this is compared with when the horse is not weight-bearing. The degree of flexion of the distal limb may influence blood flow in some instances. It should be recognized that blood flow is increased after exercise and may influence the PD evaluation.

Normal tendons/ligaments exhibit little blood flow in the smaller vessels. Pathologic flow is considered to occur when there is a significant (subjectively) overall increase in vessel number within the affected tendon/ligament and also within the paratendinous/paraligamentous structures. Flow patterns can range from an increased number of discrete, visible small vessels to a soft tissue "blush" appearance presumably due to an increased density of very small vessels (microvascular flow). In addition to cases of tendon injuries within synovial sheaths or bursae, there can be an increase in vessel number and blood flow in the wall of the synovial structure that is considered pathologic. When the US examination documents an alteration of blood flow, it should be compared with the asymptomatic contralateral limb.

Serial grayscale ultrasound in combination with PD examinations is proving to be quite valuable in diagnosing acute and chronic injuries and to guide rehabilitation during the progressive loading of the injured structure(s). Potential limiting factors in the use of PD include the need for some form of standardization of the examination technique and technical parameters utilized in performing a PD assessment as this examination can be significantly affected by operator experience and training, as well as by the quality of the US machine and image processing.

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NUCLEAR MEDICINE/SCINTIGRAPHY

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Nuclear medicine techniques image the blood flow to bone as well as the function or the physiological activity of bone with minimal anatomic detail compared with ultrasound, computerized tomography (CT), and magnetic resonance imaging (MRI) produce images that reveal anatomic detail. Nuclear medicine imaging is one tool that may augment but does not replace the basic lameness examination.[10,58,61,65,74,80,81,95,99] This chapter discusses the principles of, techniques of, indications for, and interpretations of nuclear medicine imaging in the evaluation of the musculoskeletal system of horses.

PRINCIPLES OF NUCLEAR MEDICINE

Nuclear medicine imaging, also known as scintigraphy, is based on the functional distribution of a radiotracer also known as radiopharmaceutical in the body. The radiotracer is made of a radionuclide, most commonly Technetium-99m (^{99m}Tc), and is labeled to a pharmaceutical, which determines the target tissue of the radiopharmaceutical in the body. Technetium-99m decays by emitting a 140-kEv γ-ray. A γ-ray is identical to an X-ray, except that it originates from the nucleus of an unstable atom (^{99m}Tc in this case) as the atom strives toward a more stable state. Nuclear medicine imaging can also be described as an emission imaging technique because the image is made by γ-rays that are emitted by the ^{99m}Tc inside the horse. Radiography is considered a transmission imaging technique because the X-rays that produce the image are transmitted through the patient.

The pharmaceutical part of the radiopharmaceutical determines the distribution of the tracer radionuclide in the body. There are various molecules or cells that can be labeled. Red blood cells can be labeled for the evaluation of the circulating blood compartment, most commonly to study cardiac function. Intravenous administration of ^{99m}Tc-pertechnetate ($^{99m}Tc0_4$) or ^{99m}Tc-labeled red blood cells (^{99m}TcRBCs) is scintigraphic techniques looking at the perfusion (blood flow) of soft tissue structures such as the joints of the distal limbs. White blood cells can be selectively labeled with ^{99m}Tc-hexamethylpropyleneamine oxime (HMPAO) to look for areas of active inflammation/infection.[9,51,63] ^{99m}Tc-labeled biotin (^{99m}TcEB1) has also been used to detect soft tissue inflammation in horses.[46] Renal function can be studied using ^{99m}Tc-diethylenetriamine pentaacetate (DTPA), and quantitative hepatobiliary studies can be performed with ^{99m}Tc-disofenin. Functional lung ventilation studies have been described using aerosolized ^{99m}Tc-DTPA[58] and lung perfusion with ^{99m}Tc macroaggregates of albumin (MAA).[8] Although each of these techniques uses ^{99m}Tc, the distribution of the radiotracer varies, based on the biokinetics of the pharmaceutical or cell to which the ^{99m}Tc has been labeled.

The most common use of nuclear scintigraphic exams in the equine is orthopedic related. Bone scans are conducted using radiolabeled polyphosphonates, which have a high affinity for the Ca-hydroxyapatite molecules in bone. Images made at 2–4 hours' postinjection are a representation of the uptake pattern in the bones. A very predictable uptake pattern is seen in normal animals, and abnormal radiotracer is seen with increased or decreased blood flow or increased osteoblastic activity. Either ^{99m}Tc-oxidronate (HDP) or ^{99m}Tc-methylene diphosphanate (MDP) is administered intravenously at a dose of about 0.35-mCi/kg or 12.95-MBq/kg body weight. ^{99m}Tc-HDP has the advantage of faster soft tissue clearance, thus allowing image acquisition to begin sooner after injection.[4] A second advantage of ^{99m}Tc-HDP is improved visualization of bones surrounded by large amounts of muscle, e.g. spine, pelvis, and hips. An average 450-kg horse receives about 160 mCi (5.92 GBq) of the radiolabel. The dose rate can be adjusted for age, i.e. increased by about 10% in older patients and decreased by about 10% in juveniles because of the difference in metabolic activity of bone tissue. Approximately 50% of the injected radiolabel is excreted in the urine, which results in the effective $T_{1/2}$ being shorter than the natural $T_{1/2}$.[70]

A nuclear medicine evaluation of the musculoskeletal system may consist of three phases. Phase 1, known as the blood flow or vascular phase, represents the radiotracer in the blood vessels before diffusion into the extracellular fluid. It lasts for 1 or 2 minutes after injection. The body region to be evaluated must be positioned in front of the γ camera at the time of injection, and dynamic rapid frame acquisition is made as the radiolabel perfuses the vasculature. Multiple images are acquired over the first few minutes while the radiolabel is within the vascular system, before diffusion into the extravascular space occurs. The vascular phase is used to compare the blood flow, especially to the distal limbs, e.g. in cases of degloving injuries, but it can also be used to document perfusion deficits in different anatomical regions.

Phase 2, known as the pool or soft tissue phase, represents the radiopharmaceutical distribution in the extracellular fluid and is visualized from 3 to approximately 10 minutes postinjection. This phase is used to evaluate blood flow to soft tissues. An increased signal is observed with hyperemia due to edema, inflammation, etc. Increased radioactivity during the pool or soft tissue phase is best used in the distal limbs and has been associated with navicular bone disease, inflamed joints, tendinopathy, and desmopathy.

Early intense bone uptake of the radiopharmaceutical (^{99m}Tc-HDP or ^{99m}Tc-MDP) can sometimes be seen as soon as 5 minutes after injection, especially in cases of intense delayed phase bone uptake, e.g. fractures or infectious processes. This can sometimes make the evaluation of soft tissues challenging at best, if not impossible. A scintigraphic technique for looking at soft tissues only, without any possibility of bone uptake, is to use $^{99m}Tc\text{-}O_4$ (pertechnetate, unlabeled to a pharmaceutical)

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administered intravenously and images made as soon as radiopharmaceutical equilibrium in the extracellular space is achieved. A similar dose to that of the bone-scanning agents is recommended. ^{99m}Tc-RBC is another alternative for evaluating blood perfusion to soft tissues without the risk of bone uptake overlap.[83]

Phase 3, known as the delayed or bone phase, occurs several hours later when approximately 50% of the injected radiotracer has attached to the bone. The remainder of the tracer is excreted by the kidneys in the first 1 or 2 urine voids' postinjection. The uptake pattern of normal bone is quite predictable and is described later in this chapter. The diaphysis of long bones has the least uptake, and greatest uptake of the tracer occurs in the juxtaphyseal and subchondral bone in normal subjects. Increased radiotracer by or near the joints during the delayed (bone) phase has been related to osteoarthritis (OA), various enthesopathies, periarticular bone sclerosis, septic arthritis, etc. These changes from the normal radiotracer pattern are discussed later in this chapter.

RADIATION SAFETY AND PROTECTION

ALARA means that the radiation dose to which one is exposed will be kept "As Low As Reasonably Achievable." These procedures and protocols are intended to protect the clinician, others, and the environment from unnecessary risks due to radiation exposures.

Film badges (personal dosimeters) should be worn to monitor the total cumulative radiation dose. Finger (ring) thermoluminescent dosimetry (TLD) film badges should be worn by personnel preparing and injecting the pharmaceutical.

Eating, drinking, or smoking while handling a radioactive patient or radioactive materials is not permitted.[98] Lead or tungsten syringe shields are designed to help reduce the radiation dose to the fingers.

The nuclear medicine clinician should be conscious of two basic methods of exposure to ionizing radiation: internal contamination and external radiation. Care must be taken to avoid internal contamination through the accidental ingestion of the radionuclide. Latex gloves should always be worn when working with radiopharmaceuticals or radioactive patients.

External radiation occurs when one is in the immediate vicinity of the horse, for example, when scanning. The three basic rules of radiation safety are "time, distance, and shielding." Therefore, the time spent with the horse should be only that necessary for performing the study. Doubling the distance between the horse and the personnel reduces the radiation exposure by one fourth. Similarly, the exposure is quadrupled by halving the distance from the horse. Standing 1 m away from the horse significantly reduces the radiation exposure to personnel. For example, the exposure rate at the surface of a horse of 6.6 mrem/h decreases to 1.3 mrem/h at 1 m. Wearing a lead apron also reduces overall exposure while working with a radioactive patient by filtering lower emitted energies.

After the horse has been released, the stall is closed off for an additional 24 hours before it is cleaned. This allows four more half-lives of natural decay of the ^{99m}Tc that was excreted via the urine, reducing the exposure to the personnel even further (ALARA). Radiation safety rules may vary from state to state; therefore, the practitioner should consult the local radiation safety officer and comply with the specific state regulations.

IMAGING EQUIPMENT

The gamma camera contains a collimator made of small holes in a lead plate that allows only perpendicular γ-rays through. This reduces scatter, thereby improving image resolution. The γ-rays interact with a fluorescent crystal (a thallium-activated sodium iodide crystal is commonly used), changing the γ-energy to light photons. The light photons interact with a photocathode, generating electrons that are amplified by an array of photomultiplier tubes. The *x*–*y* coordinates of the electrons are then recorded, and the image is reconstructed. The resultant image represents the geographic distribution of the radiotracer in the horse. Images can be acquired in several matrices (64×64, 128×128 and 256×256) (Figure 3.126). A 256×256 matrix to optimize image resolution.

Various techniques have been devised to suspend the gamma camera such as a stationary actuated lift or track-and-column mounted detectors (Figure 3.127).[69] The tract system can moved around the horses, whereas in the stationary system, the horse is moved around the camera.

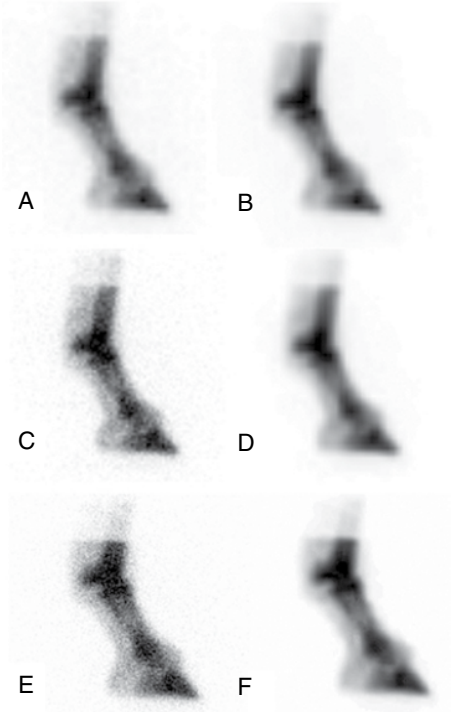


Figure 3.126. Image of right front foot acquired using several matrices. (A) 64×64. (B) 64×64 with statistical and heuristic image noise extraction (SHINE) processing, a technique designed to reduce the Poisson noise contained within the image, preserving resolution and contrast. (C) 128×128. (D) 128×128 (SHINE). (E) 256×256. (F) 256×256 (SHINE).

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Figure 3.127. A gamma camera mounted on a lift system with a yoke and track system that can be used to move the camera in different directions anywhere around the horse.

The gamma camera computer acquires the data and reconstructs the images and sends the digitized images to the processing computer. That computer is usually dedicated to the nuclear medicine facility and is used to perform post-processing of the images. Post-process may include segmentation of certain anatomic regions, addition of color palates, application of image transformation to improve signal in remove noise, and application of regions of interest. The standard image format (.dcm or .xds) is then sent to an archival system to review and store.

The equipment needed for a nuclear medicine imaging service consists of relatively high technology electronic instrumentation and must be maintained appropriately under controlled temperature to ensure that optimal diagnostic images are acquired. Although the maintenance of the equipment is beyond the scope of this text, it should be noted that image quality and resolution can be affected by, among others, one or more of the following factors:

1. Insufficient counts, e.g. too short acquisition time, too low ^{99m}Tc dose, extravascular injection of radiopharmaceutical, inadequately peaked camera
2. Incorrect collimator
3. Inadequate correction floods
4. Motion of patient, camera, or both
5. Excessive distance between gamma camera and patient

Table 3.1. Suggested minimum image acquisition counts.

Body region	Number of counts (×1,000)
Foot	100–150
Carpus	100–150
Elbow	150–200
Shoulder	200–300
Tarsus	150–200
Stifle	150–200
Sacroiliac area	200–300
Spine	200–300
Soft tissue (pool) phase image	75–100
Motion correction acquisition	2 seconds/frame for 40–50 frames

Image acquisition is determined either by the number of counts or the acquisition time. The greater the number of counts/image, the better the image quality, excluding motion artifact. A certain minimum number of counts is needed for a diagnostic image, and more counts result in a superior image. Table 3.1 offers suggestions for minimum counts/image. A longer image acquisition time is needed for more counts/image, although at some stage a long image acquisition time becomes impractical due to patient motion and time imaging. However, the nuclear medicine software can analyze images using algorithms, which are designed to remove artifact or noise to improve image quality. Using these computer algorithms to filter noise can be particularly useful when subtle pathologic change is present or if adequate counts cannot be obtained due to patient disposition.[31]

The risk with limiting the acquisition to a certain number of counts (as opposed to time) is that if there is urine contamination under a foot or another limb or the urinary bladder is in the field of view, then the counts recorded by the acquisition computer will include these aberrant γ-rays, which do not contribute to image quality. This will "steal" counts from the remaining anatomy and diminishing the number of γ-rays used for image reconstruction. This problem may be overcome with segmentation or repeat images of the areas' post cleaning. Typically, the number of counts gives more uniform images throughout the exam. Current software can acquire images with and without the imaging algorithm to improve image quality; however this is typically done in a dynamic acquisition at 2 seconds/frame for 40 frames.

METHOD FOR A SCINTIGRAPHIC EXAM OF THE MUSCULOSKELETAL SYSTEM

The radiolabeled pharmaceutical, either ^{99m}Tc-MDP (methylene diphosphonate) or ^{99m}Tc-HDP (oxidronate), is generally used at a dose of 0.35 mCi/kg (12.95 MBq/kg).

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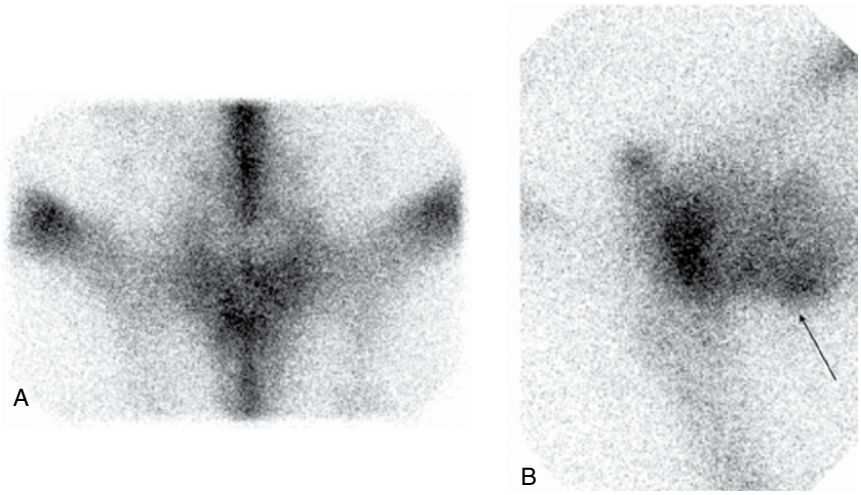


Figure 3.128. Delayed phase dorsal (A) and lateral (B) views of the pelvis of a normal horse. Note the diffuse abnormal radiotracer seen in the region of the sacrum on the dorsal view that corresponds to radioactive urine in the urinary bladder, as seen on the lateral image (arrow).

The radiolabel must be given intravenously; otherwise, slow release of the ^{99m}Tc will result in suboptimal images due to continuous release and thus high levels of circulating radioactivity. Patient control is very important because images generally take about 60–90 seconds to acquire. Chemical restraint is useful in reducing patient motion. Standing sedation protocols often vary among institution but often include alpha-2 antagonist with or without an opiate. Image processing software packages have integrated motion correction tools that allow some degree of motion with minimal or no image deterioration.

Imaging Technique

The radiotracer is given intravenously, and the blood flow (phase 1) images are acquired immediately if required. Pool phase images are acquired within the next 10 minutes if desired. Pool phase images must be limited to about three or four anatomical regions to ensure that they are completed before significant bone uptake occurs. Delayed phase images are acquired from 2 to 4 hours after injection to allow an optimal bone-to-soft tissue ratio. Furosemide may be given IV 60–90 minutes before the delayed phase starts if lumbar spine, pelvis, and stifle images are being acquired. This increases the chances of voided bladder because the ^{99m}Tc-HDP is excreted by the kidneys and urine in the bladder obscures visualization of the stifles, lumbosacral junction, sacroiliac (SI) joints, and the coxofemoral joints (Figure 3.128).

Lateral images of the limbs are made, being careful to position the camera lateral to the region being imaged (which is not necessarily lateral to the horse). Dorsal views of the carpi are generally performed. Orthogonal views of a lesion should always be attempted to help document the third dimension. Lead sheets are used to shield scatter radiation from the other limbs (Figure 3.129). Lead also should be placed medial to the olecranon and the stifle to shield the sternum and the penis/urinary bladder, respectively. Slightly overlapping the views will ensure that no area is left unscanned.



Figure 3.129. Gamma camera positioned in a pit below floor level for the lateral view of the right fore distal limb. Lead shielding is used to block out the contralateral limb.

There can be soft tissue uptake during the soft tissue (pool) phase up to 14 or 17 days after intra-articular or perineural anesthesia, respectively.[90,91] Local nerve blocks (intra-articular or perineural) do not, however, affect bone uptake in the delayed phase.[37]

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The following views are recommended for the full evaluation of the thoracic and pelvic limbs and spine using a detector with a 20-inch (50-cm) field of view.

Thoracic Limb

Lateral and dorsal views of the foot (these views include distal metacarpus, metacarpophalangeal joint, and phalanges), solar distal phalanx, lateral and dorsal carpus, lateral and cranial humerus, and lateral scapula. The metacarpus is included in the views of the foot and the carpus, the radius and elbows are included in the views of the carpus and humerus, and the shoulder is included in the views of the humerus and scapula. If an area of abnormal uptake is seen in the metacarpus, radius, or elbow, or in an area where only lateral views were obtained, additional images including orthogonal views centered over the areas of interest should be acquired.

Pelvic Limb

Lateral and plantar foot (these views include distal metatarsus, metatarsophalangeal joint, and phalanges), lateral and plantar tarsus, lateral and caudal stifle, lateral femur, right and left lateral, right and left dorsal oblique (RDO/LDO) hip, dorsal pelvis, caudal pelvis, and (RDO/LDO) sacrum. The metacarpus is included in the views of the foot and the tarsus, and the tibia is included in the views of the tarsus and stifle. As on the thoracic limb, if an area of abnormal radiotracer is seen on the metatarsus, tibia, or femur, additional images including plantar/caudal views centered over the area of interest should be obtained.

Spine

Right and left lateral views of the cervical spine (cranial, mid, and caudal), RDO/LDO cranial thoracic spine, RDO/LDO caudal thoracic spine, RDO/LDO lumbar spine, RDO/LDO lumbosacral junction (Figure 3.130), dorsal pelvis, caudal pelvis (tail on detector, TOD), and RDO/LDO sacrum. Lateral and dorsal views of the spine are less effective due to the great distance that the camera is from the vertebra. They can, however, be included as orthogonal views when a lesion is found. A point source, also known as fiducial marker, (e.g. cobalt marker or the syringe and needle that was used for the ^{99m}Tc-HDP injection, sealed in a latex glove) can be placed along the dorsum of the back to localize the exact position of a lesion, which is then marked with a permanent marker.



Figure 3.130. Gamma camera positioned for the right dorsal oblique (RDO) view of the thoracolumbar spine.

Foot

Different options exist for obtaining lateral, dorsal, and palmar views of the feet. In some institutions, the nuclear medicine suite has a pit in the floor into which the gamma camera is lowered to have it centered over the distal limb (Figure 3.131). In these cases, the horse is positioned to obtain lateral, dorsal, and palmar views. These views can also be made by placing both forefeet (or both hindfeet) on a wooden box about 25 cm high if a pit is not available. The top of the box should be strong enough (3.75-cm or 1.5-in., plywood) to support a large horse. The gamma camera can then be lowered to acquire the views. Construction of a platform for the horse to stand on during the entire exam can also be used. At these facilities the camera moves around the



Figure 3.131. Gamma camera positioned in a pit below floor level for the dorsal view of both fore distal limbs. Lead shielding is used to block out the hindlimbs to avoid shine through radiation and improve image quality.

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Figure 3.132. Gamma camera positioned for the solar view of the right forefoot.

horse and is placed where needed, including below foot level for lateral, dorsal, and palmar views of the foot. An alternative option is to have a ramp on which the horse can place the front feet or hindfeet. Both the platform and ramp take up more space, and in most cases the horse cannot remain still on it for the entire examination.

Solar views of the feet (for the navicular bone) are best made by stretching the forefoot out cranially and placing it on the camera face that has a plexiglass covering to protect the collimator (Figure 3.132). The solar view of the feet has been shown to be more sensitive for the evaluation of the navicular bone than the lateral view.[45] The camera is positioned face up in front of the horse, with the surface of the camera at or just below the level of the carpus. A solar view of the hindfoot is done less frequently and can be made by stretching the limb caudally, placing the foot on a wooden box and positioning the camera behind it. An alternative method is to have a pit in the floor into which the camera is placed face up. The pit is covered with appropriate material, and the horse stands over the camera. While it is easier to position the horse over the pit than to stretch the foot out forward and hold it on the camera, the downside is superimposition of the pastern and fetlock over the navicular bone region.

INDICATIONS FOR NUCLEAR SCINTIGRAPHY OF THE MUSCULOSKELETAL SYSTEM IN HORSES

Vascular Phase (Phase 1)

The vascular phase is an excellent technique for the evaluation of blood flow to specific areas and in the evaluation of trauma to the distal extremities or to areas where inadequate blood perfusion is suspected such as laminitis, frost bite, and vascular trauma from breakdown injury. The vascular phase can also be helpful in documenting aortoiliac thromboembolism. Increased blood flow to a particular region may be associated with acute inflammatory conditions or infectious processes. In most institutions, the vascular phase is not included as part of a routine scintigraphic exam of the musculoskeletal system.

Soft Tissue Phase (Phase 2)

Soft tissue phase (pool phase) images provide more useful information in cases of acute lameness, particularly in the distal limb, because of the ability to identify changes (especially increases) in blood flow to local areas. For example, hyperemia of the synovium or joint capsule secondary to acute synovitis/capsulitis, or at the proximal attachment of the suspensory ligament due to acute desmopathy and periligamentous inflammation, may be detected during the soft tissue phase.[28] Focal areas of trauma can also be evaluated for altered soft tissue perfusion. It can sometimes be difficult to differentiate between early bone uptake by a lesion and increased blood flow to an area. Therefore, the more accurate soft tissue phase images are those done with pertechnetate ($^{99m}TcO_4$) or labeled RBCs (^{99m}Tc-RBC) and not with a bone-seeking radiolabel.

The practitioner should be aware that detection of increased blood flow to a specific region is more common in acute conditions, and a negative result on a soft tissue phase does not rule out a subtle or more chronic injury.

Delayed Phase (Phase 3)

Delayed phase images provide information for evaluation of the skeleton. The high sensitivity of this phase to detect early changes in bone metabolism before these changes are radiographically evident makes this

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part of the study most useful for evaluation of acute lameness, e.g. incomplete or stress fractures in racehorses or high-performance horses. The delayed phase may also help in the diagnostic workup of horses presenting for other reasons such as ill-defined lameness or lameness that is difficult to diagnose with regional anesthesia, multiple causes of lameness in the same limb or different regions of the body, acute lameness of unknown origin, recheck of known lesion (to follow progress of healing), evaluation of the physiologic activity of radiographic lesions, and evaluation of areas that are difficult to radiograph such as the proximal thoracic and pelvic limb, spine, and pelvis including the SI and coxofemoral joints. Delayed phase imaging is also useful for the assessment of bone viability and as a general survey in prepurchase examinations. Soft tissue uptake in the muscles, seen during the delayed phase, can be seen in cases of rhabdomyolysis[13,59] or dystrophic mineralization of soft tissues (Figure 3.133).

A scintigraphic examination should not replace a complete physical and lameness examination. When evaluated as a stand-alone technique, nuclear scintigraphy does not meet the requirement for a diagnostic test.[65] False-negative scintigraphic results do not rule out a possible source of lameness suspected during clinical evaluation of the patient.

NORMAL BONE SCAN

Vascular phase images are best viewed as a cine loop on the acquisition computer (a computer software application that allows the images to be viewed sequentially in rapid order). A composite of vascular phase images also can be generated, and it should show good perfusion. When looking at the distal limbs, try to have both limbs in a dorsal or palmar/plantar view and look for perfusion symmetry. Composite images of the distal aorta should show the bifurcation of the aorta into the internal and external iliac arteries (Figure 3.134).

Soft tissue (pool) phase images of the foot show some vascular activity, but the fetlock and pastern regions should have homogeneously smooth uptake (Figure 3.135). The palmar/plantar blood vessels are seen as a distinct linear activity, and the coronet also has increased activity due to a vascular plexus. Additionally, the distal phalanx has a generous blood supply to the sensitive laminae, and increased activity is seen. Soft tissue images proximal to the carpus or tarsus are usually unrewarding due to the normal radiotracer of overlying musculature.[24]

Normal delayed phase uptake patterns vary among horses, depending on the patient's age, breed, and

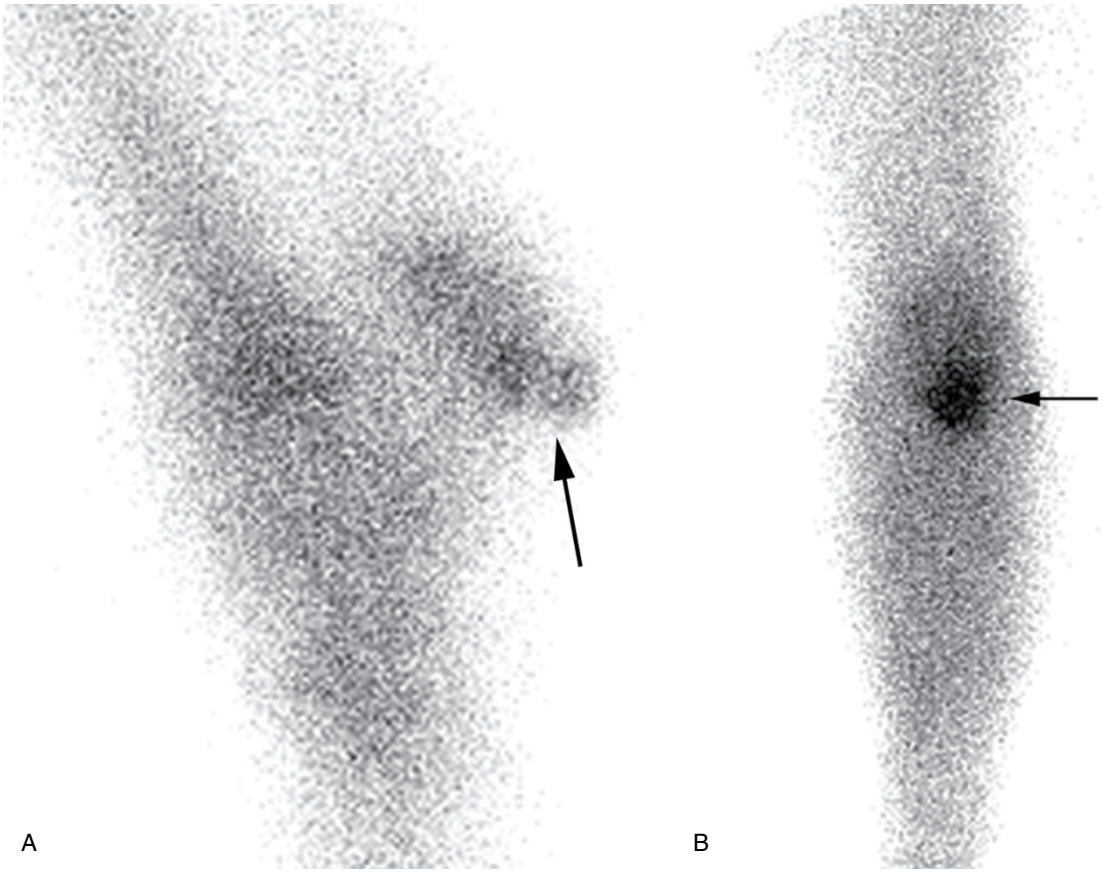


Figure 3.133. Delayed phase lateral (A) and plantar (B) views of the left tarsus, showing a focal moderate abnormal radiotracer area within the soft tissues just plantar and distal to the calcaneal tuberosity (arrows), corresponding to dystrophic mineralization of the superficial digital flexor tendon. *Source*: Courtesy of Dr. Erik Bergman.

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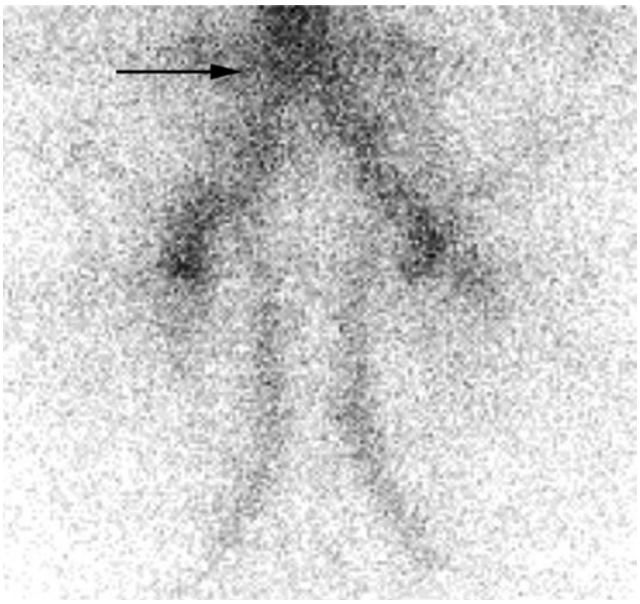


Figure 3.134. Composite image of blood flow in the region of the caudal aorta, showing normal bifurcation of the aorta (arrow) into internal and external iliac arteries.

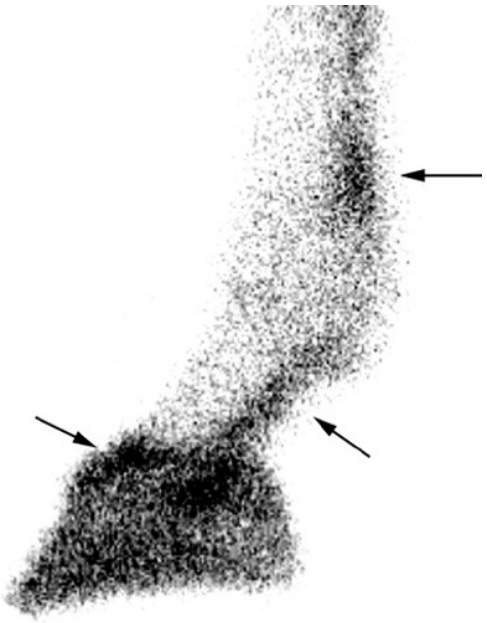


Figure 3.135. Soft tissue (pool) phase view of a normal left forefoot, showing vascular activity on the palmar aspects proximal and distal to the fetlock and in the area of the coronary band (arrows). Note that the fetlock and pastern regions have homogeneously smooth uptake.

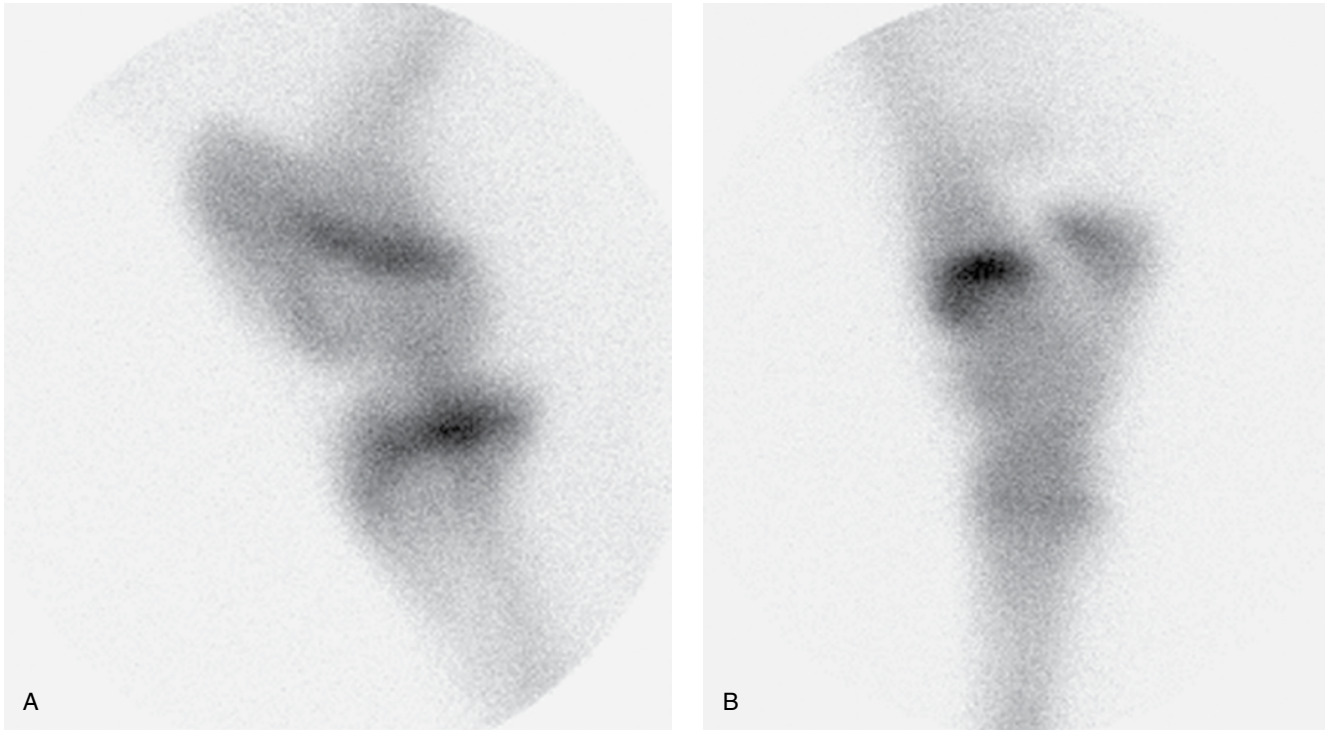


Figure 3.136. Delayed phase lateral views of the left stifle (A) and left tarsus (B) of a normal skeletally immature horse, showing normal intense radiotracer in the region of the physes. *Source*: Courtesy of Dr. Erik Bergman.

occupation.[96] For example, in athletic horses, most of these normal variations represent an exercise-induced bone remodeling secondary to an ongoing bone response to stress and do not necessarily produce clinical signs of lameness. Skeletally immature animals normally have bilaterally symmetric increased areas of uptake at growth plates and secondary centers of ossification (Figure 3.136).

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The diaphyses of long bones have less uptake than other parts of the bones, due to relatively low metabolism in the diaphyses of normal subjects. The uptake should, however, be smoothly homogenous with no focal areas of increased uptake. Contralateral imaging can be useful when evaluating borderline lesions. Physes, epiphyses, and apophyses demonstrate increased uptake due to increased metabolic rates of bone tissue in these regions.

Distal Limb (Foot, Pastern, and Fetlock)

On the lateral view, a normal horse has similar uptake in the coffin, pastern, and fetlock joints. The navicular bone should not be seen as a distinct entity due to a homogeneous uptake with the rest of the distal phalanx (Figure 3.137). Normal mild decreased uptake in the metacarpal condyles, when compared with the proximal phalanx and sesamoid bones, has also been reported.[100] The dorsal view (plantar view if evaluating the pelvic limbs) is useful for comparison of ipsilateral structures in the same image (Figure 3.138).

The solar view of a normal horse has a very uniform pattern of uptake with less than 10% change between the regions of the navicular bone, deep digital flexor tendon (DDFT) insertion, toe, and medial and lateral aspects of distal phalanx (Figure 3.139).[21] Mild increases in radiotracer can be seen at the insertion of the DDFT or the lateral or medial aspects of distal phalanx secondary to foot conformation. Radiotracer is often seen in the medial aspect of the distal phalanx, palmar cortex of the proximal phalanx, and proximal sesamoid bones as horses start into training.[35]



Figure 3.137. Delayed phase lateral view of the distal limb of a normal horse. Note the similar uptake between the fetlock, pastern, and coffin joints.



Figure 3.138. Delayed phase dorsal view of both fore distal limbs of a normal horse (SHINE processing in place). The uptake should be uniformly similar between both limbs.



Figure 3.139. Delayed phase solar view of a normal horse. Note the homogenous uptake throughout the entire image without distinction of any particular area.

Incidental bilateral focal increase radiotracer can be seen at the dorsal mid aspect of the proximal phalanx in dressage, show jumping, or eventing horses (Figure 3.140).[6] The palmar or plantar cortices may also show focal abnormal radiotracer. The etiology of this uptake is likely due to adaptive remodeling from a normal response to training.

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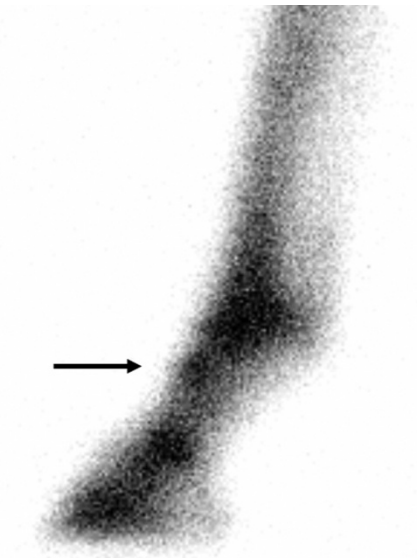


Figure 3.140. Delayed phase lateral view of the left distal limb of a normal horse. The focal moderate radiotracer on the dorsoproximal diaphysis of P1 (arrow) is a normal finding as a response to the athletic activity usually seen bilaterally in performance horses.



Figure 3.141. Delayed phase lateral view of the left metacarpus of a normal horse. Note the uniform uptake along the diaphysis.

Metacarpus and Metatarsus

There should be a uniform uptake along the diaphysis of the metacarpus and metatarsus with no distinction between the second, third, and fourth metacarpal bones (Figure 3.141).

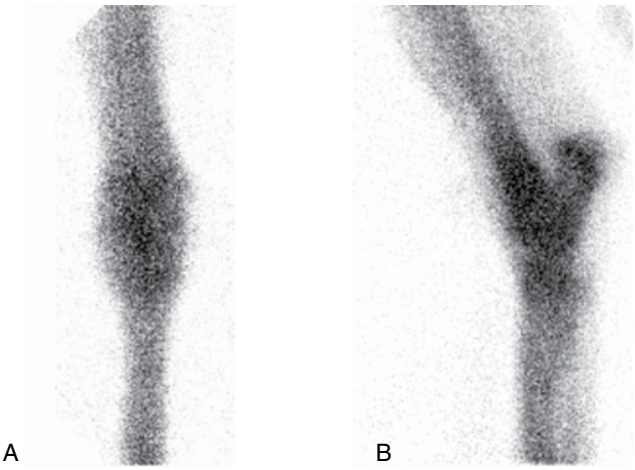


Figure 3.142. Delayed phase dorsal view of the carpus (A) and lateral view of the tarsus (B) of a normal horse.

Carpus and Tarsus

Normal radiotracer distribution is uniformly greater in the carpal and tarsal bones when compared with the diaphysis of the metacarpus and radius or metatarsus and tibia, respectively (Figure 3.142). Focal areas of increased radiotracer in the cuboidal bones are typically considered abnormal. However, a general increase in the distal row of carpal bones can be seen as horses begin or are in early training.[35]

Upper Limb Long Bones

The distal physis of the radius and tibia can be seen with an increased uptake for several years after radiographic closure. The radial physis can be seen past the age of 8 years. Growth is no longer occurring at these sites, but there is still sufficient osteoblastic activity to differentiate the physis from the adjacent metaphysis and the epiphysis. The increased radiotracer in those areas is the result of different histologic architecture after closure, which exposes more bone crystal to diphosphonate binding.[96] A uniform pattern of uptake should be seen along the diaphysis of the normal tibia[97] and radius. The deltoid tuberosity is easily visualized as an area of greater radiotracer on the cranioproximal cortex of the humerus due to the closer proximity of the bone to the gamma camera (Figure 3.143). The third trochanter is an important landmark and should be seen as an area of greater radiotracer because of proximity to the camera. The cranial and caudal parts of the greater trochanter should be identified as separate structures (Figure 3.144).

Elbow, Shoulder, and Stifle Joints

Increased radiotracer is often seen in the radioulnar joint of normal elbows (Figure 3.145). The normal shoulder joint demonstrates increased uptake in the areas of the greater and lesser tubercles and the humeral head (Figure 3.146). The glenoid cavity, however, should have less activity than the humeral head.

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Figure 3.143. Delayed phase lateral view of the shoulder of a normal horse. A normal focal intense area of radiotracer on the cranial aspect of the proximal diaphysis represents the normal deltoid tuberosity (arrow).

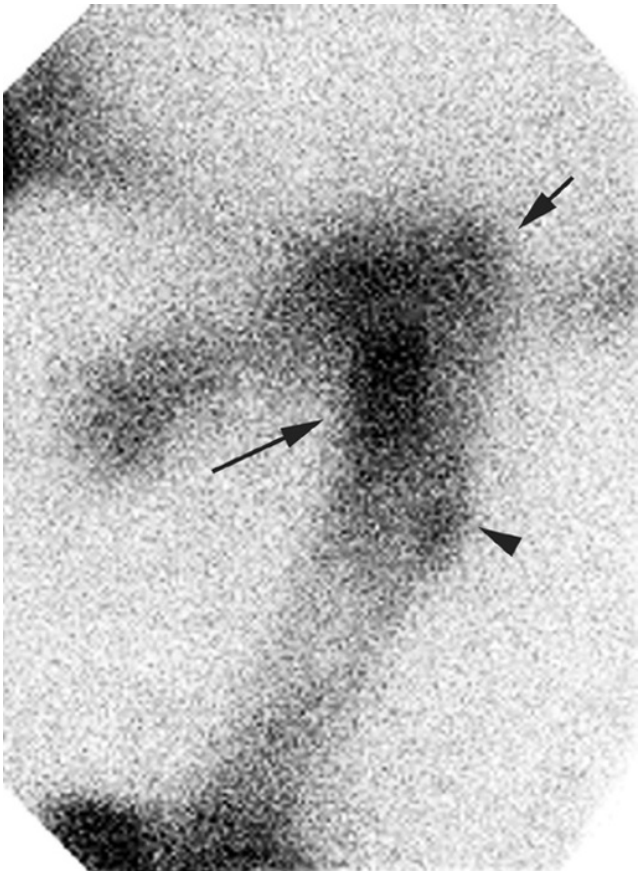


Figure 3.144. Delayed phase lateral view of the femur of a normal horse. Note the cranial (large arrow) and caudal (small arrow) parts of the greater trochanter and the third trochanter (arrowhead) as separate structures.



Figure 3.145. Delayed phase lateral view of the elbow joint of a normal horse.



Figure 3.146. Delayed phase lateral view of the shoulder joint of a normal horse.

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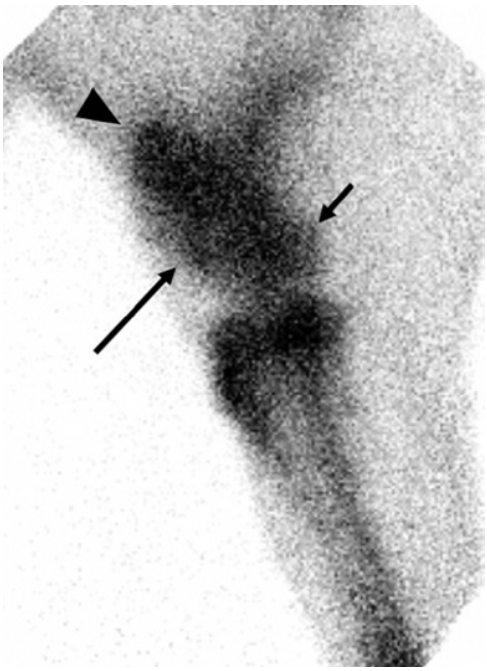


Figure 3.147. Delayed phase lateral view of the stifle joint of a normal horse. Note the uniform uptake between the patella (arrowhead), trochlea (large arrow), and condyles (small arrow).

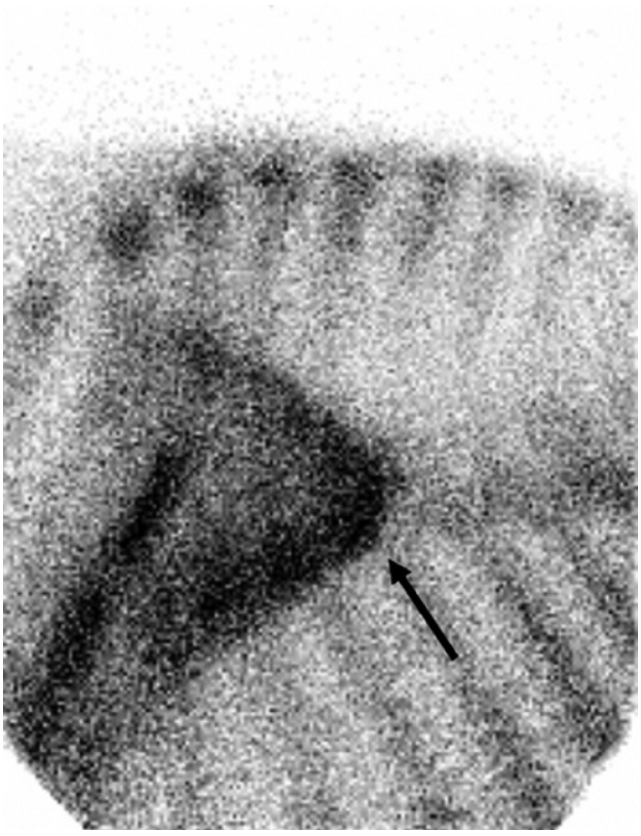


Figure 3.148. Delayed phase lateral view of the scapula of a normal horse. Note the spine of the scapula and a superimposition artifact (arrow) created by overlap between the caudodorsal angle of the scapula and the thoracic vertebral bodies.

A similar uptake between the patella, trochlea, and condyles should be seen in the stifle joint (Figure 3.147). However, in adult horses it is also normal to see a mild increases in radiotracer on the patella secondary to adaptive bone remodeling.[26] In immature horses, a normal bilobed appearance of the proximal tibia is commonly seen, corresponding to the center of ossification of the tibial tuberosity and the superimposition of the caudal tibial plateau and the head of the fibula.[26,71] Adult horses show less uptake at the tibial tuberosity than the caudal tibial plateau/fibular head region. In addition to the superimposition of the head of the fibula and the caudal aspect of the tibial plateau, increased radiotracer in this region is also attributed to the weight-bearing and greater load transmission in this area.[26]

Scapula

The scapula should be easy to visualize, and in some cases the withers can be evaluated at the same time (Figure 3.148). Superimposition artifact from the withers and thoracic spine often occurs on images of the scapula, creating an area of perceived increased uptake. The caudal border and the spine of the scapula have normally greater uptake than the supra- and infraspinatus fossae.

Pelvis, SI, and Coxofemoral Joints

The dorsal view of the pelvis is often used for the evaluation of the tuber sacrale and SI region. The uptake is normally increased due to the close distance and minimal soft tissue attenuation between the sacral tuberosities and the γ-ray detector (Figure 3.149). The tuber sacrale in the young horse is well defined. This tends to diminish with age, having more radiotracer lateral to the tuber sacrale. The SI joints can be evaluated on a dorsal view or a dorsal 45° oblique view. Asymmetric uptake can be better visualized on the dorsal oblique view, and

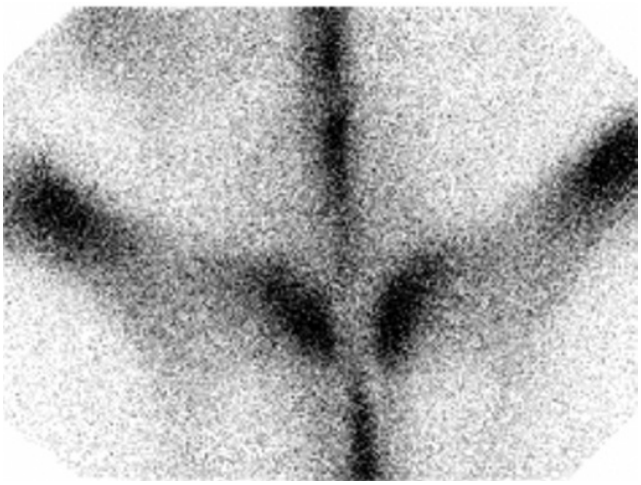


Figure 3.149. Delayed phase dorsal view of the sacrum of a normal horse. The two oval and symmetric focal areas of increased radiotracer correspond to the sacral tuberosities. The relative distinction of this area diminishes with age.

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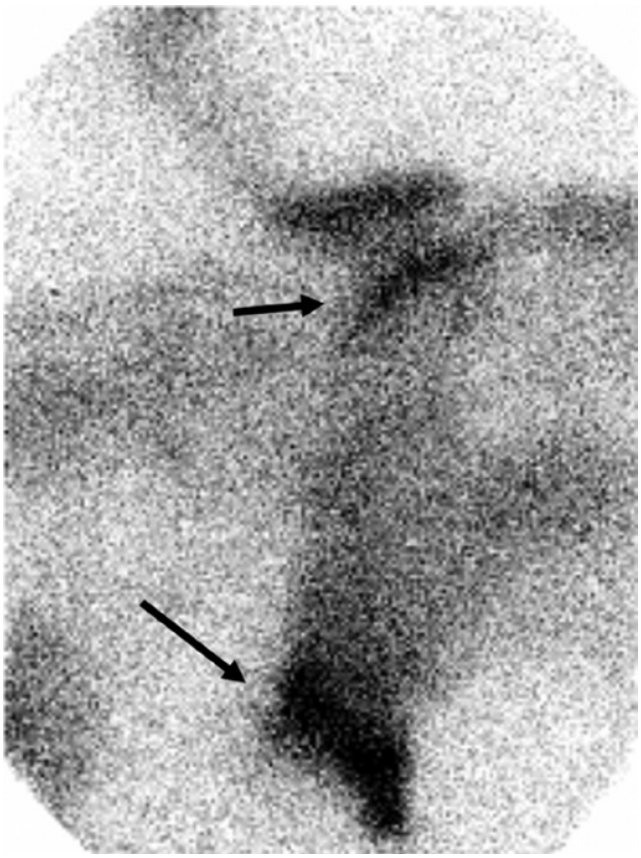


Figure 3.150. Delayed phase oblique view of the sacroiliac joint of a normal horse. Note the sacral (small arrow) and iliac (large arrow) tuberosities.

the entire extent of the joint can be better seen on the oblique view with less superimposition of pelvic canal structures (Figure 3.150). Although the dorsum of the sixth and seventh lumbar vertebrae and the sacrum can also be seen on the dorsal view, the spinous processes are best evaluated on the dorsal oblique views.

The caudal view of the pelvis (tail-on detector view, TOD) helps in the evaluation of the floor of the pelvis and the tuber ischia and proximal aspect of the femurs (Figure 3.151). The camera is positioned caudal to the pelvis of the horse for the TOD view. Ensuring the gamma camera is equidistant from the left and right tuber ischii during the acquisition of this view will reduce false-positive increase radiotracer due to asymmetric distance from the camera. Asymmetric uptake by the tuber ischii is considered abnormal. Angling the camera dorsally (slope of the rump) should be done if the urinary bladder interferes with the image. This projects the bladder above the tuber ischii.

Lateral images of the coxofemoral joint should identify the cranial and caudal portions of the greater trochanter and third trochanter of the femur (Figure 3.152). Commonly, increased radiotracer is seen in the coxofemoral joint region on the lateral view because the cranial part of the greater trochanter (convexity) is partially superimposed over the joint region. Dorsal oblique views (approximately 45°) of the coxofemoral joint allow improved visualization of the femoral head and neck and acetabular region

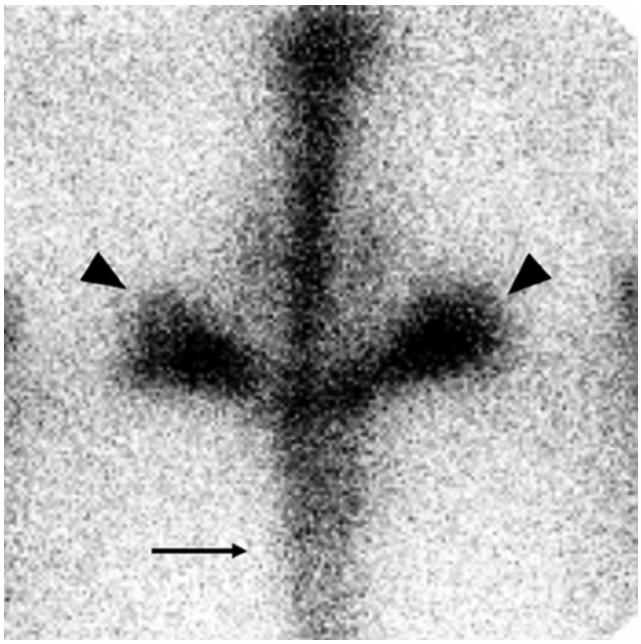


Figure 3.151. Delayed phase tail on detector (TOD) view of a normal horse. Note the good visualization of the floor of the pelvis, especially the symmetric ischiatic tuberosities (arrowheads). The tail is seen as a midline structure (arrow).

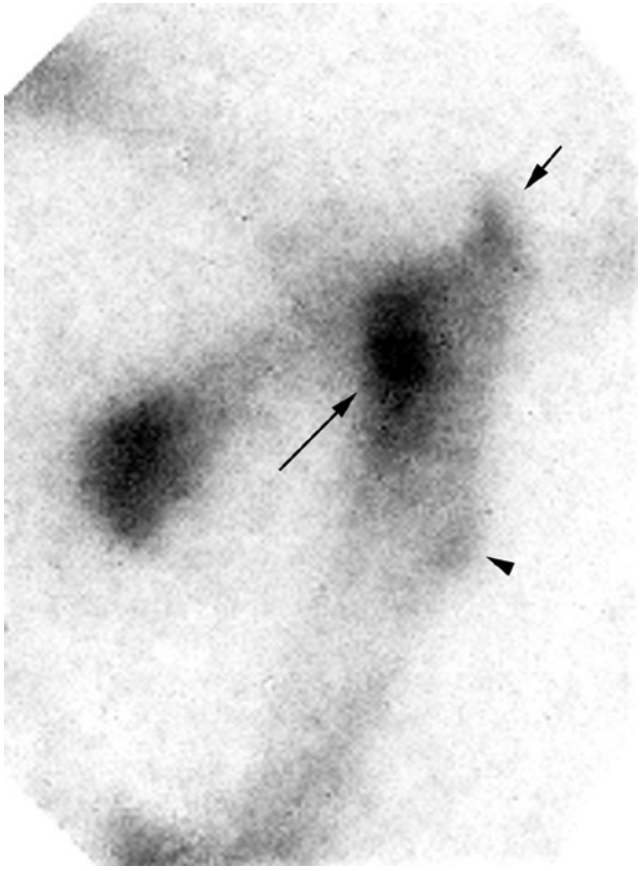


Figure 3.152. Delayed phase lateral view of the left coxofemoral joint of a normal horse. Note the cranial (large arrow) and caudal (small arrow) parts of the greater trochanter and the third trochanter (arrowhead) as separate structures.

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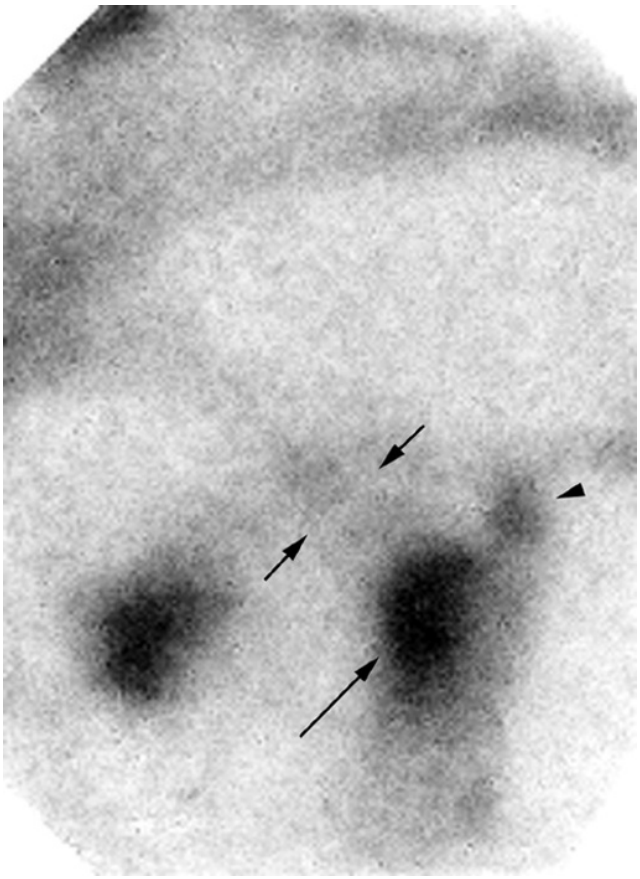


Figure 3.153. Delayed phase oblique view of the left coxofemoral joint of a normal horse. The coxofemoral joint is located between the two small arrows and is not superimposed over the greater trochanter. The cranial and caudal parts of the greater trochanter (large arrow and arrowhead, respectively) are still visualized as separate structures.

without superimposition of other structures (Figure 3.153). The normal acetabulum should not be seen as a separate entity (i.e. the acetabulum should not have a signal greater that of the ilium). Urine in the urinary bladder sometimes obscures visualization of the hip joint and can be mistaken for disease. Dorsal or caudal views of the hip region should be made to differentiate the urinary bladder (a midline structure) from the coxofemoral joints.

Spine

Lateral images of the cervical spine should show similar uptake between articular facets and their corresponding vertebral bodies with the exception of the C6–C7 articular facets. Typically a relatively higher uptake is seen in the body of C6 due to the shorter and thicker transverse processes. Similar uptake should be seen between cervical articular facets at adjacent levels (Figure 3.154). The region of the dens usually has a greater radiotracer than the surrounding bony structures (Figure 3.155).

The thoracic and lumbar spine can be evaluated with lateral images; however, the large rib cage and epaxial musculature maintain a great distance between the vertebrae and the γ-ray detector, making subtle areas of increased uptake difficult to detect. Therefore, LDO and

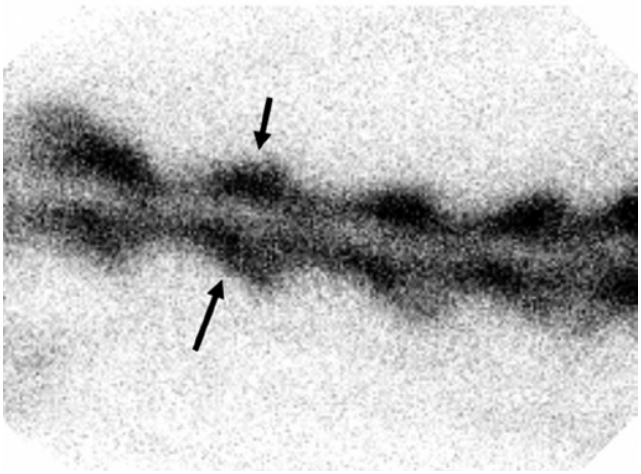


Figure 3.154. Delayed phase right lateral view of the mid-cervical region of a normal horse. Note the similar uptake between the articular facets (short arrow) and the vertebral body (large arrow).

RDO images are invaluable for the evaluation of the spine (Figure 3.156). When obtaining the dorsal oblique view, care should be taken when evaluating the caudal thoracic vertebrae for superimposition of the vertebral bodies and the normal increased uptake in the right kidney. Overlap should exist between views to include all anatomical regions. Resolution of the dorsal spinous and transverse processes should be possible in all horses; however, patients greater than 1,500 lbs. can be so bulky that the γ-rays may be reduced or attenuated significantly enough to reduce dorsal spine and transverse process resolution. The most dorsal aspect of the spinous processes of the withers usually have normal intense focal areas of radiotracer (Figure 3.157). Adjacent spinal units should have similar amounts of uptake to each other. Be sure to evaluate the dorsal spines, articular facets, and transverse processes of the entire spine, including the sacrum and tail head (sacrococcygeal region).

The dorsal view provides limited information in most clinical cases. However, soft tissue and delay phase dorsal views should be obtained when a lesion is suspected after clinical evaluation.

Although the distribution of the radiopharmaceutical in the normal horse, young and old, is predictable, experience is necessary for interpreting bone scans. Comparing the relative uptake in opposite limbs, or comparing images from horses of a similar age, can assist in arriving at significant conclusions.

It is important to look at relative uptake between structures on the same image and then compare it with the ratio between the same structures on the contralateral image. This is important because of the phenomenon that occurs when an entire leg might have less uptake than the opposite limb. In these cases it is inappropriate to compare the uptake of, for example, the fetlock of one limb with the uptake of the fetlock of the other. It is more accurate to evaluate the uptake ratio between the fetlock and the pastern region in one limb and compare it with the same ratio in the other limb. This reduces the overreading of radiopharmaceutical uptake disparities that occur as natural phenomena or

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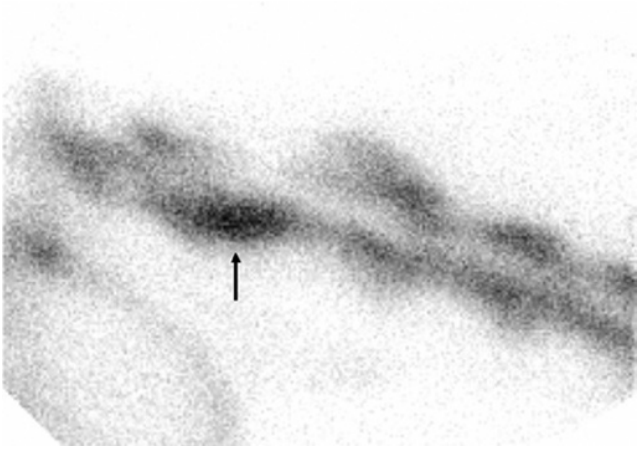


Figure 3.155. Delayed phase left lateral view of the cranial cervical region of a normal horse. Note the normal radiotracer in the region of the dens (arrow).

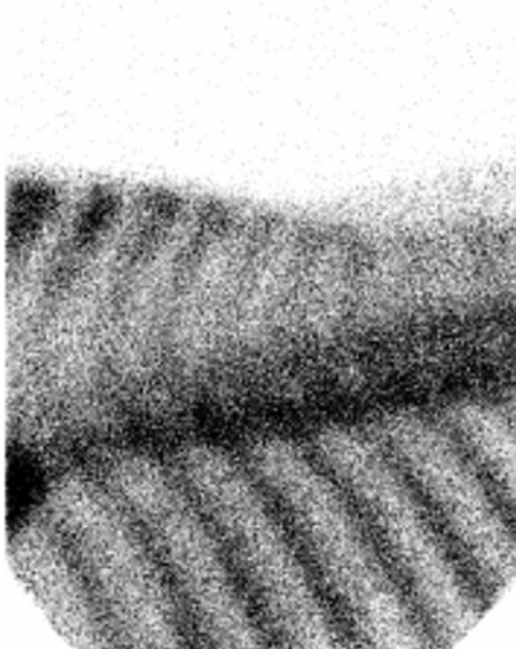


Figure 3.156. Delayed phase dorsal oblique view of the thoracic spine of a normal horse. Note the good resolution of the dorsal spinous processes and the ribs.

as image acquisition artifacts (for example, camera-body-distance inequalities, collimator decentering, etc.). The same interpretation philosophy of comparing relative uptake ratios should be used in other body regions. Generally speaking, the more intense the uptake, the more severe the condition.

SCINTIGRAPHIC SIGNS OF DISEASE

Vascular Phase

Increased blood flow can be seen as a subacute or chronic response to trauma during the healing phase.

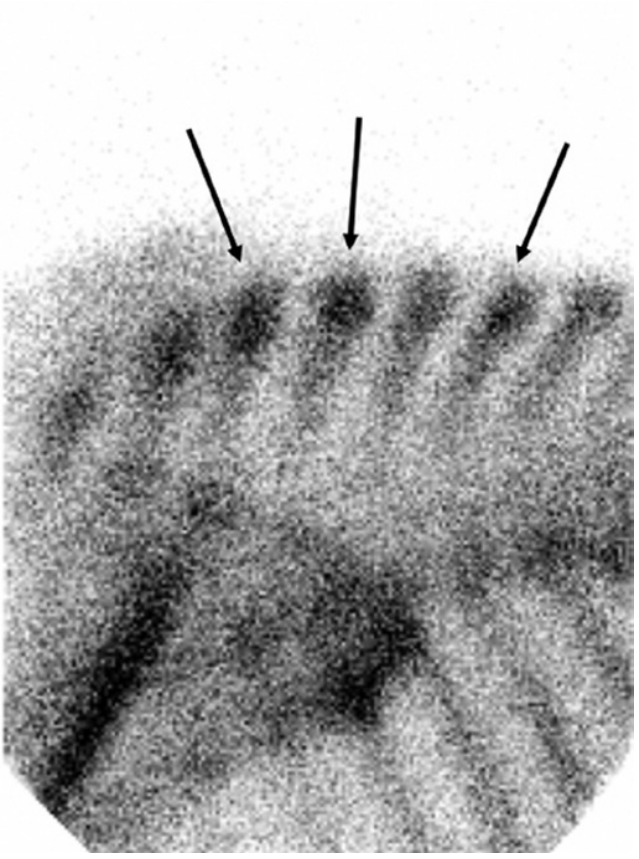


Figure 3.157. Delayed phase left dorsal oblique (LDO) view of the cranial thoracic spine of a normal horse. Note the normal radiotracer at the summit of the spinous processes of the withers (arrows).

Vascular imaging is useful to determine the amount of perfusion to the distal extremity and can therefore help determine the best therapy as well as prognosis. It generally takes about one minute for the leading edge of the tracer to reach the distal limb. The radiopharmaceutical can reach the distal limb quicker if the horse is sedated with acepromazine.[78]

The images can be viewed either individually or as a composite image, or the data from the individual images can be displayed in the form of a graph. Figure 3.158 illustrates the composite image of the vascular phase of a horse that suffered from a near-degloving injury to the distal left metacarpus 1 week prior. Ninety images of 2 seconds each were acquired after the intravenous administration of about 150 mCi ^{99m}Tc-O_4. The study was performed to evaluate whether there was blood flow to the pastern region.

The composite image shows increased blood flow to the distal metacarpus, where granulation tissue had already started to form. The pastern, however, showed more blood flow than the (normal) contralateral pastern region. This was thought to be due to reflex hyperemia secondary to the healing process and was considered a positive sign, indicating that there was good blood flow to the foot. Figure 3.159 is a graphical representation of the vascular phase. The curve represents the amount of

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radioactivity in the left and right pastern regions, determined by the region of interest that was drawn around them. Each data point on the curve is 1 of the 90 images. Quantitation of the respective blood flow can be done by determining the area under the curve of each pastern and calculating the percentage of total flow. Empirical evaluation of the images is generally sufficient in most clinical cases.

Decreased blood flow to a region is often associated with nonviable bone or other tissue. Although rare, this can be used to evaluate large sequestra, cases of severe blood flow compromise from a traumatic/degloving injury (especially distal limb), or different conditions such as aortoiliac thrombosis.[18]

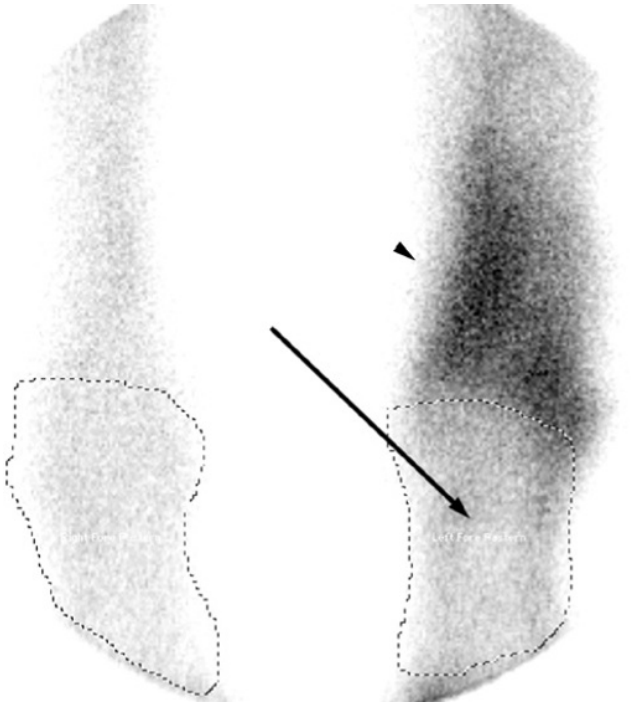


Figure 3.158. Composite vascular phase dorsal view of the pastern area of a horse 1 week after a near-degloving injury to the distal left front cannon bone region, showing moderately increased blood flow to the left pastern region (arrow) and markedly increased blood flow to the distal left cannon bone region (arrowhead).

Soft Tissue Phase

Increased activity in soft tissues is a good method to document increased blood flow to specific regions, for example, joints with synovitis/capsulitis. In these cases, nuclear scintigraphy is sensitive at detecting increased periarticular blood flow around inflamed joints before radiographic changes of OA are evident. Fetlock joint capsulitis has a region greater radiotracer over the joint when compared with the distal metacarpal region and proximal phalanx (Figure 3.160; compare with normal, Figure 3.135). Increased soft tissue phase uptake with a normal delayed phase image is compatible with a more acute degenerative condition, whereas if it is accompanied by increased uptake in the delayed phase, the condition is probably more chronic in nature.

Soft tissue phase imaging may be helpful in diagnosing enthesopathy and desmopathy of the proximal attachment of the suspensory ligament. These injuries may not be evident on ultrasound or radiographic studies. Deep lying regions, for example, the coxofemoral and SI joints, are difficult if not impossible to evaluate during the soft tissue phase because of the meager nature of the signal and the large amount of

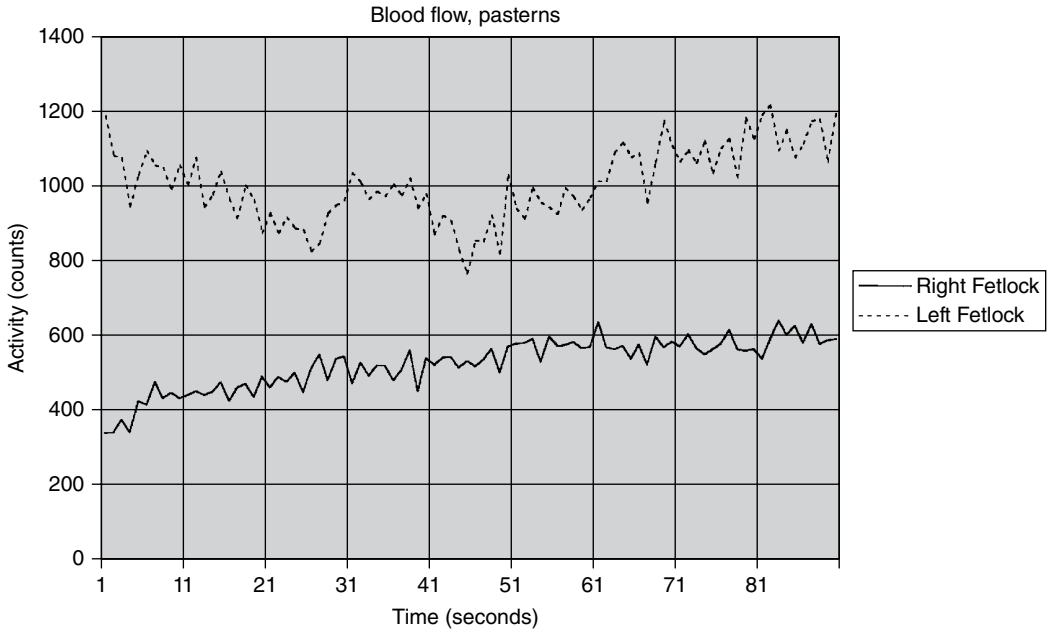


Figure 3.159. Graph of the data from the composite image shown in Figure 3.158, confirming that the left fetlock region had more blood flow than the right one.

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other tissues between the hip and the camera. These tissues make up several half-value layers (a layer of tissue resulting in the reduction of the signal by one half), which attenuate the beam significantly before reaching the gamma camera.



Figure 3.160. Soft tissue (pool) phase image of the left forefoot, showing increased blood flow to the fetlock region (arrow), which is suggestive of hyperemia associated with joint synovitis and/or capsulitis. Figure 3.135 provides the normal comparison.

Care must be taken to not overinterpret soft tissue phase images when severe increased uptake is also seen in the delayed phase images. Soft tissue phase "hot spots" in these cases often represent early bone uptake. For example, the soft tissue image in Figure 3.161a probably represents early bone uptake of the radiopharmaceutical by the proximal phalanx because of the intense uptake seen in the proximal phalanx in the delayed phase (Figure 3.161b). This horse had a fissure fracture of P1 that was also seen radiographically (Figure 3.161c).

Delayed Phase

Regions with increased blood flow and osteoblastic activity demonstrate increased uptake of the radiopharmaceutical. The severity or intensity of the increased uptake can vary and is often associated with conditions such as fractures, stress fractures, OA, enthesopathy, osteomyelitis, and neoplasia. Fractures and infectious processes have similar scintigraphic behavior in most bones, and in some cases it is difficult to differentiate between the two conditions. Therefore, correlation with clinical signs and other imaging findings is extremely important for making the diagnosis.

The amount of abnormal radiotracer that a fracture demonstrates may help determine the time of onset (acute vs. chronic) or the nature (pathologic vs. traumatic) of the fracture. Chronic and subacute fractures (older than 48 hours) have intense increased uptake due to the considerable osteoblastic activity (Figure 3.162). Acute fractures have less radiotracer because it takes approximately 24 hours for the osteoblastic activity to be greater than surrounding bone. In fact, acute

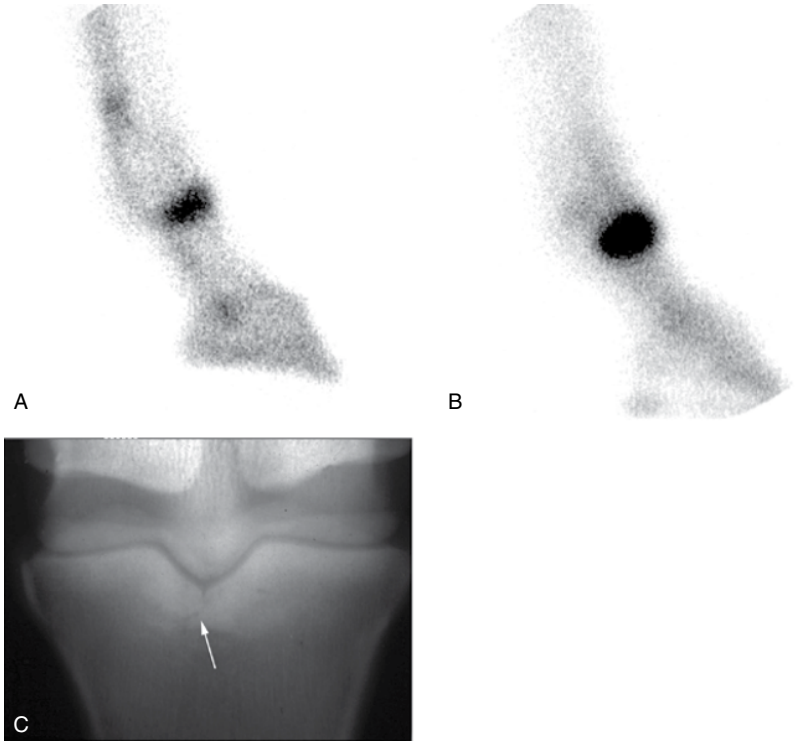


Figure 3.161. Right forefoot of a horse suffering from a chronic proximal first phalanx fracture. (A) Soft tissue (pool) phase image showing marked increased blood flow to the proximal portion of the first phalanx. (B) Delayed phase image showing marked abnormal radiotracer by the proximal portion of the first phalanx. (C) Radiograph showing a proximal first phalanx fracture (arrow). The radiotracer seen in the soft tissue image likely represents early radiopharmaceutical uptake by the bone, not just increased blood flow, because of the marked abnormal radiotracer seen in the delayed phase image.

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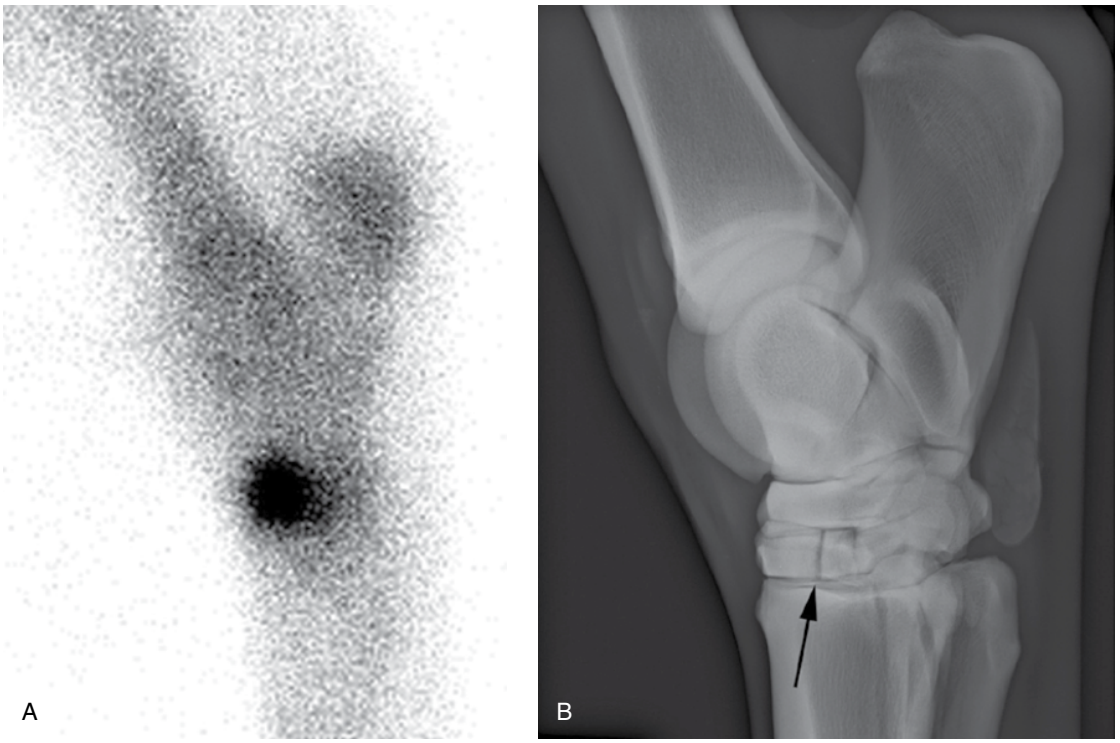


Figure 3.162. (A) Delayed phase lateral view of the left tarsus of a horse with focal and intense abnormal radiotracer in the region of the distal tarsus corresponding to a third tarsal bone fracture. (B) Lateromedial radiograph of the same tarsus showing the well-defined radiolucent fracture line extending from the proximal to the distal articular surfaces of the third tarsal bone, consistent with a slab fracture (arrow). *Source*: Courtesy of Dr. Erik Bergman.

traumatic fractures less than 24 hours' duration fail to show increased tracer uptake when compared with adjacent bone. Figure 3.163 is a delayed phase image of a comminuted middle phalangeal fracture 48 hours' post-injury. Although mild increased uptake is seen, the fracture is best diagnosed due to anatomic abnormality and not its physiologic peculiarity. Compare this with Figure 3.161, a chronic proximal phalangeal fracture with intense uptake but minimal anatomic displacement. Fracture uptake in humans is expected at about 24 hours' post-injury (although it takes longer in older patients) and is expected to last for 6–12 months or longer in older patients.[88] The uptake by a fracture should decrease over time as fracture healing occurs.

Multifocal areas of abnormal radiotracer have been described with different diseases such as enostosis-like lesions,[7,66] hypertrophic osteopathy,[50] neoplasia,[27,43] and horses with a bone fragility disorder,[2] a recently reported condition of unknown etiology that affects the axial and proximal appendicular skeleton. Figure 3.164 shows intestinal adenocarcinoma metastases to the ribs and distal left humerus. The same horse also had metastatic disease to several cervical, thoracic, and lumbar vertebrae, multiple ribs, and the sternum.

Localized delayed phase uptake of the radiopharmaceutical by soft tissues is not commonly seen and can occur with various conditions, e.g. dystrophic mineralization of ligament and tendon injuries, regional anesthesia,[1] rhabdomyolysis,[13,41,59] and repeated intramuscular injection of butorphanol[49] (Figures 3.133, 3.165,



Figure 3.163. Delayed phase image of a comminuted left front middle phalangeal fracture 48 hours' post-injury. Anatomic displacement is apparent, although radiopharmaceutical uptake is only mildly increased.

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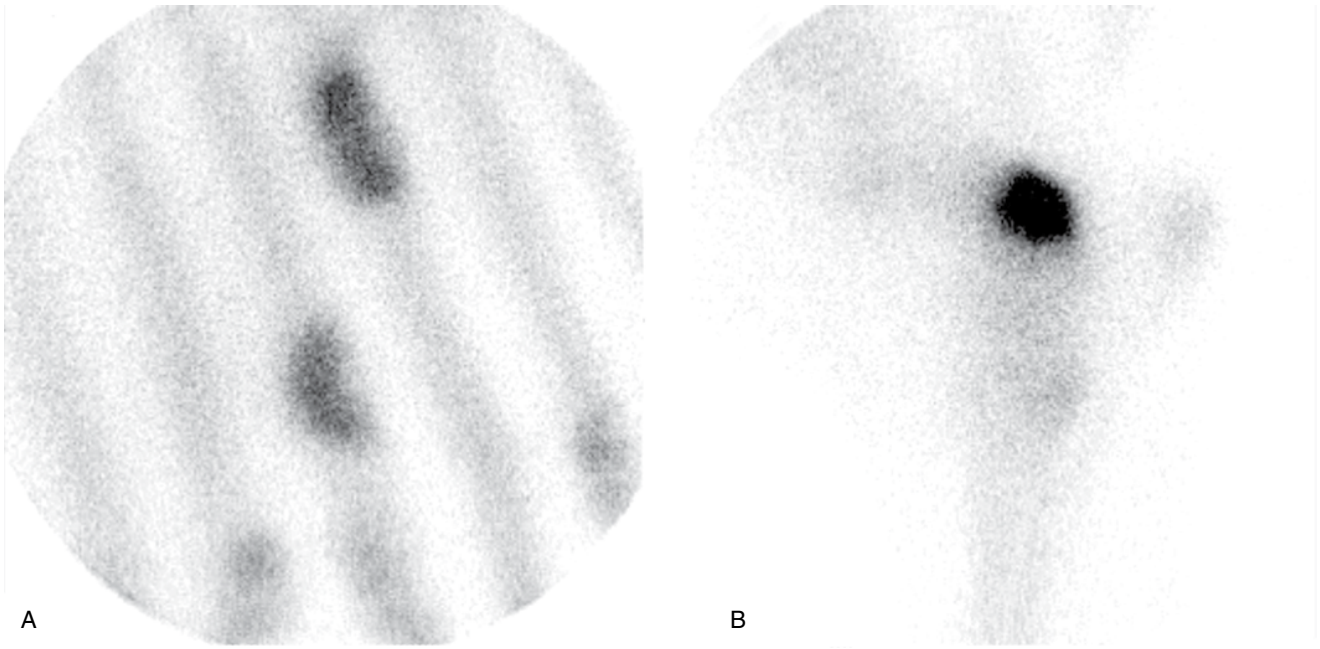


Figure 3.164. Delayed phase image showing a metastatic intestinal adenocarcinoma, which resulted in focal and intense abnormal radiotracer in (among others) the ribs (A) and distal left humerus (B).

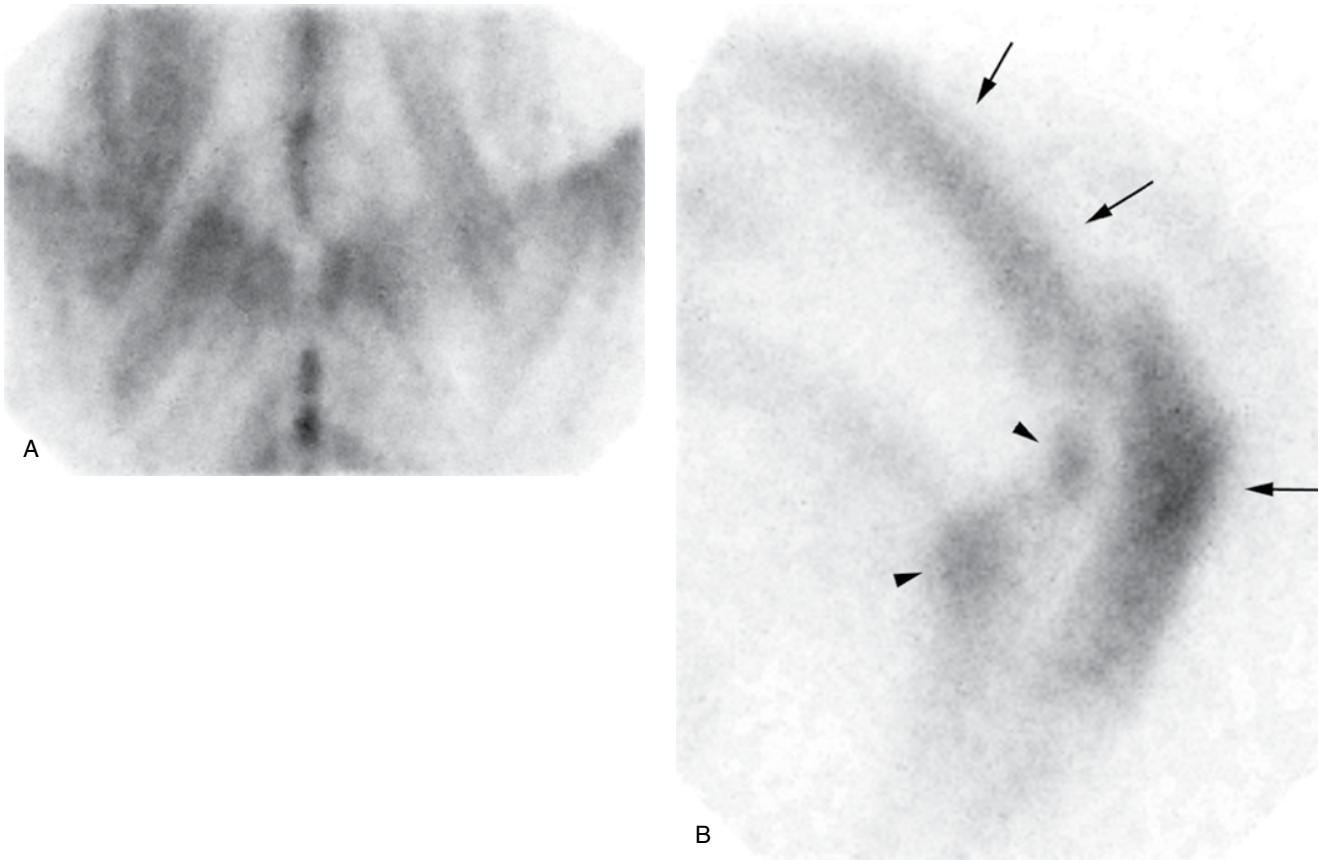


Figure 3.165. Delayed phase dorsal (A) and lateral (B) views of the pelvis, showing linear abnormal radiotracer (arrows) in the muscles of a horse with rhabdomyolysis (tying-up syndrome). The arrowheads correspond to normal bone uptake within the pelvis. *Source*: Courtesy of Dr. Kent Allen.

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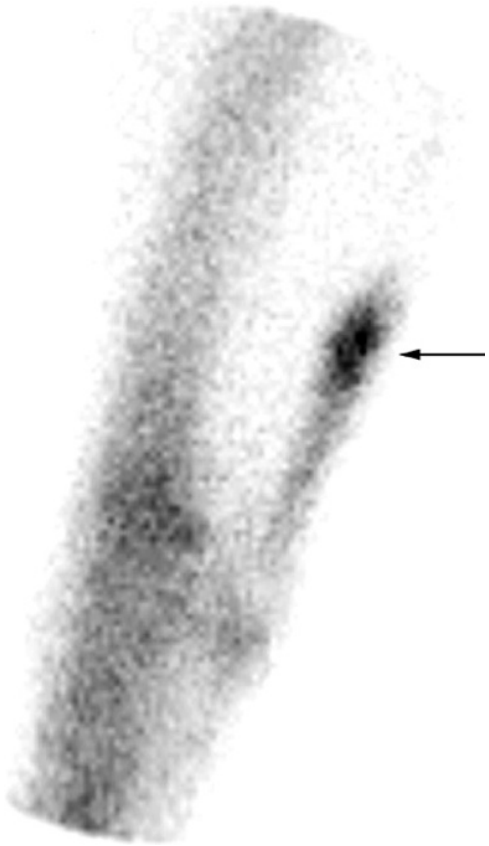


Figure 3.166. Lateral delayed phase view of the left carpus obtained 3 weeks after an ulnar nerve block. Note the focal moderate to marked soft tissue radiotracer at the injection site proximal to the accessory carpal bone (arrow).

and 3.166). Rhabdomyolysis (tying up) is seen as linear or diffuse uptake patterns in the muscles, such as the gluteals, semimembranosus, and semitendinosus.

ABNORMAL CONDITIONS FOR SPECIFIC ANATOMICAL REGIONS

Distal Limb (Foot, Pastern, and Fetlock)

The distal phalanx and navicular bone can experience different pathologic conditions involving the osseous anatomy or the multiple soft tissue structures related to them. Radiographic changes of the navicular bone can be difficult to interpret because many different architectural variations have been seen in both lame and sound horses.[44] The presence of increased radiotracer in the navicular bone region in the delayed phase is highly sensitive for the diagnosis of active navicular disease,[92] especially when solar views are obtained.[45] Remember that the navicular bone should not be seen as a separate entity on the normal foot; therefore, abnormal radiotracer in that region is indicative of active disease (Figure 3.167). Laminitis results in moderate to severe radiopharmaceutical uptake in the distal aspect of the distal phalanx, seen on both the lateral but especially the solar view (Figure 3.168). Other conditions, such as P3 osteitis, P3 fractures, and subsolar bruising, can also appear as areas of variably radiotracer uptake (Figures 3.169 and 3.170).



Figure 3.167. Delayed phase solar view of the left forelimb, showing a focal and intense radiotracer in the region of the navicular bone, consistent with navicular disease.



Figure 3.168. Delayed phase solar view of the right hindlimb, showing intense radiotracer along the distal portion of P3, compatible with laminitis.

A normal scintigraphic image does not show a discrete insertion of the DDFT on P3 and collateral ligaments of the distal interphalangeal (DIP) joint. However, the anatomical location of these structures can be identified, especially on solar views (Figure 3.171). If possible,

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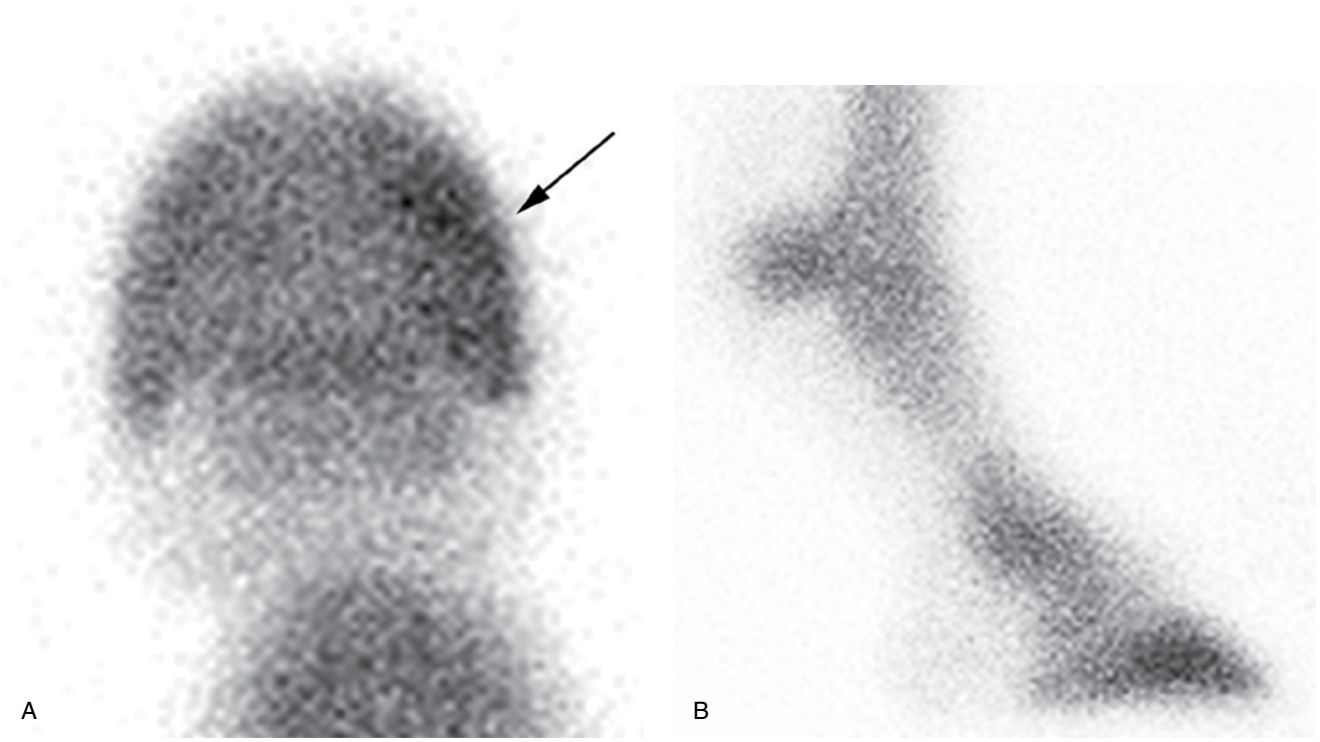


Figure 3.169. Delayed phase solar (A) and lateral (B) views of the distal limb of a horse with diffuse increased radiotracer along the medial aspect of the solar margin of P3 (arrow), compatible with pedal osteitis. *Source*: Courtesy of Dr. Erik Bergman.

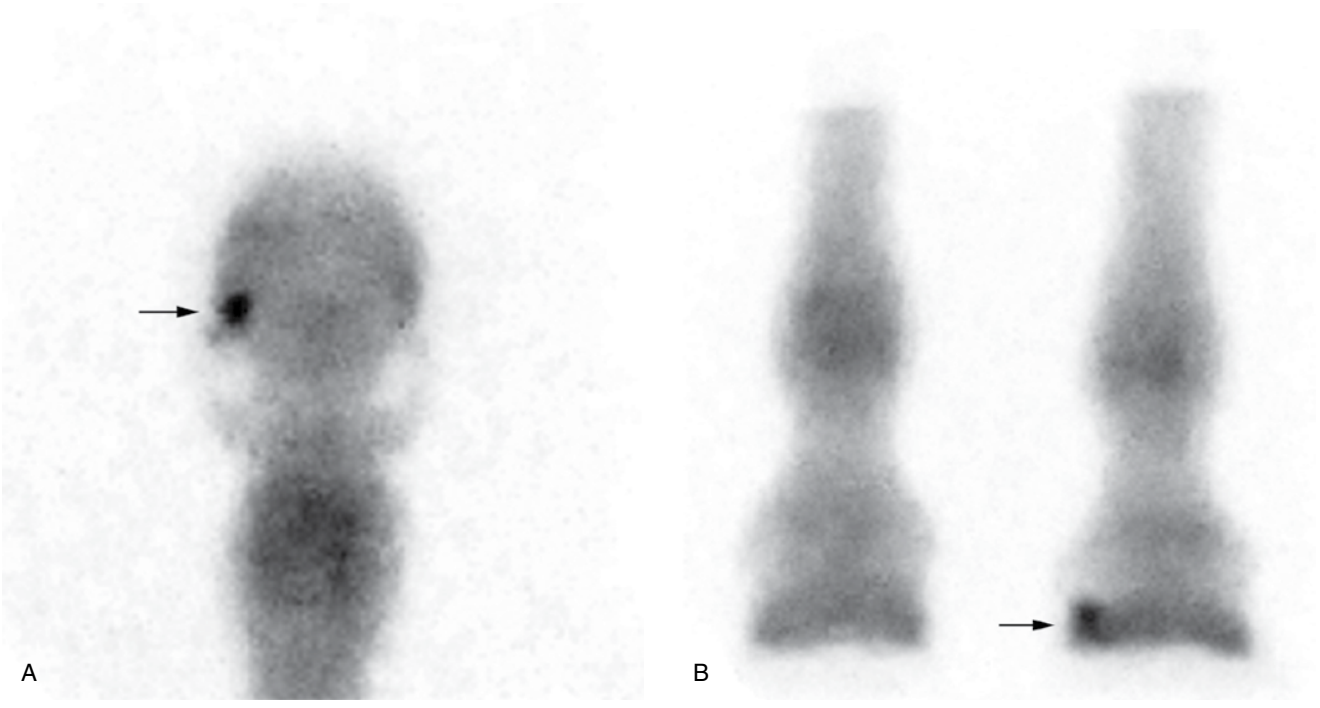


Figure 3.170. SHINE-processed, delayed phase solar (A) and dorsal (B) views of the left forefoot of a horse with a focal and intense radiotracer on the medial aspect of P3 (arrows) representing a palmar process fracture. *Source*: Courtesy of Dr. Kent Allen.

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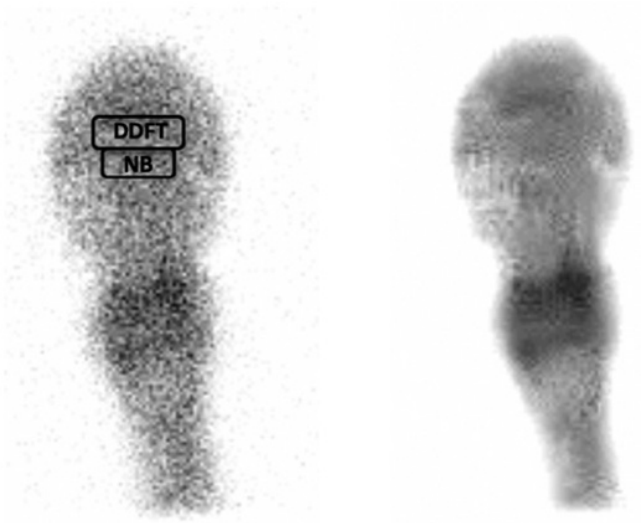


Figure 3.171. (A) Delayed phase solar view of a normal horse. Note the uniform uptake along the entire image. NB = navicular bone area and DDFT = deep digital flexor tendon area. (B) SHINE-processed image.

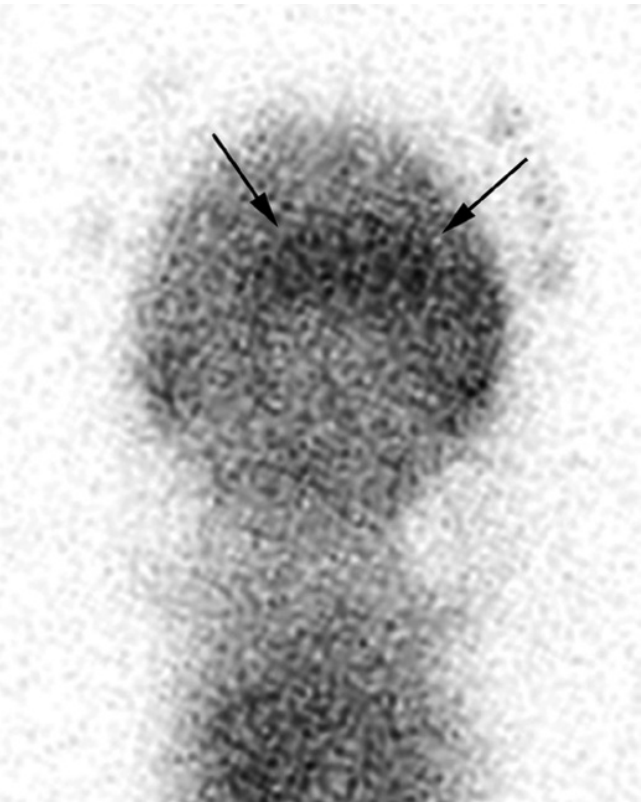


Figure 3.172. Delayed phase solar view of a horse with moderate diffused uptake in the region of insertion of the DDFT (arrows), suggesting enthesopathy. *Source*: Courtesy of Dr. Erik Bergman.

abnormal uptake at these sites should also be documented on the lateral and dorsal/palmar views. Areas of increased radiotracer in these regions in combination with a positive response to local anesthesia help to identify active pathologic changes (Figure 3.172). These changes could be further characterized with MRI.

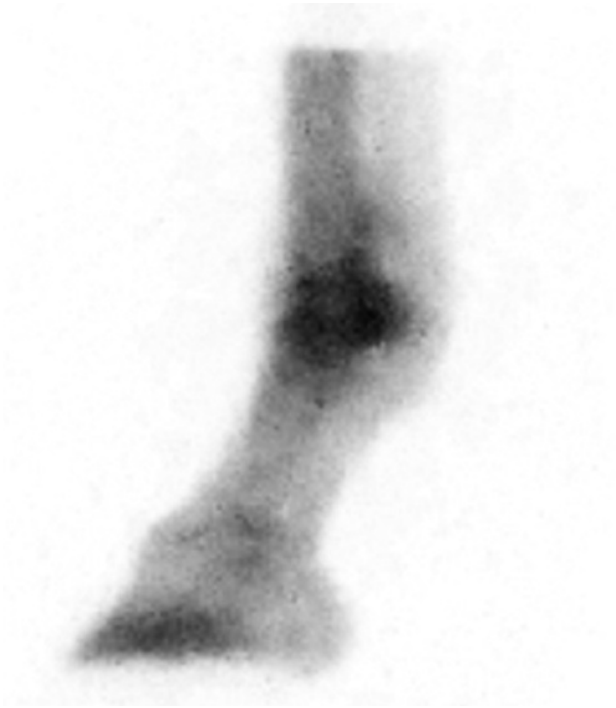


Figure 3.173. SHINE-processed delayed phase lateral view of the left fore distal limb of a horse showing diffuse radiotracer in the fetlock joint region, consistent with osteoarthritis. Note the difference in uptake between the abnormal fetlock and the normal proximal and distal interphalangeal joints. *Source*: Courtesy of Dr. Kent Allen.

OA of the DIP, proximal interphalangeal (PIP), and fetlocks joint can be identified when increased radiotracer is seen at these regions, typically areas along the dorsal aspect of the joint, when compared with the adjacent joints. There can also be relative increased radiotracer ratios in the opposite limb (Figure 3.173).

The proximal articular surface of the first phalanx is a common location for abnormal radiotracer that represents active injuries to the subchondral bone that in many instances are not detected radiographically and would benefit from additional studies such as computed tomography and MRI.[25,53,73] The proximal sesamoid bones are also susceptible to inflammatory changes that may appear as areas of increase radiotracer, which likely are related to stress from the different ligamentous attachments (Figure 3.174). Injuries to the axial border of the proximal sesamoid bones are usually, but not necessarily, traumatically induced, involving the intersesamoidean ligament, and can be the result of septic or nonseptic inflammation.[12,23,51,101]

Metacarpus and Metatarsus

Areas of increase radiotracer are often seen in the distal cannon bone of racing Thoroughbreds and Standardbreds. Both front limbs and hindlimbs can be affected, but Thoroughbreds more frequently show increase radiotracer in the distal metacarpus and Standardbreds in the distal metatarsus.[5,55] Stress remodeling and incomplete condylar fractures are common attributable pathologic changes for this increase uptake.[55,72,75]

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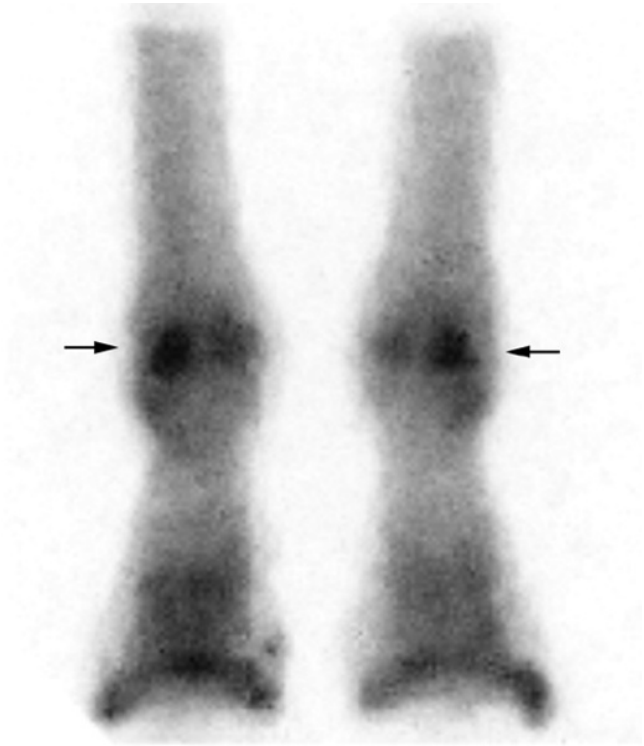


Figure 3.174. SHINE-processed delayed phase dorsal view of both front distal limbs of a horse with focal and intense radiotracer in the region of the lateral sesamoid bones (arrows), consistent with sesamoiditis. *Source*: Courtesy of Dr. Kent Allen.

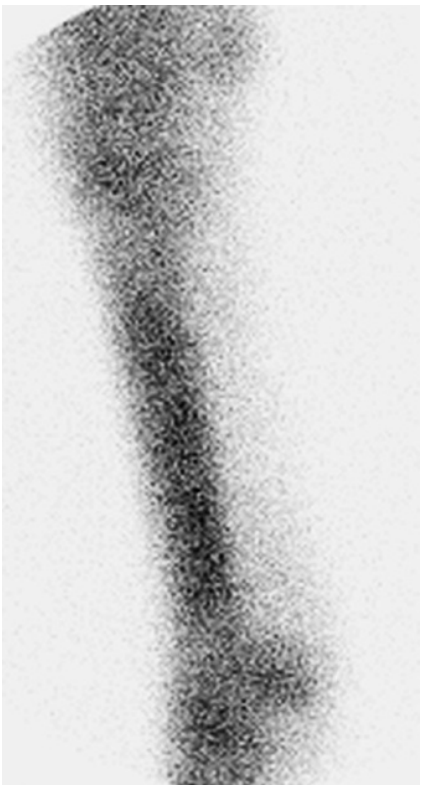


Figure 3.175. Delayed phase lateral view of the left metacarpus of a horse showing diffuse abnormal radiotracer along the diaphysis of MCIII, compatible with dorsal metacarpal disease ("buckshins"). *Source*: Courtesy of Dr. Erik Bergman.

Diffuse dorsal metacarpal increased radiotracer is common in racing horses and represents reactive periostitis or stress remodeling[47] (Figure 3.175). Focal and intense areas of radiotracer on the dorsal cortex of the cannon bone are likely indicative of a stress fracture (Figure 3.176).

Increased radiopharmaceutical uptake on the palmar or plantar proximal cannon bone is a common finding in cases of desmitis at the origin of the suspensory ligament that produces stress reaction or avulsion fracture on the bony attachment[28] (Figure 3.177).

Carpus

Exercise-induced bone remodeling is seen frequently on the third carpal bone, especially in Standardbreds,[30] but it may be seen in other racing breeds and Warmbloods. The increased radiotracer on the third carpal bone corresponds to sclerosis or fractures seen in radiographs (Figure 3.178).

Proximal Limb Long Bones

Stress fractures are common injuries seen on the tibial and humeral diaphyses in racing horses[62] that appear as a focal and intense areas of radiotracer located near a cortical margin. Humeral stress fractures are more common in Thoroughbreds than Standardbreds.[48] Common sites of tibial stress fractures in Thoroughbreds include the lateral cortex of the mid-diaphysis, caudal cortex of the proximal diaphysis, and less commonly the medial cortex of the distal diaphysis (Figure 3.179).[54,62] Stress fractures in the humerus occur at the cranio- and



Figure 3.176. Delayed phase dorsal view of the distal forelimbs of a horse with a focal and intense radiotracer on the mid-distal diaphysis of the right MCIII, consistent with a stress fracture or enostotic lesion.

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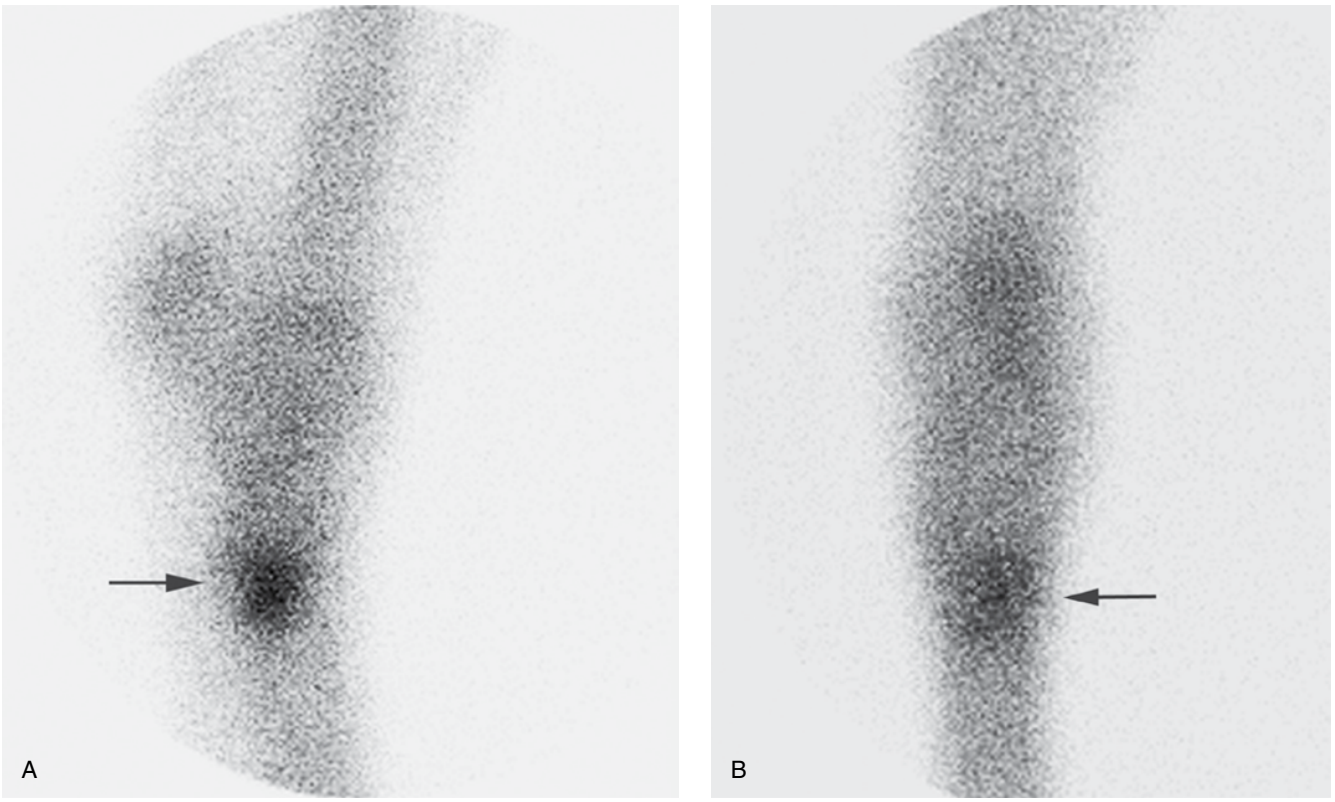


Figure 3.177. Delayed phase lateral (A) and plantar (B) views of the right tarsus of a horse, showing focal and intense radiotracer at the origin of the suspensory ligament on the proximal and plantar aspects of MTIII (arrows), compatible with an origin of the suspensory desmitis and enthesopathy. *Source*: Courtesy of Dr. Erik Bergman.

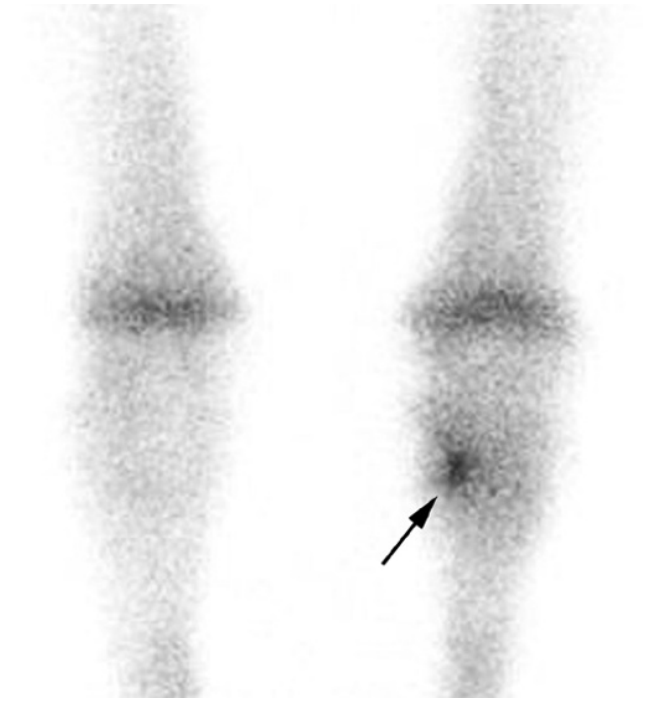


Figure 3.178. Delayed phase dorsal view of both carpi, showing a focal and intense radiotracer in the medial aspect of the left third carpal bone region (arrow), suggestive of sclerosis, chip fracture, or slab fracture. *Source:* Courtesy of Dr. Ryan Carpenter.

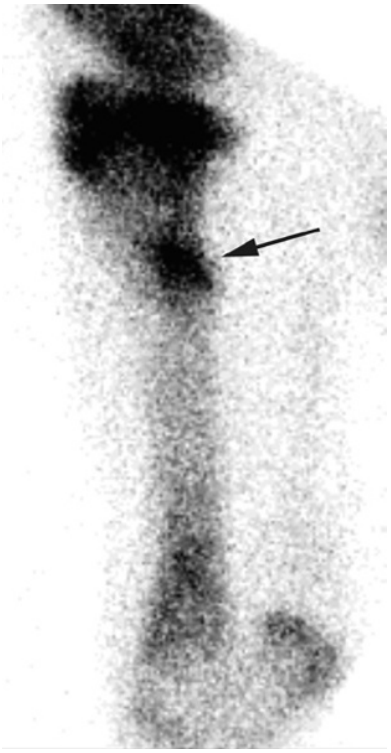


Figure 3.179. Delayed phase lateral view of the left tibia of a horse, showing a focal and intense radiotracer on the caudoproximal cortex of the tibia (arrow), consistent with a stress fracture.

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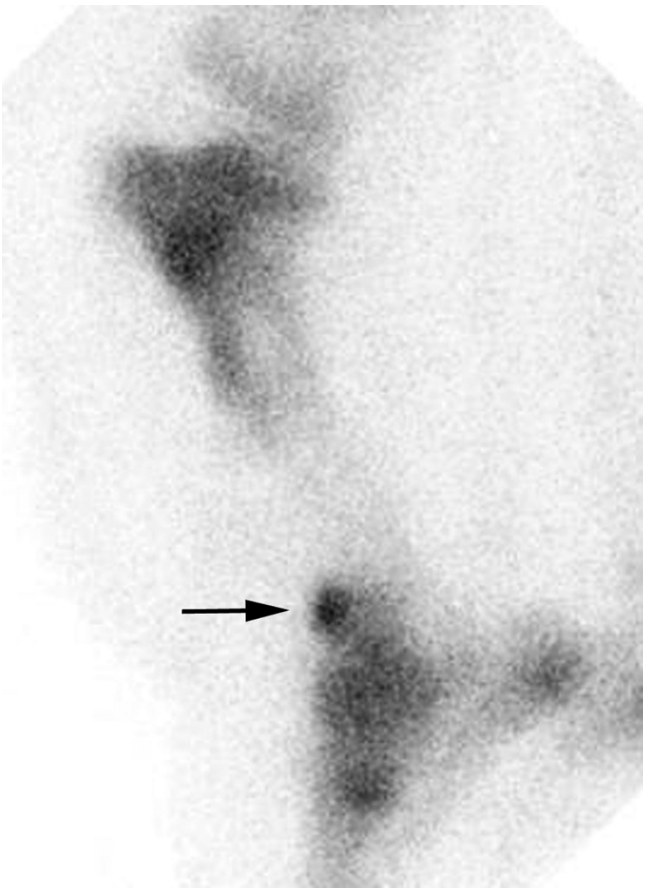


Figure 3.180. Delayed phase lateral view of the left humerus, showing a focal and intense radiotracer on the craniodistal humeral diaphysis (arrow), consistent with a stress fracture. Source: Courtesy of Dr. Ryan Carpenter.

caudodistal, caudoproximal, and less commonly craniproximal cortices (Figure 3.180).[62] Stress fractures of the radius are less common being reported to have a mid-diaphyseal occurrence.[52] Focal increase radiopharmaceutical uptake in the medullary cavity of long bones was first described as "bone infarcts"[68] and subsequently as enostosis-like lesions (Figure 3.181).[7,66] These lesions are frequently seen in the tibia, radius, humerus, and MCIII/metatarsal bones. Radiographically, the lesions appear as well-defined areas of increased opacity in the medullary cavities. It appears to be a transient condition with the lesions resolving in follow-up radiographic and scintigraphic examinations. The pathophysiology of these lesions is not well understood, and it has been reported that not all of the lesions are associated with lameness.[7,65]

Increased radiotracer on the caudal aspect of the distal femoral diaphysis just proximal to the stifle joint on a lateral view and localized laterally on the caudal view has been seen in cases of injury to the origin of the gastrocnemius muscle.[87]

Elbow

Ulnar fractures are relatively common, especially in young horses.[14] Other traumatic injuries such as Salter–Harris fractures and subluxations can also occur. These injuries are usually diagnosed clinically and radiographically. However, fractures can sometimes be very small or minimally displaced; in such cases scintigraphy may add useful information in the diagnosis. Osteomyelitis secondary to trauma to the proximal radius with possible progression to septic elbow arthritis has been documented as a large and intense area of radiotracer in that region.[86] Radiographically, these lesions may be subtle or not apparent if the infection has not advanced enough to cause substantial bone lysis.

Subchondral bone cysts and cartilage lesions may not show scintigraphic abnormalities unless the underlying subchondral bone is affected.

Shoulder and Scapula

Injuries to the shoulder and scapula in the horse are uncommon. Fractures of the greater tubercle may occur, but frequently the diagnosis is based on clinical evaluation in combination with radiography or ultrasonography.[57,94] An area of increased radiotracer in the cranioproximal humerus may represent a fracture of the tubercle(s) or extension of an infectious/inflammatory process related to the bicipital bursa (Figure 3.182). An unusual case of abnormal radiotracer in the cranioproximal humerus corresponding to a cyst-like lesion of the intermediate tubercle in absence of bicipital bursitis was reported.[67] In general, subchondral bone cysts and small cartilage defects are lesions that are not detected scintigraphically unless there are inflammatory changes extending to the adjacent subchondral bone.

The supraglenoid tubercle is the most frequent location for a traumatic fracture of the scapula.[19,20] Complete fractures of the neck and body of the scapula have also been reported sporadically.[19,20] The scintigraphic diagnosis of stress fractures of the scapula has been documented in a report of two cases in which the location of the focal and intense radiotracer corresponding to the fractures was located in the caudal midbody and the ventral aspect of the neck, respectively.[15]

Tarsus

Distal tarsal radiotracer is usually seen in jumping horses, followed by racing Standardbreds and less commonly Thoroughbreds.[5,29,30] The increased radiotracer can be unilateral or bilateral; a distribution along the entire area of the distal tarsal region of the dorsal aspect is the most common pattern.[60] Increased radiopharmaceutical uptake in the distal tarsal region corresponds to OA or degenerative changes that may or may not be radiographically apparent (Figure 3.183). Talocalcaneal OA is rare but has also been documented as focal and intense radiotracer predominantly medially and more plantar and proximal than distal tarsal radiotracer (Figure 3.184).[77]

Scintigraphy is very useful in localizing fractures of the central and third tarsal bones, especially when the fracture is not displaced, hence radiographically occult.[84] These horses have a history of acute onset of lameness, usually following exercise. The fracture will appear as a focal and intense region of radiotracer in the mid to distal tarsal region. Incomplete sagittal fracture of the talus

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Figure 3.181. (A) Delayed phase lateral view of the left tibia of a horse, showing a focal and intense radiotracer in the medullary cavity of the proximal diaphysis, suggestive of enostosis-like lesion. Radiographic (B) and computed tomographic (C) images showing the presence of ill-defined, irregular medullary sclerosis, compatible with an enostosis-like lesion. *Source*: Courtesy of Dr. Erik Bergman.

is a rare condition in horses and can be a diagnostic challenge because the patient can present without evidence of soft tissue swelling or tarsocrural joint effusion.[16]

A focal and moderate to intense area of radiotracer at the level of the proximal talus should lend suspicion to a sagittal fracture, which is not always evident on radiographs.[16] A similar pattern of abnormal radiotracer was also reported on a 2-year-old Thoroughbred with unusual osteochondral lesions at the proximal articular margin of the medial trochlear ridge of the talus.[76] Osseous cyst-like lesions in the tarsus have been described as focal areas of abnormal radiotracer in the distal tibia (medial or lateral malleoli or intermediate sagittal ridge) or talus.[36] Adding a flexed lateral view to the routine scintigraphic exam protocol of the tarsus can help to differentiate whether the abnormal radiotracer is located in the distal tibia or the talus.

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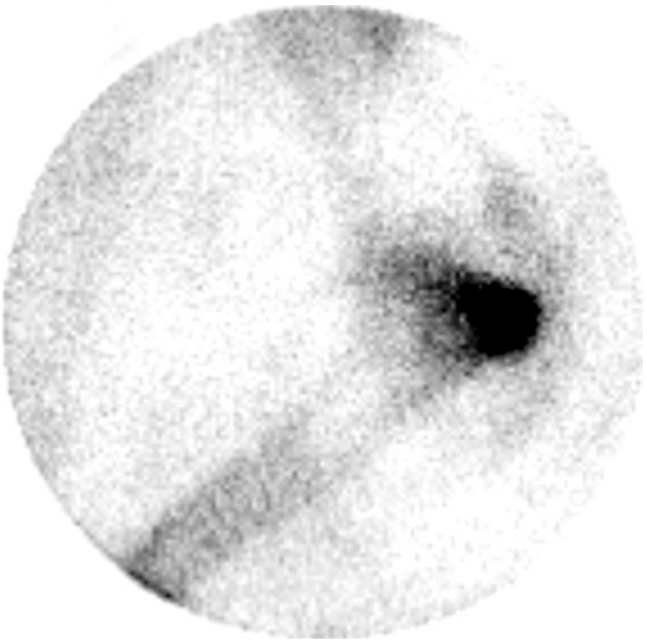


Figure 3.182. Delayed phase lateral view of the right humerus, showing focal and intense abnormal radiotracer in the greater tubercle, caused by a suppurative process.

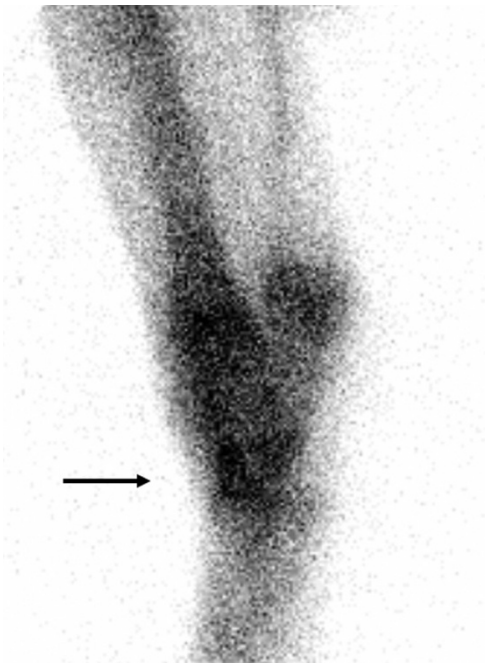


Figure 3.183. Delayed phase lateral view of the left tarsus of a horse, showing focal areas of abnormal radiotracer on the dorsal aspect of the distal intertarsal and tarsometatarsal joints (arrow), consistent with osteoarthritis.

Stifle

Medial femoral condyle subchondral bone cysts are one of the most common pathologic changes seen in the stifle joint of horses. However, as previously mentioned, these lesions do not appear as areas of abnormal radiotracer unless an osteoblastic response or juxta-cyst osteosclerosis occurs in the adjacent subchondral bone (Figure 3.185). A case of a yearling horse with radiographically evident bilateral enlarging subchondral bone cysts after surgical debridement was reported to have normal postoperative follow-up scintigraphic examinations, despite the persistence of lameness localized to the stifle joints.[79] It was proposed that the lesions were mostly osteoclastic; hence no abnormal radiotracer was observed. Subchondral cystic lesions can also appear in the proximal tibia secondary to osteochondrosis or as a manifestation of OA, which scintigraphically may exhibit focal abnormal radiotracer.[89]

Ligamentous/tendinous avulsions may be suspected when a localized area of abnormal radiotracer is seen at an enthesis during the delayed phase. In these cases, other approaches such as ultrasound, MRI, or arthroscopy should be considered for better evaluation of the soft tissues.

Pelvis, SI, and Coxofemoral Joints

Complete, stress, and avulsion fractures at different anatomical locations in the pelvic region have been documented as areas of abnormal radiotracer in delayed phase scintigraphy.[11,13,38,64] Injuries to superficial structures such as the tuber coxae, ischial tuberosity, dorsal sacrum/tail head, greater trochanter, and third trochanter can be the result of direct trauma, often blunt in nature, from a kick, trailer accident, or self-inflicted as can occur from rapid movement or falling accidents. Fractures of the tuber coxae and tuber ischium can be detected by the intense abnormal radiotracer (Figure 3.186) and the possible asymmetric location of the radiotracer between affected and unaffected sides in cases of displaced fractures. Decreased uptake of a fractured tuber coxae has been associated with a ventrally displaced fracture fragment.[13] Oblique views of the pelvis are very useful for evaluating the iliac wing in suspected cases of stress fractures.[42] Trochanteric bursitis is more common in Standardbred racehorses, usually as a result of trauma, either direct or related to stress from racing.[56] Most of the time the lesion involves soft tissue structures, but in some cases the subchondral bone and cartilage overlying the greater trochanter are injured, and a focal abnormal radiotracer is seen over the greater trochanter (Figure 3.187).

Coxofemoral joint pathology is very difficult to assess with dorsal views due to the great γ-ray attenuation from the superimposed musculature. Forty-five degree oblique views are very useful in cases of suspected hip pathology (Figure 3.188).[13]

The SI region is also susceptible to pathologic change commonly attributed to regional SI desmopathy or arthropathy. Injuries to the sacral tuberosity at the origin of the SI ligaments have been described as abnormal radiotracer at the affected enthesis[25] possibly with the tuberosity being displaced cranially.[13] The SI joint can be evaluated on the dorsal oblique view and the straight dorsal view.[32] The straight dorsal view is better to compare left and right on the same image; however, the oblique view gives a better evaluation of the extent of

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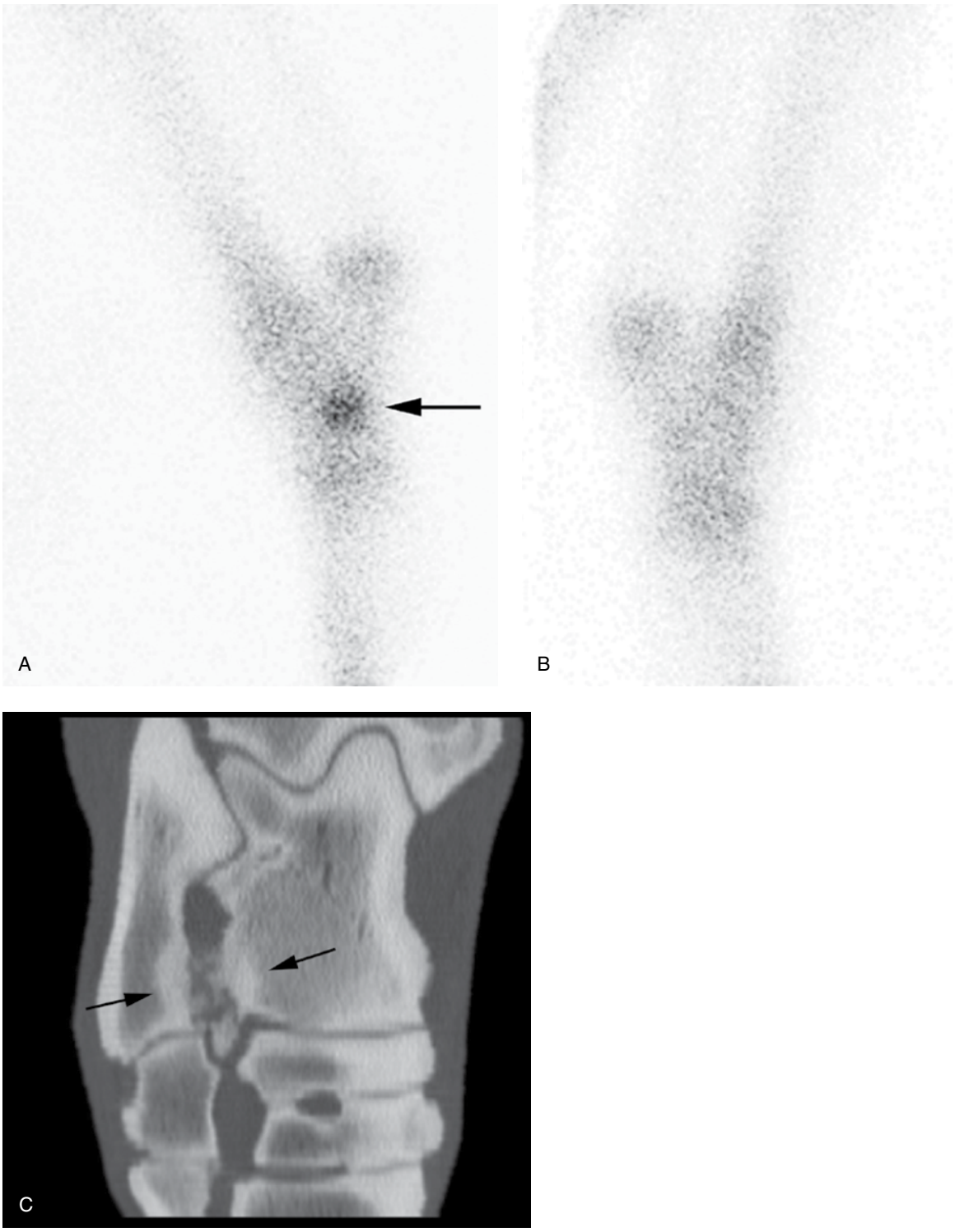


Figure 3.184. Delayed phase lateral views of the left (A) and right (B) tarsi. Note the focal and intense abnormal radiotracer seen in the region of the talocalcaneal joint on the left tarsus (arrow), compatible with osteoarthritis. Compare with the uniform uptake of the normal right tarsus. Computed tomographic image (C) of the left tarsus confirms the osteoarthritis with evidence of irregular bone margins with adjacent sclerosis and soft tissue mineralization of the intertarsal region (arrows). *Source:* Courtesy of Dr. Sarah M. Puchalski.

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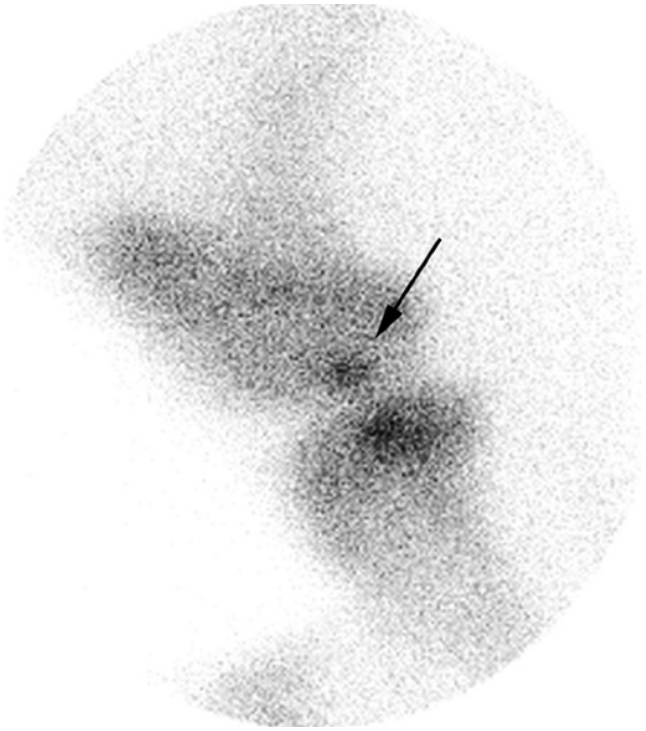


Figure 3.185. Delayed phase lateral view of the left stifle joint of a horse, showing a focal increased radiotracer at the articular surface of the condyles (arrow), suggestive of a bone cyst with reactive adjacent subchondral bone. *Source*: Courtesy of Dr. Erik Bergman.

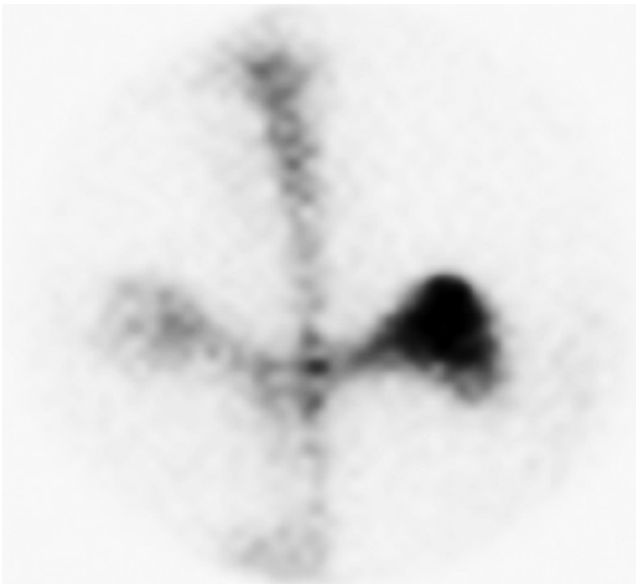
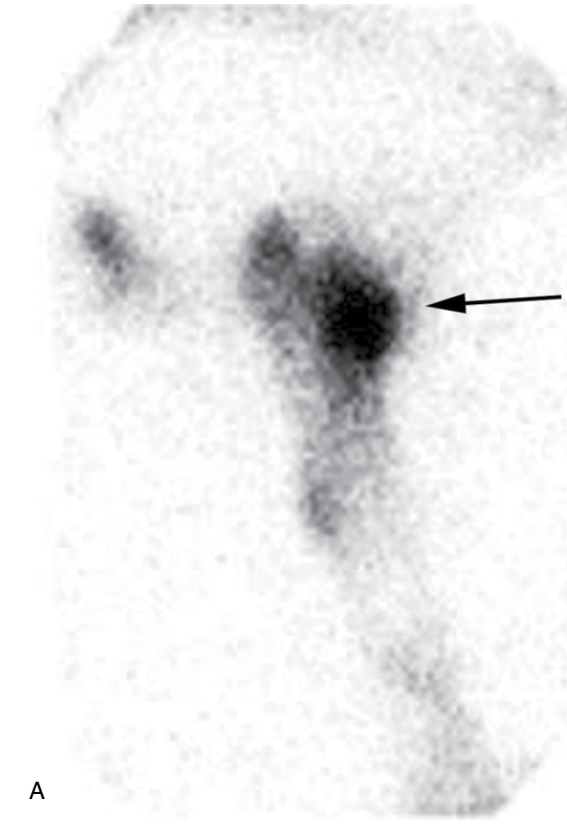
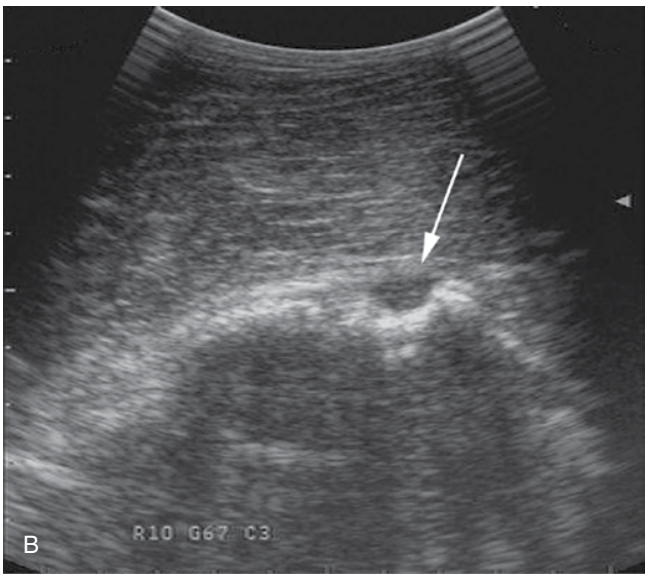


Figure 3.186. Delayed phase tail on detector (TOD) view of the pelvis of a horse, showing focal and intense increased radiotracer on the right ischial tuberosity, compatible with a fracture. *Source:* Courtesy of Dr. Erik Bergman.



A



B

Figure 3.187. (A) Delayed phase lateral view of the right hip joint of a horse, showing focal and intense increased radiotracer in the region of the right greater trochanter (arrow), compatible with trauma and gluteal muscle (medial or deep) enthesopathy, aseptic trochanteric bursitis, or a suppurative process. (B) Transverse ultrasound image of the greater trochanter seen in (A), showing a mildly distended trochanteric bursa (arrow). Ultrasound-guided local anesthetic injection resulted in significant lameness improvement.

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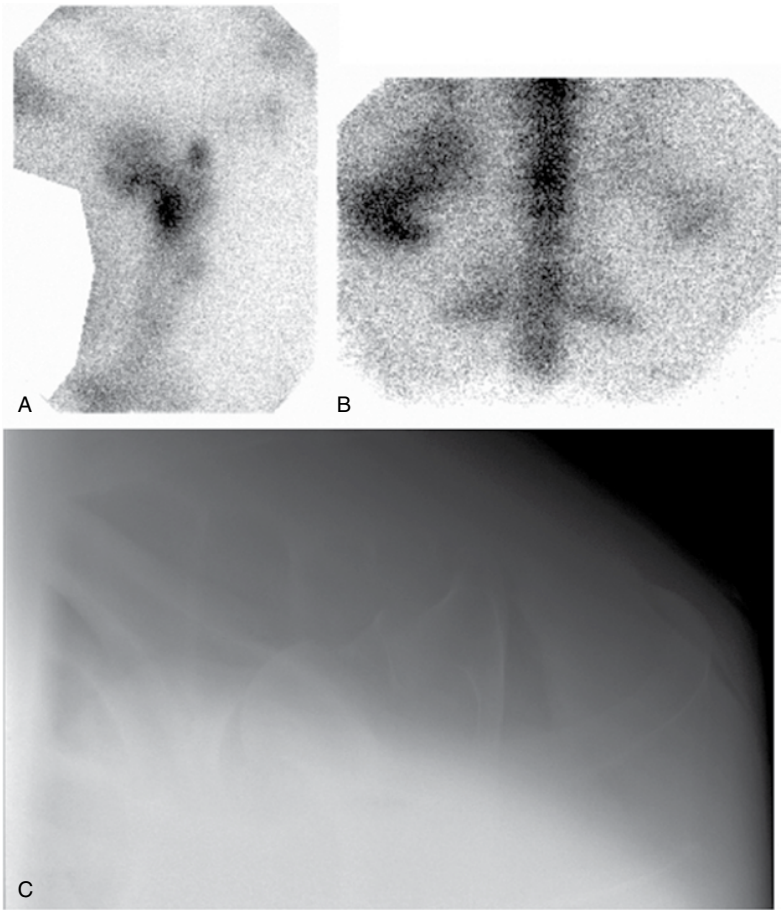


Figure 3.188. Delayed phase (A) left lateral and (B) tail on detector views of the left hip joint showing diffuse intense abnormal radiotracer in the region of the joint. The horse was subsequently diagnosed with subluxation of this joint on radiography (C).

joint surface because it appears wider and in some cases helps to avoid superimposition of the urinary bladder. Areas of abnormal radiotracer at the SI joint region are often correlated with SI joint pain/injury (Figure 3.189).[22,93] However, increased radiotracer in the SI region has also been found in normal horses and those in which the lameness was attributed to a different anatomical region.[13,25] Variations on the anatomical conformation of the sacral wings and the cranial sacral borders may also play a role in the pattern of radiopharmaceutical uptake.[39] Areas of abnormal or increased radiotracer in the SI region should be interpreted with caution, and the diagnosis of pathology should not be based on the scintigraphic findings alone because of the lack of a "gold standard" and the possibility of false-positive results.[3,25]

In general, the results of pelvic scintigraphic evaluations should be strongly correlated with clinical and physical exam findings and supported, as frequently as possible, with ultrasound or radiography and/or local anesthesia.

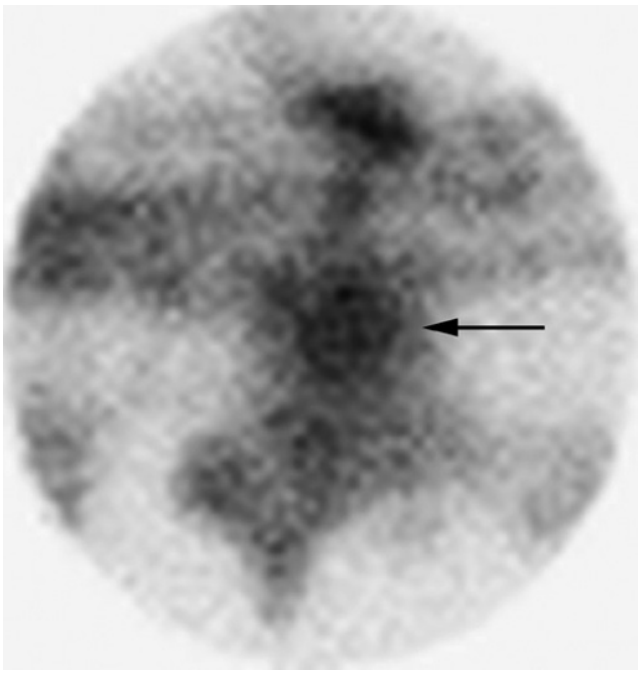


Figure 3.189. Delayed phase LDO of the left sacroiliac region of a horse, showing marked and diffuse increased radiotracer in the sacroiliac joint (arrow), compatible with osteoarthritis. *Source:* Courtesy of Dr. Erik Bergman.

Spine

Degenerative changes in the spine are most commonly associated with impingement of the dorsal spinous processes (kissing spines), as well as OA of the articular facets. Impingement of the dorsal spinous process is common on the thoracic and lumbar spine. Good-quality radiographs of these regions, especially

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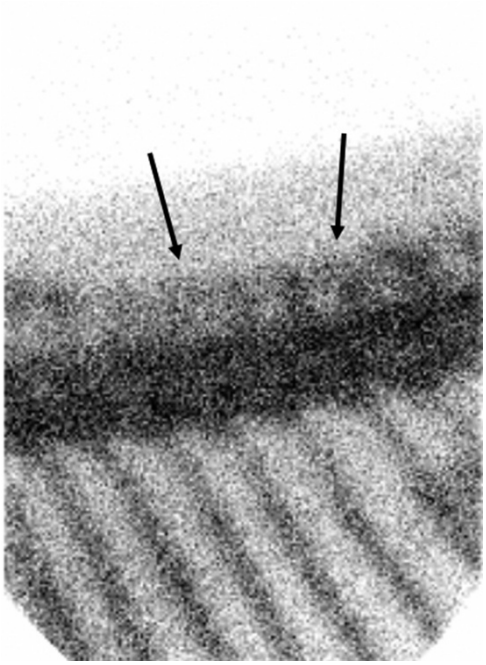


Figure 3.190. Delayed phase LDO view of the mid-thoracic vertebrae of a normal horse. Note the relative increased uptake of the vertebral bodies when compared with the corresponding dorsal spinous processes (arrows).

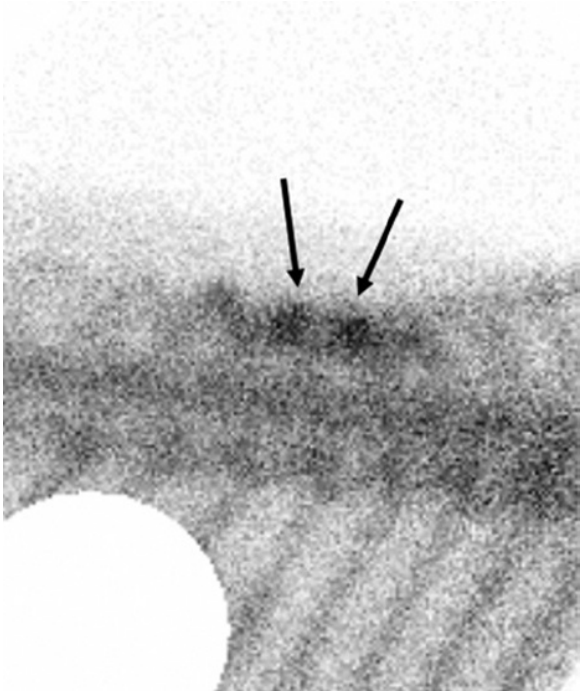


Figure 3.191. Delayed phase LDO view of the mid-thoracic vertebrae of a horse, showing focal and intense abnormal radiotracer at least two adjacent dorsal spinous processes (arrows), suggestive of impingement.

the lumbar spine, can be very difficult to obtain because of the large amount of musculature causing considerable scatter radiation and because if the technique is increased, the most dorsal aspect of the dorsal spinous processes can be overexposed. This makes the scintigraphic evaluation of the spine an invaluable tool for evaluation of the back (Figure 3.190). LDO and RDO views of the spine are the best, although straight lateral and dorsal views are made if a lesion is found. Areas of mild abnormal radiotracer at the dorsal aspect of the spinous processes can be seen in horses with no clinical signs of back problems; therefore, these findings should be interpreted with caution.[33,34] Focal and intense areas of radiotracer can be seen with fractures, severe impingement with or without ligamentous damage, osteomyelitis, or neoplasia (Figures 3.191 and 3.192). Diagnostic differentials should be made, taking into consideration history, physical/clinical exam, and blood analysis.

Increased radiotracer in the cervical articular facets is most commonly seen in cases of OA. The increased radiotracer can be identified when the bone activity in the region of the articular facets is greater than in the corresponding vertebral body or adjacent joints. Affected cervical vertebral facets may be unilateral or bilateral, and more than one joint may be affected (Figure 3.193). Therefore, left and right views should be obtained, and the practitioner should be aware of the possibility of multiple adjacent sites affected and not rely only on the comparison of adjacent joints for increased radiotracer in the evaluation.

Increased radiotracer in the vertebral bodies is uncommon and has been reported with spondylosis deformans[17] and cases of discospondylitis.[40,85]

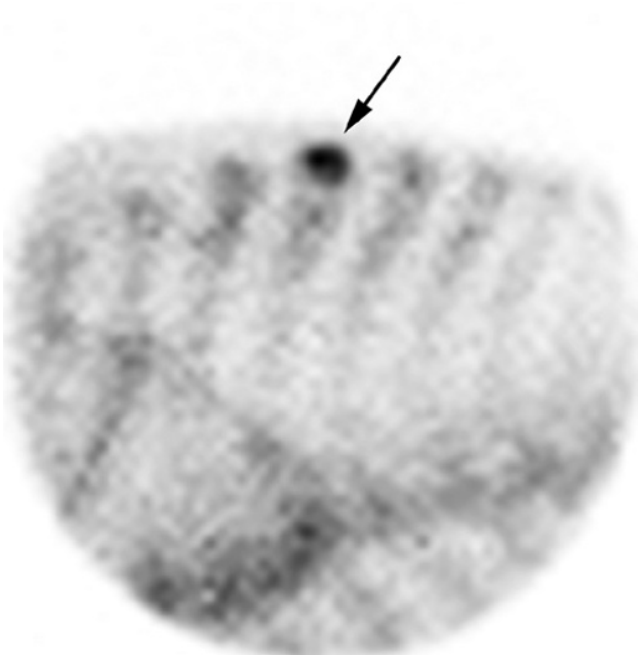


Figure 3.192. Delayed phase LDO view of the withers of a horse, showing a focal and intense abnormal radiotracer involving only the most proximal aspect of one of the spinous processes (arrow), suggestive of a fracture. *Source*: Courtesy of Dr. Erik Bergman.

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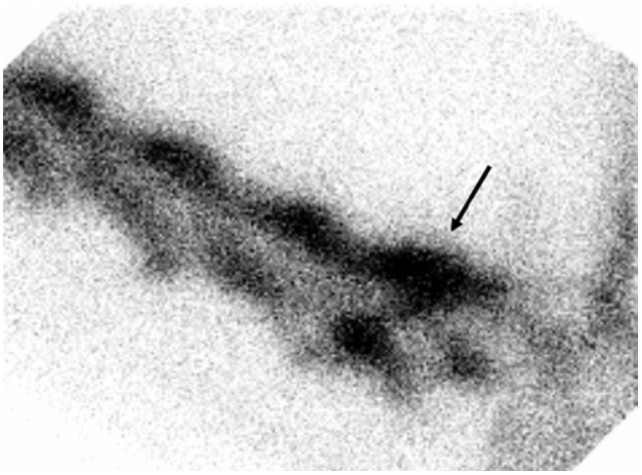


Figure 3.193. Delayed phase left lateral view of the caudal cervical region of a horse, showing marked, focal, and intense abnormal radiotracer at the articular facets of C6–C7 (arrow), compatible with osteoarthritis.

LIMITATIONS OF NUCLEAR MEDICINE

As mentioned previously, nuclear medicine traces physiologic processes in the different body systems and therefore has a high sensitivity in detecting early changes in the metabolism. This is the main reason why nuclear scintigraphy of the musculoskeletal system is an invaluable diagnostic tool in equine lameness. However, the specificity of bone scintigraphy is very low in the majority of cases. During the evaluation of the bone scintigraphy results, the practitioner should be aware that an area of increased radiotracer only means that there is an area of increased osteoblastic activity in a particular region. If the increased radiotracer is considered pathologic, in most situations, a list of differential diagnoses should be made and further imaging performed to gain a better idea of the anatomical changes of the affected region.

In general, bone scintigraphy is used to localize an area of abnormal bone metabolism that may explain the source of lameness and not necessarily the specific pathologic change. There are some cases in which the diagnosis can be made from the bone scintigraphy results. For example, one can diagnose a stress fracture in a sound racehorse that becomes lame immediately after a training session or a race and has a focal increased radiotracer in the diaphysis of the tibia. On the other hand, if a similar area of increased radiotracer is found in a retired older horse that spends most of the time in the pasture, other differential diagnoses should be considered, such as blunt trauma with or without a fracture, enostosis-like lesion, osteomyelitis, or neoplasia.

False-negative results occur secondary to many different reasons and can be considered as a limitation of bone scintigraphy. For example, a bone lesion with minimum uptake in the proximal region of a limb may not be apparent due to the significant γ-ray attenuation produced by the surrounding musculature. As in cases of subchondral bone cysts, cartilage or meniscal injuries will likely give a false-negative result if the adjacent bone is not affected.

In summary, the practitioner should be aware of the potential limitations when interpreting the results of a nuclear scintigraphic examination; correlate them with the signalment, history, clinical signs, and physical/lameness exam findings; and consider further diagnostics when appropriate.

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COMPUTED TOMOGRAPHY

Mathieu Spriet

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INTRODUCTION

Computed tomography (CT) is a cross-sectional imaging technique, relying similarly to radiography, on differential X-ray attenuation by the tissues being imaged. Images are acquired as slices of the anatomy of the imaged subject, providing three-dimensional (3D) information. These slices of anatomy are obtained by rotating an X-ray generator around the imaged body area and recording attenuation of the X-ray beam at multiple different angles. The attenuation data are then digitally processed to reconstruct an image.

The imaging principles, relying on X-ray attenuation, are similar to radiography. CT is excellent at imaging bones, with dense bones appearing white (hyperattenuating), whereas gas will show up black due to a lack of X-ray attenuation. Soft tissue structures will display an intermediate (gray) opacity. Based on calibration of the attenuation data, CT is better than radiography at identifying small differences in tissue density. For this reason, tendon, synovial fluid, and connective tissue can be differentiated on CT, while they typically have the same opacity on radiographs. CT also presents the advantage over radiographs to eliminate superimposition of structures. The combination of the increased contrast resolution and abolition of superimposition explain why CT detects lesion not recognized on radiographs.

CT has been used in the horse since the late 1990s, mostly for head and distal limb imaging. Due to technological progress, CT cost and availability have greatly improved in the recent years. Also modification of the size and configuration of the scanners have led to the development of new clinical applications, such as imaging of the neck and more proximal areas of the limbs.

In this chapter, the basic principles of CT will be described along with the different systems that are available. The clinical applications of CT will be limited to musculoskeletal imaging due to the focus of this publication. Imaging of the equine head, although a major indication for CT imaging, will not be covered.

EQUIPMENT AND PRINCIPLES OF CT

Classic CT scanners consist of a gantry, containing a rotating X-ray generator and an array of detectors, and a table translating the patient through the gantry for image acquisition. Obviously, the standard human table needs to be adapted to the size and weight of the equine patient. This is typically accomplished by adding a large table top over the human table but presents a technological challenge as the patient needs to be moved precisely through the gantry during image acquisition. A few commercial solutions are available, but many systems rely on custom-made tables. An interesting variation in the scanner design is the configuration of a few systems having a translating gantry. In these systems, the patient remains stationary during image acquisition as the gantry itself travels over the area to be imaged. The horse can be positioned on a standard equine table, simplifying the logistics.

In addition to the table design, the gantry diameter is a limitation to the range of body areas that can be imaged. Most classic human gantries have a diameter of 55–75 cm, limiting applications in horses to the distal limbs and head to cranial neck (typically to the level of the third or fourth cervical vertebrae). Larger gantries have been designed for bariatric human patients with a diameter up to 90 cm. These systems provide the ability to image larger body areas such as the equine caudal neck and stifles. Most recently scanners designed for intraoperative use in human neurosurgery even offer larger gantries, with a diameter over 100 cm.

The recent development of cone-beam CT technology brought versatility in the design of scanners. Cone-beam scanners use a flat panel detector, similar to a digital radiography plate. This allows modification of the configuration of the classic gantry with asymmetric shape and larger aperture. A robotic arm CT system has also been developed: the classic gantry has been replaced by two robotic arms, one carrying the X-ray generator and one the detector plate. This potentially provides more versatility for scanning standing horses and imaging larger areas. Cone-beam CT provides an excellent spatial resolution, but limitations exist regarding contrast resolution and sensitivity to motion.

As an X-ray-based imaging technique, the generator current intensity (mA) and voltage (kV) are important technical parameters for CT similar to radiography. Additional parameters, including slice thickness, reconstruction algorithm, field of view, and matrix size, are also important to understand the optimization of CT image quality. Depending on the type of tissue imaged, the technique will be different.

In order to optimize images for bone, it is important to have a high spatial resolution and sharp edges. This is typically achieved with using thin slices, an edge enhancement algorithm, a small field of view, and a large matrix (Figure 3.194). The slice thickness in standard multislice CT is typically less than 1 mm, classically 0.65 mm. The in-plane spatial resolution is governed by the matrix size and the reconstruction field of view. Most CT images will use a 512 × 512 matrix. The pixel size is obtained by dividing the field of view by the matrix size. This is important to keep in mind as most classic CT scanners can image a 50-cm field of view; however in the configuration the pixel size will be ~1 mm (500/512). Reducing the field of view to 25 cm, which is usually enough to cover the limb anatomy to image, permits bringing the pixel size to 0.5 mm (250/512), doubling the in-plane spatial resolution. The edge enhancement algorithm is a filter that is applied to the acquired data to help make the image edges more

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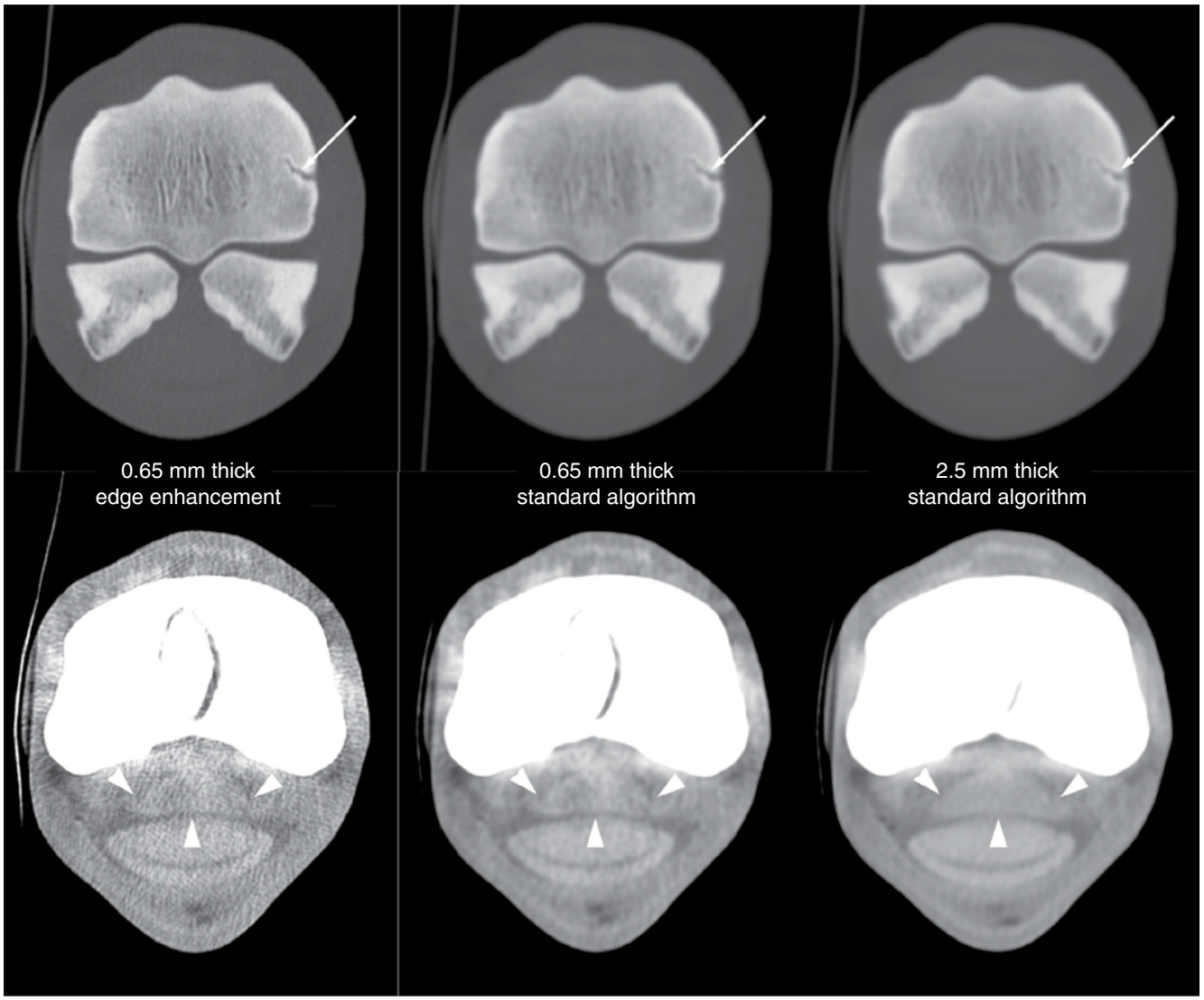


Figure 3.194. Effect of slice thickness, algorithm, and window on CT image appearance.Transverse CT images through the distal third metacarpal bone (top row) and the proximal aspect of the proximal phalanx (bottom row). The top row is displayed in a bone window (window level [WL] 600, window width [WW] 2,600) and the bottom row in a soft tissue window (WL 40, WW 350). Notice how bone detail can be appreciated in the top row, whereas the bone appears uniformly white on the bottom row. Inversely tendons and ligaments can be appreciated in the bottom row but appear uniformly gray on the top row. All the images were acquired at the same time but were reconstructed with different slice thickness and algorithms as displayed in the figure. The 0.65-mm-thick slice with edge enhancement is best at depicting fine bone detail such as small vascular canal (arrow, top left). The ligamentous structures are best defined using a 2.5-mm-thick slice with a standard algorithm as illustrated by the appearance of the straight distal sesamoidean ligament (arrow heads) in the bottom right image.

conspicuous. This gives a sharper look to the image and facilitates the identification of fracture lines or small osteophytes.

The approach to soft tissue imaging is different. Although high spatial resolution and sharp edges generally seem appealing in terms of image quality, they come with the downside of creating a "noisy" image. Signal-to-noise ratio is an important characteristic of an image. It corresponds to the amount of information in a specific voxel that relates to the actual property of the tissue versus the amount of information that is randomly generated. In a more practical way, in an image with a high signal-to-noise ratio, pixel representing the same tissue will display the same shade of gray. However, if the signal-to-noise ratio is low ("noisy image"), two pixels representing the same tissue might display a different shade of gray. This leads to a heterogeneous, grainy image, and subtle changes in signal intensity cannot be recognized. For bone imaging, the key is the high spatial resolution to identify small changes at the surface of the bone, and the lower signal-to-noise ratio is not an issue as a lot of natural contrast is present in the image to define the margins of the bone. For soft tissue imaging, however, the emphasis needs to be placed on contrast resolution, rather than spatial resolution. The difference between normal and abnormal tissues results in only a slight difference in attenuation of the X-rays. It is important to decrease the noise to be able to recognize these. A standard or "soft" algorithm will avoid the noise created by the edge enhancement. Also using a larger slice

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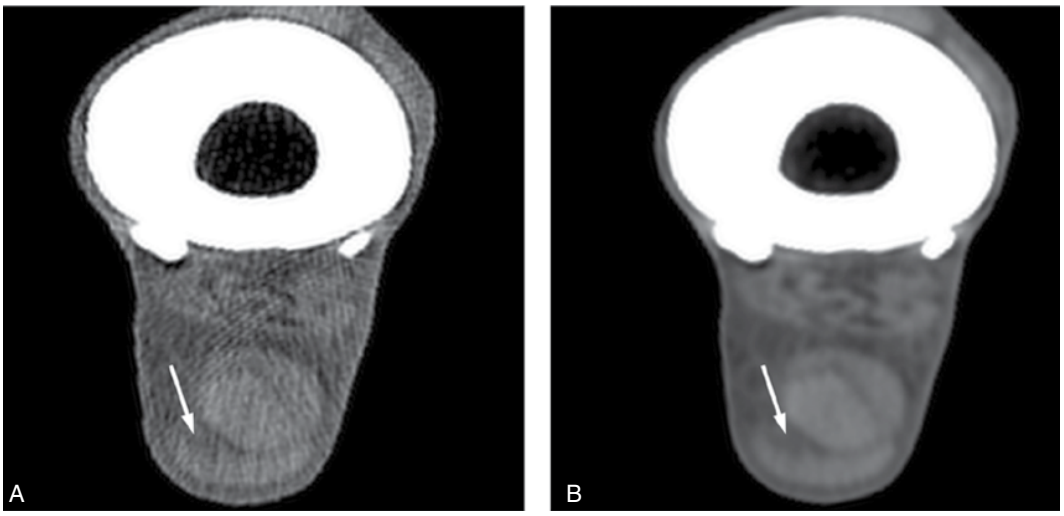


Figure 3.195. Effect of algorithm and slice thickness on lesion detection.Transverse CT images through the mid-level of the left metacarpus of a 15-year-old Warmblood gelding. Medial is to the left. Both images are displayed in a soft tissue window (WL 100, WW 300). The image on the left was reconstructed as 0.65-mm-thick slice with edge enhancement, whereas the image on the right has a 2.5-mm thickness and a standard algorithm. Notice the difference in visualization of a lesion at the dorsomedial border of the superficial digital flexor tendon (arrow). The noise present in the image on the left compromises the recognition of the lesion.

thickness leads to a larger voxel, which also improves the signal-to-noise ratio. Our preferred image technique for tendon assessment is a 2.5 mm thick in a standard algorithm (Figures 3.194 and 3.195).

These specific details of image acquisition suggest that different images are needed to assess both bone and soft tissue. The good news however is that a lot of these differences are part of the "post-processing" of the images. This means that different images can be obtained from a single acquisition, avoiding the need to perform multiple scans and decreasing radiation exposure. The reconstruction algorithm can be modified post acquisition. Multislice CT scanners also allow modification of the slice thickness post acquisition.

In addition to the acquisition parameters, understanding the viewing parameters, mainly the "window," is important. Each pixel in the image is attributed a CT value measured in Hounsfield units (HU). CT scanners are calibrated so that pure water has a HU equal to 0. Soft tissue structures typically range between 30 and 100 HU, fat is negative (from –300 to –20), air is about –1,000, and bone varies from 300 to 2000. When the image is displayed on a viewing device, the operator can choose how the gray scale of the viewing device will represent the different HU. An option can be to display the entire range, with the lower HU being attributed the darker shade of gray and the higher HU the lighter one; however in this configuration, tissues with close HU values will not be distinguished from each other. It can be advantageous to choose to spread the gray scale over a limited part of the range of HU. This is where the concept of "window" comes into play. A window is defined by a width and a level expressed in HU. If one is interested in assessing the soft tissue, since the HU of interest will be limited to a short range in the lower positive values, a classic window would have a level of 40 and a width of 350. This means that the gray scale will be spread between HU values of –135 and +215 (40 – 350/2; 40 + 350/2). Any HU lower than –135 will show up as black; any HU higher than +215 will appear white. The advantage of this window is that an HU of 0 will have a shade of gray visibly different than an HU of 50. On the other hand, in order to look at bone structures, where a large range of high HU exist, there is a need to use a higher level and a larger width. A level of 600 with a width of 2,600 is a good example. In this scenario however, a difference of 50 HU might not be visible (Figure 3.194).

CT images can simply be displayed the way they are acquired, as transverse images through the body area being imaged, but most viewing software applications offer the ability to reformat the images in different planes. The multiplanar reformatting tool (MPR) typically offers the ability to navigate the data set in three orthogonal planes (Figure 3.196). This is a very useful display format to understand the 3D appearance of lesions. 3D volume rendering is another technique that can be useful for 3D assessment of osseous structures. It is a popular viewing system for surgical planning of complex fracture repair (Figure 3.196). 3D rendering is however of limited value for soft tissue imaging due to the need for high contrast between the different structures for proper 3D visualization.

CLINICAL USE OF CT IN EQUINE ORTHOPEDIC IMAGING

Classic examples of using CT for equine orthopedic work are imaging of complex fractures for surgical planning (Figure 3.196) This is used most commonly for fractures of the phalanges and cuboidal carpal or tarsal bones. Many other clinical scenarios can also benefit from the use of CT, as CT has been shown to identify findings not detected on radiographs.[3] For example, non-displaced fractures of the central tarsal bone can be quite challenging to recognize radiographically but are usually quite evident with CT[8,13] (Figure 3.197).

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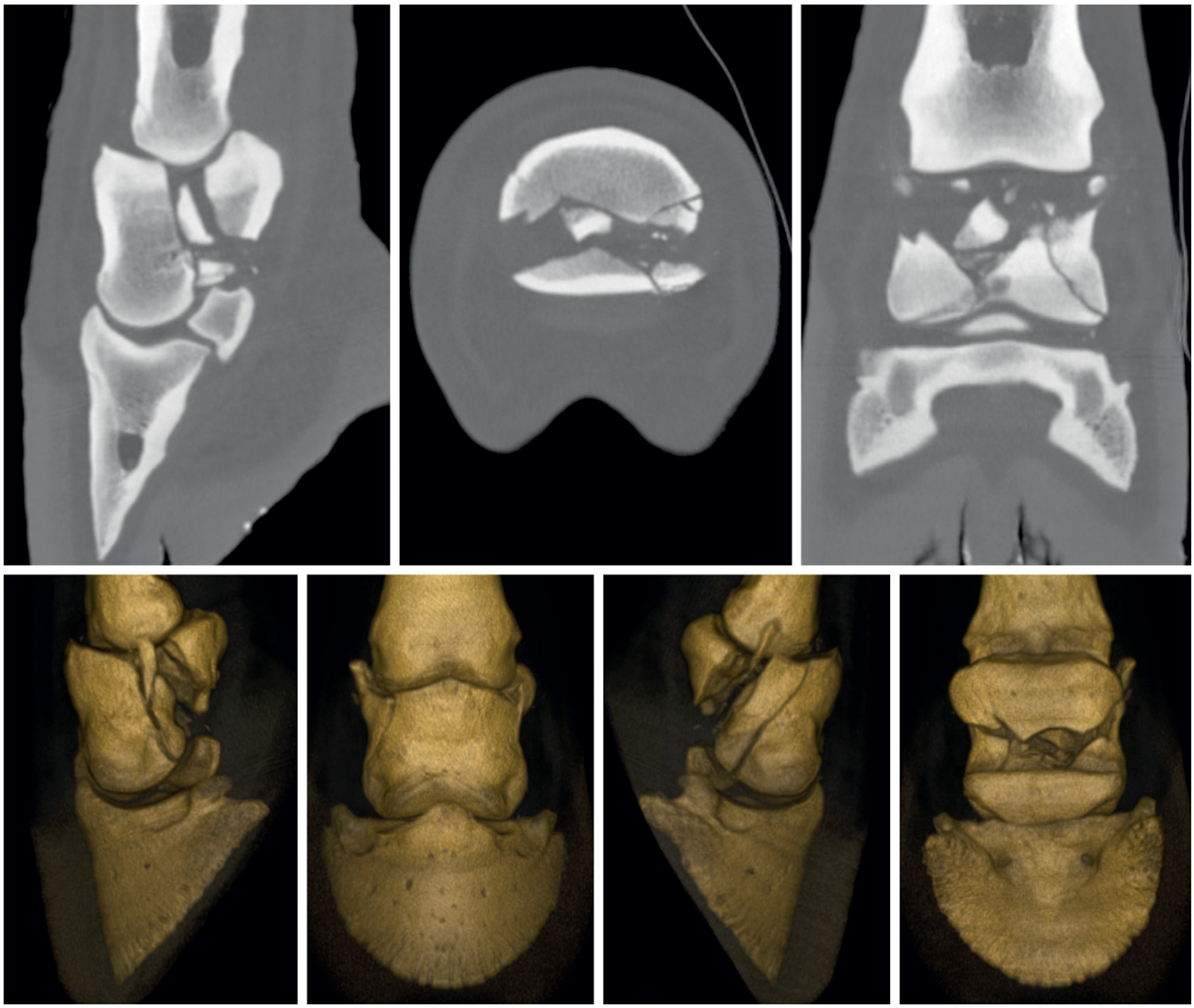


Figure 3.196. MPR and 3D rendering of a middle phalanx fracture.CT images of the left hind pastern of a 5-year-old Warmblood gelding with a complex, comminuted fracture of the middle phalanx. The top row presents multiplanar reformatted images displayed in the sagittal (left), transverse (center), and dorsal plane (right). This display provides the best assessment of the position of the different fragments. The bottom row displays four different projections of three-dimensional (3D) rendering. The 3D rendering has the advantage to provide a similar visualization to what would be seen during surgery. The information about the internal configuration of the fracture is however lost in this display.

CT is also particularly convenient to image complex structures where several abnormalities might be present concurrently such as in the foot[23,24,26] or the suspensory ligament origin.[15] In addition to providing improved assessment of the navicular bone when compared with radiographs, CT allows detection of lesions at ligamentous attachments such as the collateral ligaments of the distal interphalangeal (DIP) joint or the distal sesamoidean impair ligament, as well as assessment of the subchondral bone, the hoof wall, and the soft tissue including the tendons (Figure 3.198).

CT is also quite useful for assessment of the hoof and related structures. Keratinized structures are difficult to appreciate on MRI as they present a hypointense signal similar to the surrounding air, whereas on CT, the keratin is hyperattenuating to the soft tissue, leading to excellent visualization of the hoof wall. For this reason, CT is the imaging technique of choice for surgical planning for resection of keratomas (Figure 4.58).[12] Keratomas have a typical heterogeneous hyperattenuating appearance, and their connection with the hoof wall can be well appreciated. CT can be used for guidance to mark the localization of the keratoma on the hoof wall, in order to optimize the size and localization of the hoof wall resection (Figure 4.59).

Joint assessment is another area where CT can be quite useful. CT is excellent at assessing the subchondral bone and is beneficial in recognizing and characterizing subchondral resorptive lesions, for example, in the sagittal groove of the proximal aspect of the proximal phalanx, and other cyst-like lesions[10] (Figure 3.199). The classic radiographic findings of osteoarthritis, including

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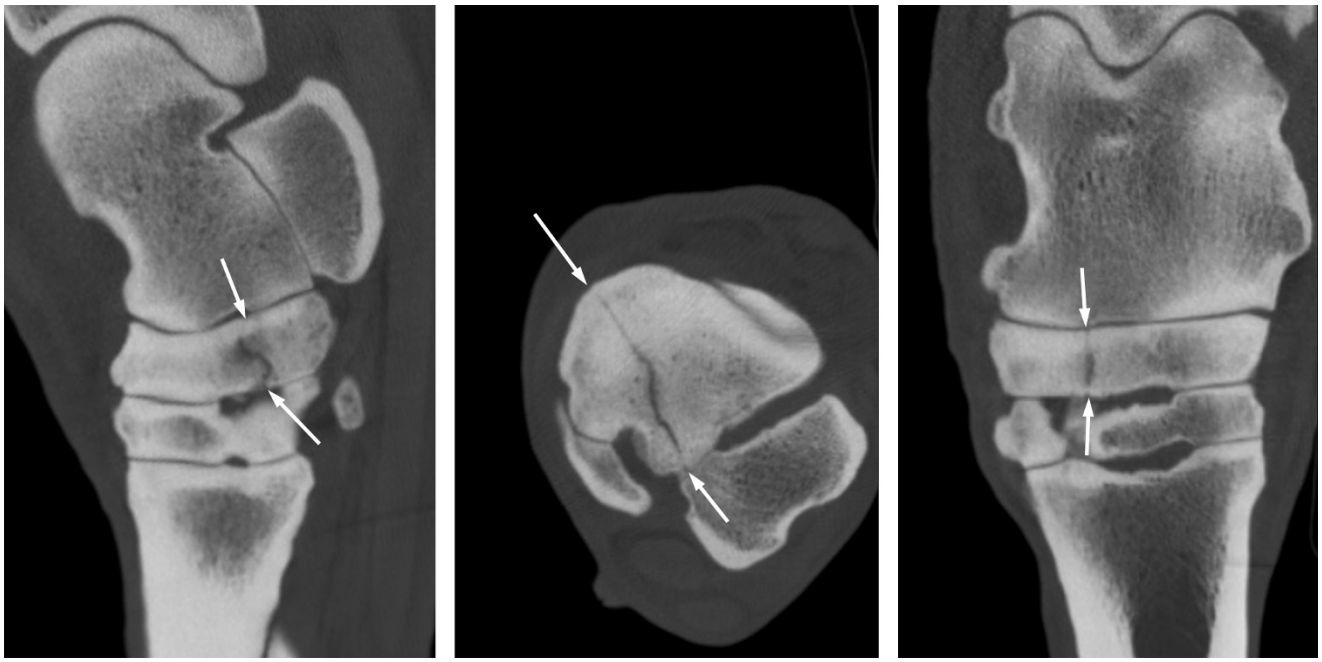


Figure 3.197. MPR of a non-displaced central tarsal bone fracture.Multiplanar reformatted CT images of the left tarsus of a 7-year-old Warmblood. A well-defined radiolucent fracture line is seen through the central tarsal bone, extending from the proximal to the distal articular surface, with a dorsomedial to plantarolateral orientation. (Arrows) Sclerosis is also recognized at the dorsomedial aspect of the central tarsal bone. This type of fracture is typically difficult to recognize on radiographs, and CT is often key in their characterization.

periarticular osteophytes, bone sclerosis, and joint space narrowing, can be also be clearly detected on CT with a better accuracy than with radiographs[18] (Figure 3.200).

The caudal cervical area has been recognized as a site that can be responsible for front limb lameness that can be difficult to localize. Although CT availability to image this area remains limited, a few reports have demonstrated its potential feasibility. It is likely that CT will play a major role in imaging of the caudal cervical spine in the future, due to the lack specificity of radiographic findings of this area.

CONTRAST-ENHANCED CT

Iodinated contrast material is commonly used in CT, either to detect lesions that were not identified without contrast or to gain additional information, especially regarding staging (acute vs. chronic) of lesions identified precontrast. Contrast can either be administered intravascular or intrasynovial depending on the indication. Due to the large dose that would be needed to perform systemic contrast administration (typically 500–1,000 mL), a peripheral arterial injection technique has been developed.[4] This technique has been used for assessment of tendon and ligament lesions, evaluation of local inflammation or sepsis, and evaluation of limb perfusion in horses with laminitis. Intrasynovial administration of contrast not only is used for joints (arthrography) to assess the cartilage for defects or thinning but also can be performed in a tendon sheath and bursa (bursogram) to detect superficial irregularities of tendons. Other indications for intrasynovial administration is to assess intra-articular ligaments (mostly in the carpus and stifle) and to evaluate synovial involvement of a wound or foreign body penetration.

CT Angiography

An arterial catheter is needed for contrast administration.[19] The technique was initially described with placing an 18-gauge catheter in the medial digital palmar artery just distal to the carpus using ultrasound guidance. Due to occasional poor perfusion of the lateral aspect of the foot using this approach, catheterization of the median artery at the distal antebrachium is now usually preferred.[9] The iodinated contrast medium, diluted 1 : 1, is injected using a power injector, starting a few seconds before imaging and continuing at a rate of 2 mL/s during the image acquisition.[19] Typically 60 mL of contrast is sufficient to cover the entire acquisition time.

Normal tendons and ligaments enhance mildly with an increased in HU of 10–20 compared with the precontrast images.[19] A few specific areas are known to have normal higher enhancement due to physiologic higher blood supply, such as the DDFT at the distal aspect of the metacarpus. Abnormal enhancement has been described using different patterns: central–peripheral, diffuse, or neovascularization[9,24] (Figure 3.201). The presence and type of enhancement is useful in characterizing the type and the chronicity of the lesion. Lack of enhancement suggests chronicity to a lesion. Peripheral enhancement is associated with central necrosis and lack of tissue repair.[9]

In cases of abscesses or penetration of foreign bodies ("street nail"), contrast-enhanced CT can be helpful in identifying focal inflammation helping to recognize penetrating or draining tracts and to assess for synovial involvement (Figure 3.202).

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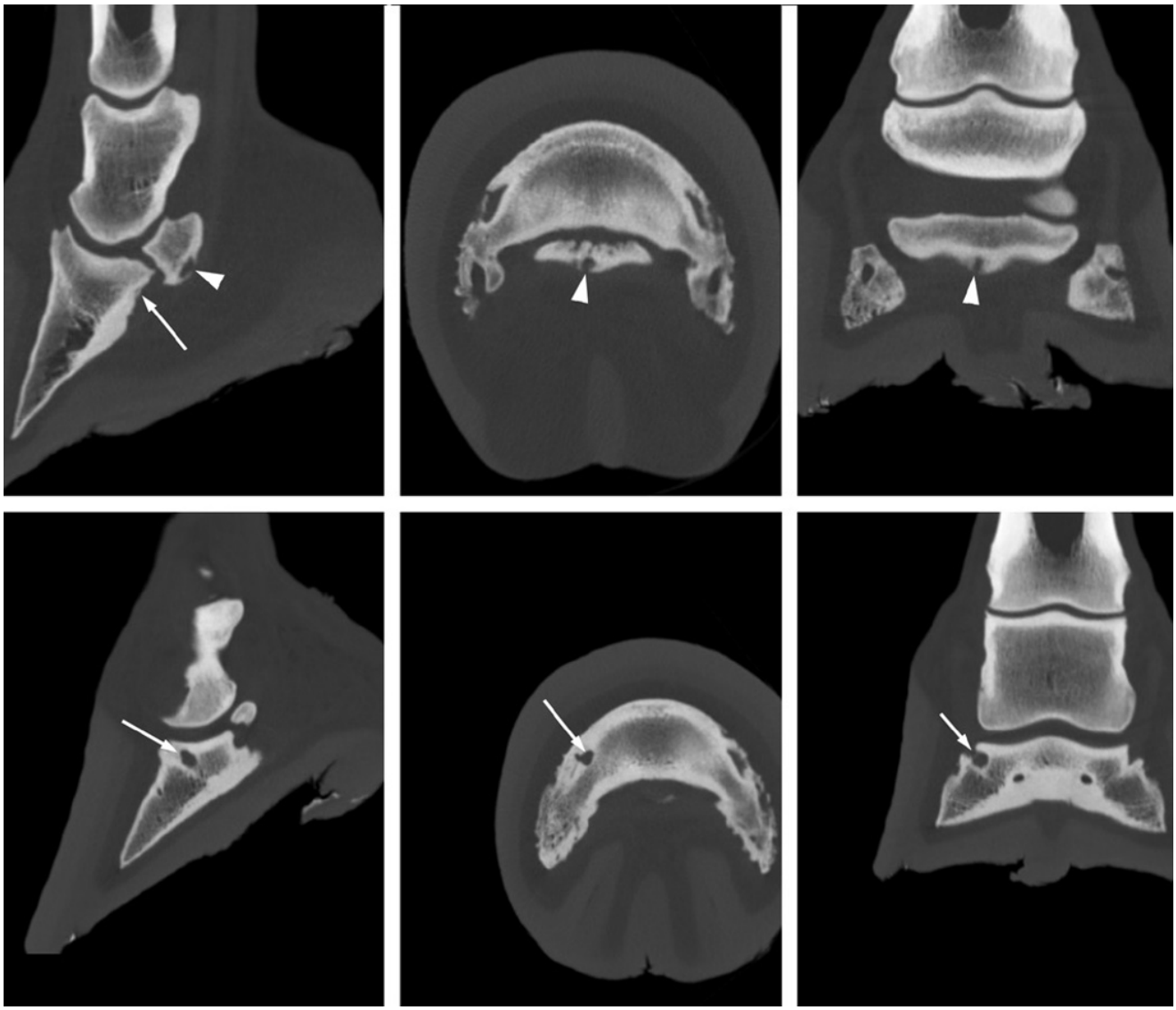


Figure 3.198. MPR of a front foot of a horse with chronic lameness.Multiplanar reformatted CT images of the right front foot of an 18-year-old Tennessee Walking horse with chronic right front lameness localized to the foot. Lateral is to the right. Multiple abnormalities are identified. There is focal osseous resorption of the central distal aspect of the flexor cortex of the navicular bone (arrow heads). Irregular contour of the flexor surface of the distal phalanx is present at the site of attachment of the distal sesamoidean impar ligament (top row, arrow). Focal osseous resorption is also identified at the attachment of the lateral collateral ligament of the DIP joint on the distal phalanx (bottom row, arrows).

CT angiography has also been used to evaluate the perfusion of the hoof wall in cases of laminitis.[14] In severe chronic cases, areas of ischemia and necrosis can be identified. In the acute phase, increased permeability of the capillary vessels can be recognized with increased retention of iodinated contrast in the lamina.

CT Arthrography and Bursography

As for all synovial punctures, the site of injection needs to be aseptically prepared. Nonionic iodinated contrast is preferred to ionic iodinated contrast in synovial cavities as it results in lower inflammation of the synovium and less intrasynovial dilution.[2] Contrast medium should be diluted prior to injection to avoid streaking artifacts. A concentration of 100–150 mg of iodine per mL is most commonly used,[7,25] but further dilutions have also been used.[11] The volume obviously depends on the joint or synovial structure injected. Adequate distension should be obtained. For examples, 20 mL has been used for the metacarpophalangeal joint,[11,17] 12 mL for the middle carpal joint and antebrachiocarpal joint,[7] 60 mL for the lateral and medial femorotibial joints,[25] and 80 mL for the femoropatellar joint.[25]

Cartilage surface irregularities, focal cartilage defects, or cartilage thinning can be appreciated with CT arthrography. The spatial resolution of the images will be key in the detection of focal defects. Differences in spatial resolution can explain the discrepancy in the literature regarding the ability of contrast CT to detect cartilage defects. Older studies using lower spatial resolution concluded a lack of sensitivity of the CT arthrography to

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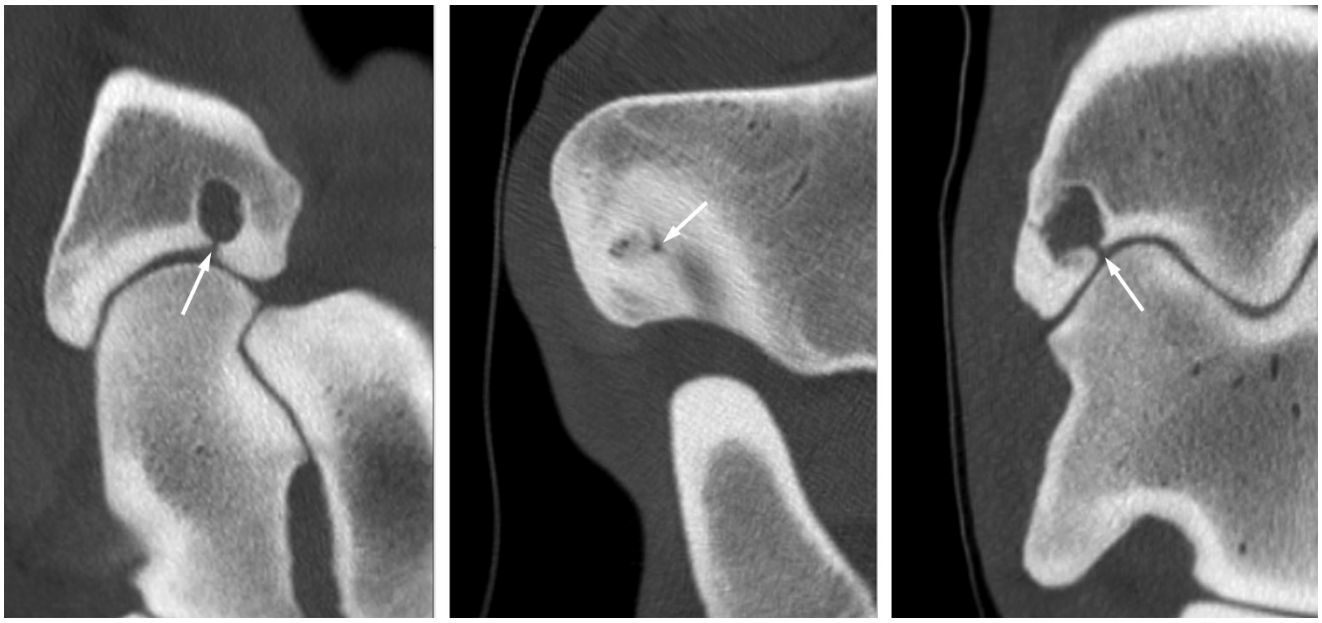


Figure 3.199. MPR of a cyst-like lesion of the distal tibia.Multiplanar reformatted CT images of the right tarsus of an 11-year-old Warmblood. There is a well-defined round lucent area in the lateral distal aspect of the tibia, with a minimal rim of sclerosis and a narrow communication with the joint through the abaxial subchondral bone of the lateral cochlear groove (arrow). CT is the ideal modality to fully characterize the configuration of such a lesion.

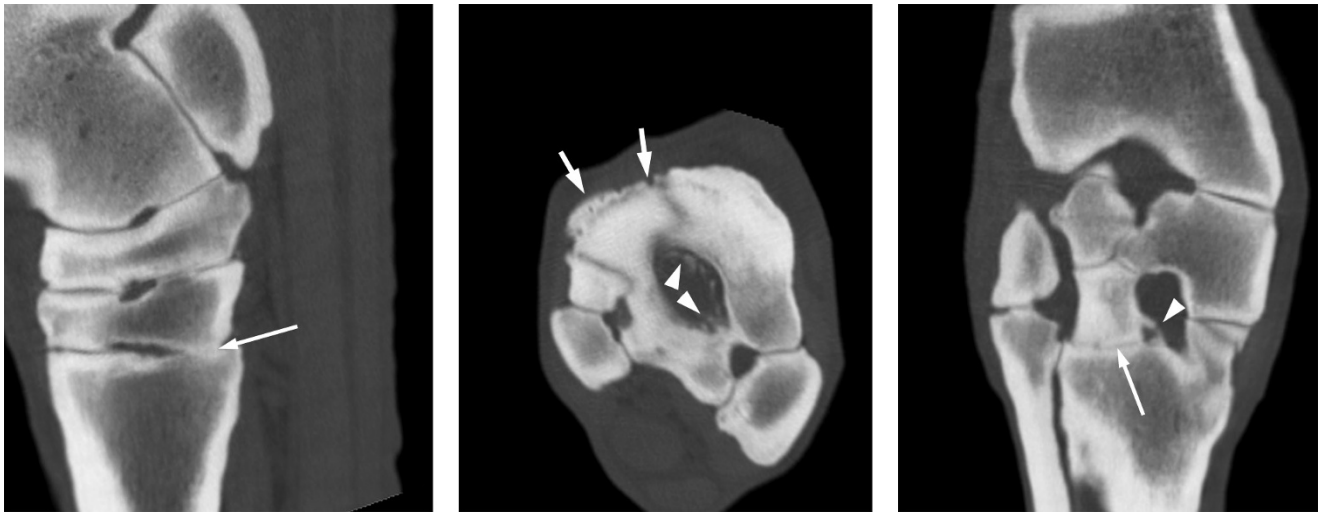


Figure 3.200. CT MPR of osteoarthritis of the tarsometatarsal joint.Multiplanar reformatted CT images of the left tarsus of an 8-year-old Quarter Horse gelding. Medial is to the left. There is marked narrowing of the plantar joint space of the tarsometatarsal joint (arrow), with adjacent sclerosis of the subchondral bone. There is periarticular osteoproliferation at the dorsomedial aspect of the proximal third metatarsal bone. There is enthesophytosis of the intertarsal ligament attachments on the third tarsal and metatarsal bones (arrowheads). CT allows a very specific characterization of the findings associated with osteoarthritis of the tarsometatarsal joint.

detect cartilage defects,[17] whereas recent studies with higher spatial resolution (voxel size 0.2 × 0.2 × 0.3 mm) found an 82% sensitivity.[11] This latter study actually demonstrated that the sensitivity for identification of cartilage defects with CT arthrography (82%) was higher than the sensitivity using a 3T MRI system (41%). This can be attributed to the difference in spatial resolution between the two systems (3T MRI, 0.4-mm pixel; CT, 0.2-mm pixel).[11] With high-resolution images, the use of multiplanar reformatting is very helpful in identifying cartilage abnormalities (Figure 3.203).

CT arthrography of the carpus allows assessment of the intercarpal ligaments, particularly the lateral and medial palmar intercarpal ligaments.[7] CT imaging of the stifle remains limited due to size considerations, and typically larger gantries are needed. CT stifle arthrography has shown promising results for the evaluation of the cruciate ligaments.[5,16,25] Superficial lesions of the

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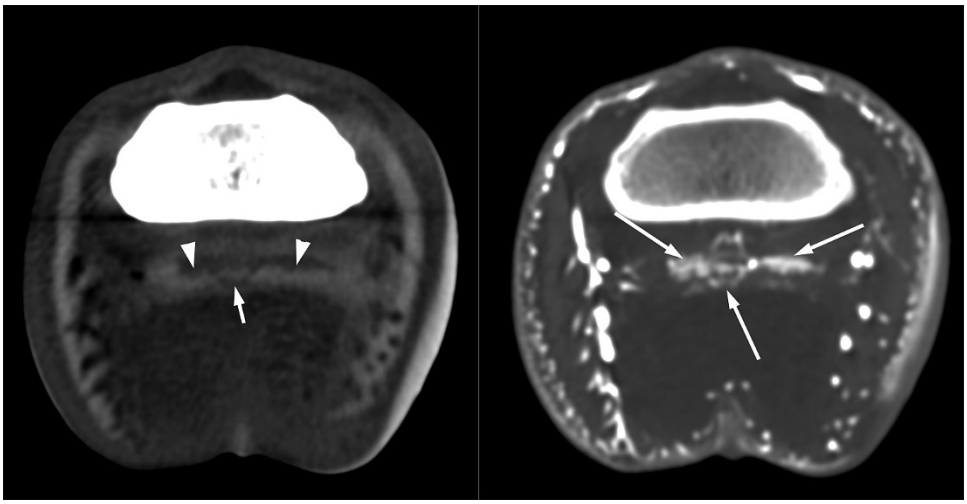


Figure 3.201. Deep digital flexor tendon lesion with arterial contrast enhancement. Transverse pre-contrast (left) and post-arterial contrast (right) CT images through the middle phalanx of a 22-year-old Quarter Horse mare. There is irregularity of the dorsal margin of the DDFT in the navicular bursa (arrow heads). A focal hypoattenuating area is present in the central palmar aspect of the tendon. (Short arrow) All these areas demonstrate marked contrast enhancement (arrows), suggesting the presence of active lesions.

deep and superficial digital flexor tendons can also be better characterized with injection of contrast within the digital sheath.[1] Navicular bursograms have been used to identify dorsal fibrillation of the DDFT or adhesion to the navicular bone.

Similarly to what is more commonly done with radiography, distension of a synovial structure with iodinated contrast medium to assess for communication of a wound or penetrating injury can be performed with CT. This increases the sensitivity of the procedure and often improves the characterization of penetrating or draining tracts. This is of particular value with penetrating injuries of the foot with suspicion of involvement of the navicular bursa.

POSITRON EMISSION TOMOGRAPHY

A recent interesting addition to CT is the development of equine positron emission tomography (PET). PET is a cross-sectional nuclear medicine imaging technique. In a way PET is to scintigraphy, what CT is to radiography. It provides similar type of information but resolves the issue of superposition. PET also has the advantage of having higher spatial resolution than scintigraphy. Several PET tracers can be used, providing information not only about bone lesions but also soft tissue lesions. PET presents functional information but does not include anatomical detail. For this reason, PET is usually combined with CT. The PET data can be fused with the CT images to demonstrate the presence of active lesions. The first equine PET scan was performed in 2015 using a portable compact PET scanner designed to image the human brain.[20]

Currently the main equine PET application is the use of the 18F-sodium fluoride (18F-NaF) radiotracer to detect areas of bone remodeling. 18F-NaF gets integrated into the hydroxyapatite matrix of bone at sites of bone turnover. This was shown useful to detect enthesiopathy or early subchondral remodeling that was not recognized with CT or MRI[21] (Figure 3.204). Also, when chronic osseous lesions are detected with CT, the presence or absence of 18F-NaF uptake allows to differentiate between active and inactive processes. 18F-NaF PET was shown particularly helpful in a Thoroughbred racehorse population to detect early stress remodeling with a higher sensitivity than all other imaging modalities. In particular PET detected focal 18F-NaF uptake in the proximal sesamoid bones of racehorses in training at sites known for catastrophic breakdown.[22] PET has the potential to become a useful tool in the prevention of catastrophic injuries.

18F-Fluorodeoxyglucose (18F-FDG) is another PET tracer commonly used in oncology imaging. 18F-FDG not only is a radioactive glucose and is not specific of neoplastic disease but also can be used to image inflammation. Limited information is currently available about 18F-FDG PET in the horse, but an exploratory study demonstrated values of the technique for assessment of tendon injuries and for laminitis research.[20] 18F-FDG leads to distinction between active and inactive tendon lesions,[6] which is of particular interest for follow-up on tendon healing and for assessment of chronic lesions (Figure 3.205). Similarly, PET could become a useful research tool in horses with laminitis to quantify and monitor the level of inflammation present in the lamina.

CONCLUSION

CT is not as commonly used as MRI for equine musculoskeletal imaging, but its role should not be overlooked. MRI tends to be preferred due to its ability to image both bone and soft tissue, but with proper understanding of CT technique and use of iodinated contrast

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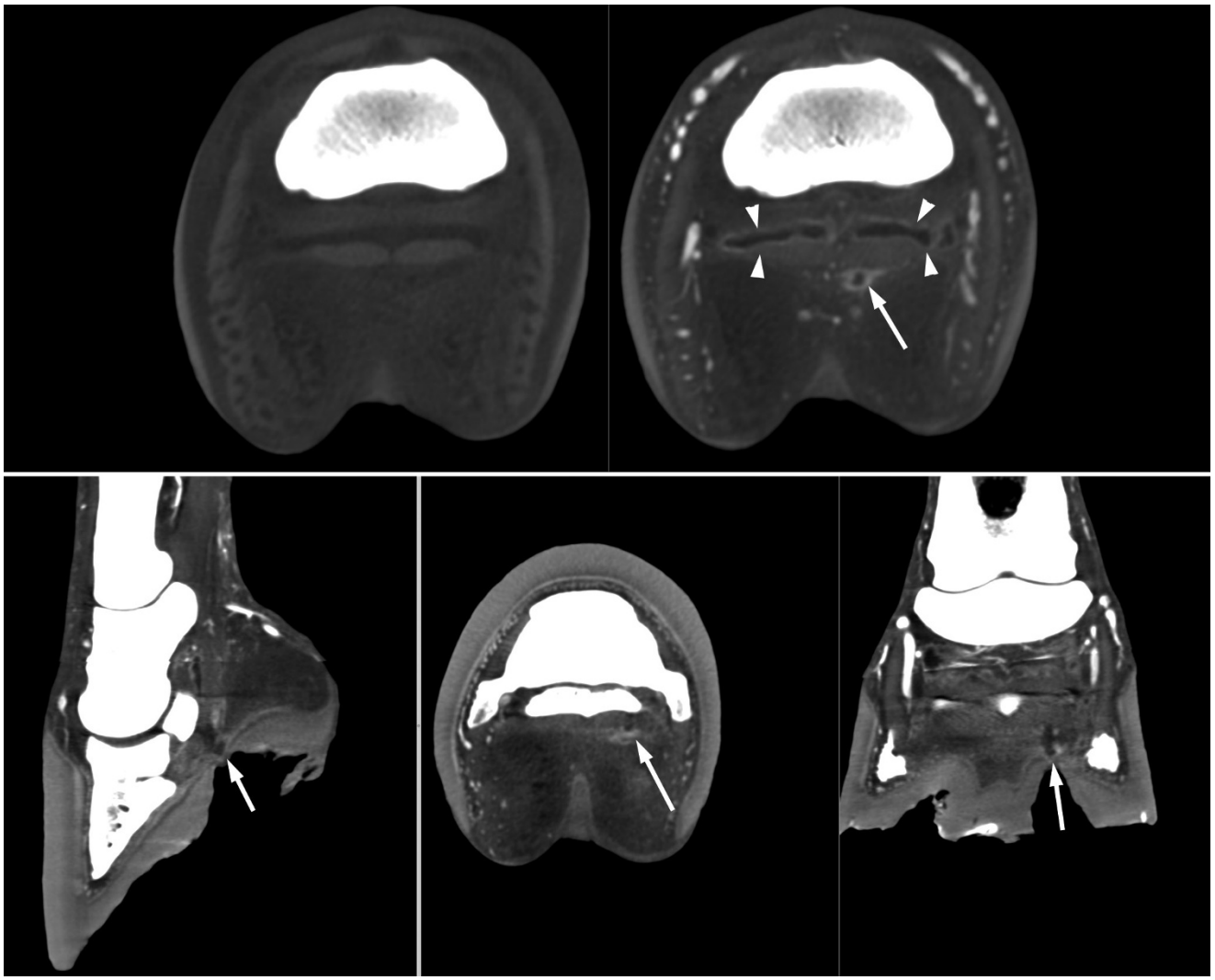


Figure 3.202. "Street nail" injury with penetration of the navicular bursa.Transverse pre-contrast (top row left), transverse post-arterial contrast (top row right), and MPR post-arterial contrast CT images of the right hindfoot of a 21-year-old Warmblood gelding, with a history of nail penetration through the sole. Lateral is to the left. A hypoattenuating tract with marked peripheral contrast enhancement is identified extending from the medial sulcus of the frog through the DDFT at the level of the navicular bone. (Arrow) There is marked synovial enhancement of the navicular bursa. (Arrow heads) This led to a diagnosis of septic navicular bursitis secondary to nail penetration.

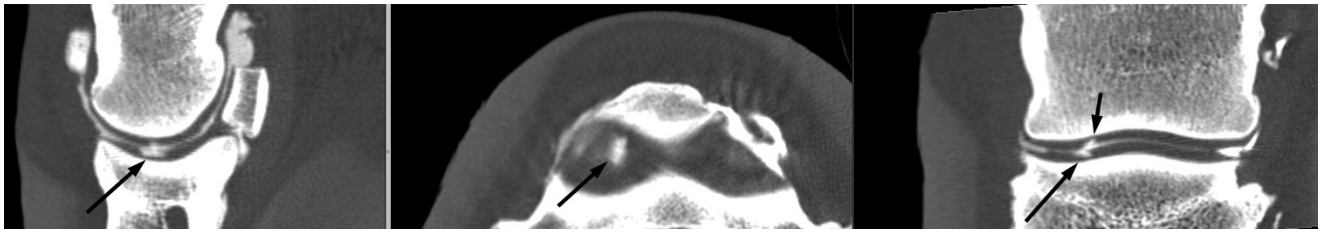


Figure 3.203. Articular cartilage defect of the distal interphalangeal joint.Multiplanar reformatted CT images of the left front foot of a 10-year-old Warmblood gelding, post-intra-articular injection of iodinated contrast material in the DIP joint. Medial is to the left. There is evidence of focal extension of the contrast material within the region of the medial articular cartilage of both the middle and distal phalanges, suggesting the presence of cartilage defects (arrows). This lesion could not be recognized with the realization of the arthrogram.

agent, pertinent soft tissue information can be obtained with CT. The comparison of the two modalities regarding bone imaging is interesting: CT has been classically considered the ideal technique for bone imaging with high contrast obtained from mineralized tissue and high spatial resolution available. However, CT suffered from the limitation of not being able to recognize acute "bone edema"-type lesions identified with MRI. The introduction

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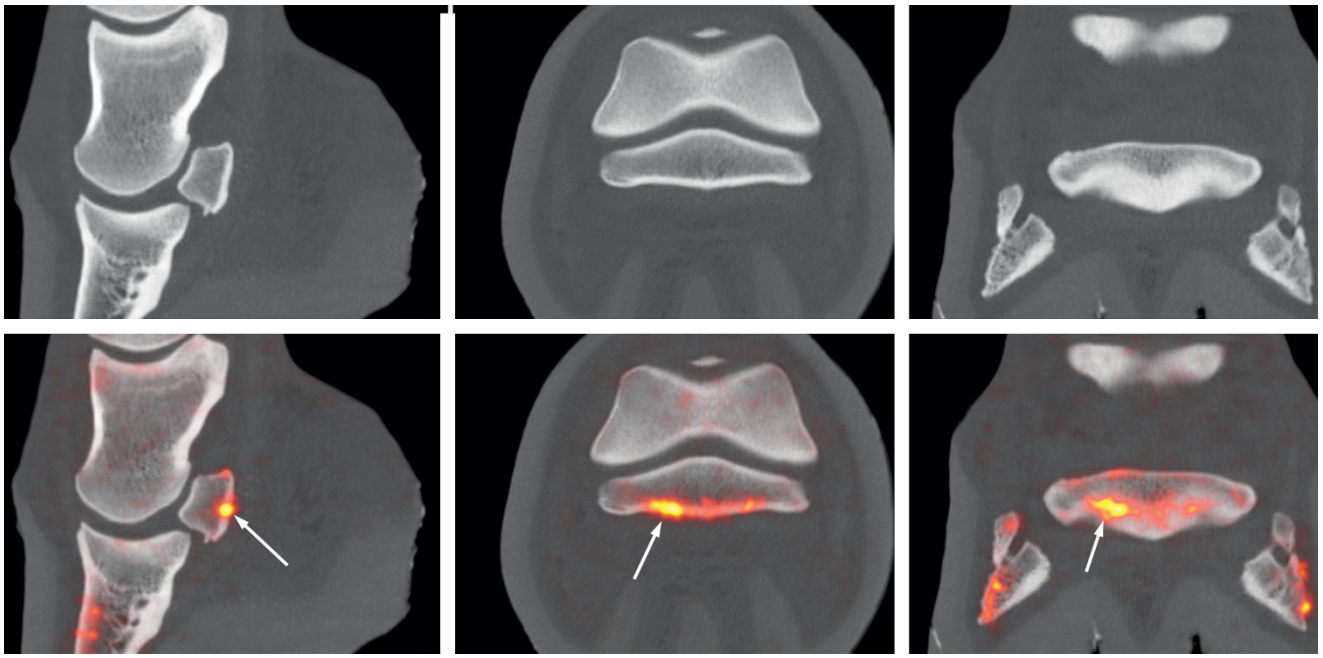


Figure 3.204. 18F-NaF PET CT of mild navicular remodeling.Multiplanar reformatted CT (top) and fused 18F-NaF PET/CT (bottom) images of the left front foot of a 10-year-old Warmblood with lameness localized to the foot. Medial is to the left. There are no significant abnormalities of the navicular bone on the CT images, but the PET demonstrates marked focal uptake of the flexor cortex of the navicular bone, worse medially. (Arrow) This indicates early remodeling of the navicular bone.

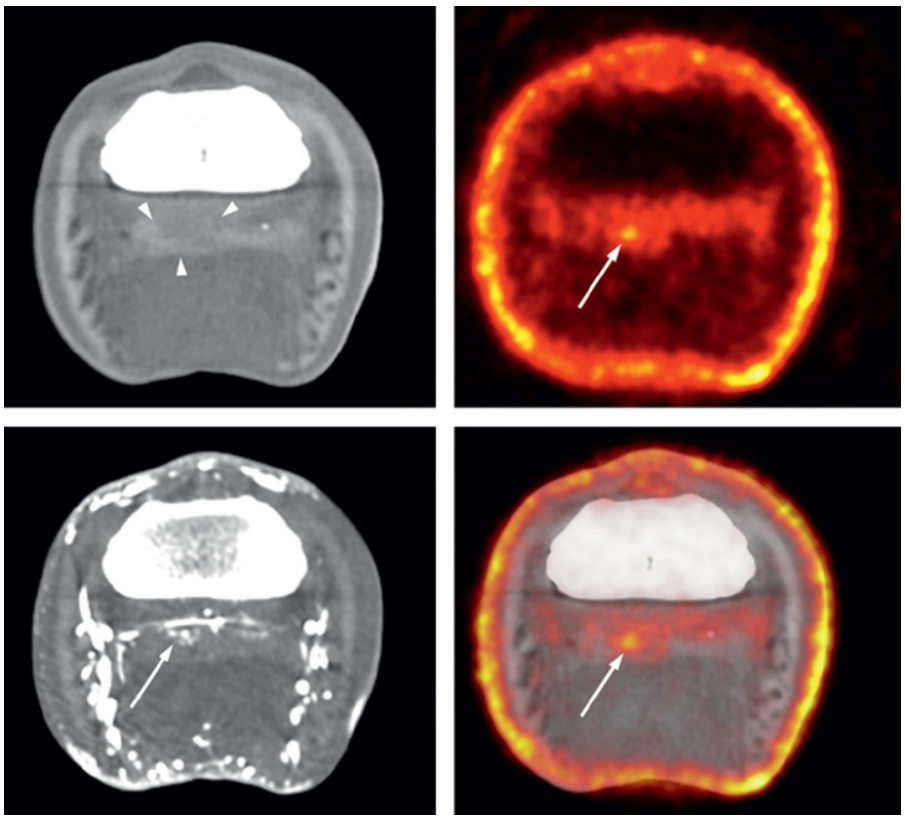


Figure 3.205. Deep digital flexor tendon lesion imaged with 18F-FDG PET/CT.Transverse CT (top left), 18F-FDG PET (top right), arterial contrast CT (bottom left), and fused PET/CT (bottom right) images of the right front foot of a 22-year-old Quarter Horse mare through the middle phalanx. Lateral is to the left. The lateral lobe of the DDFT appears enlarged with irregular margin on the pre-contrast CT (arrowheads). There is marked focal 18F-FDG uptake of the lateral lobe of the tendon (upper right arrow). Arterial contrast enhancement is also present on CT. Note that on arterial contrast CT, both the lesions and adjacent vessels demonstrate enhancement(lower left arrow), whereas on the PET images only the lesion shows increased uptake (lower right arrow).

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of 18F-NaF PET to equine imaging provides an opportunity to combine the excellent bone structural representation of CT with very sensitive information regarding acute bone changes. The combination of CT with PET potentially makes it the ideal technique for bone imaging.

CT also has a clear advantage over MRI residing in the speed of acquisition, allowing imaging of larger areas. For example, it is possible to image from the carpus to the distal extremity with CT in a couple of minutes. Imaging this same area MRI would take hours and require multiple studies. The ability to image the distal limb standing has been an important factor to the development of equine MRI. As a few CT concepts now suggest that standing CT imaging might become more widely available, it is possible that the use of CT in the years to come will be drastically changed by technology evolution.

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MAGNETIC RESONANCE IMAGING

Michael Schramme and Emilie Segard-Weisse

INTRODUCTION

Magnetic resonance imaging (MRI) is a multiplanar cross-sectional imaging modality that uses the inherent magnetic properties of tissue and is fast becoming the gold standard for diagnosis of musculoskeletal injury of the distal limb in horses. MRI allows soft tissue and bone structures to be evaluated in ways not possible with other imaging modalities. The information obtained is based on the structure and biochemical environment of hydrogen atoms in the tissues.

Because of its unique mode of image acquisition and construction, it is important to understand the factors that influence the MR signal characteristics that produce the diagnostic images and to learn the strengths and weaknesses of the technique. The clinician must understand the basic physics and the basic sequences used for acquisition in order to comprehend the relationship between signal abnormalities and pathology and distinguish between signal abnormality and artifact and between signal change and normal anatomical variation. The increasing use of MRI in equine sport medicine requires every equine practitioner to have a basic knowledge of MRI interpretation. The importance of correlating the MRI findings with results of the clinical examination, diagnostic analgesia, and other imaging modalities, in order to determine the clinical significance of each suspected lesion, cannot be overstated.

Accurate knowledge of the advantages and disadvantages of MRI helps the clinician to make the correct imaging choices for the benefit of patients and clients. MRI is not a substitute for in-depth clinical investigation and conventional imaging techniques, and many diagnoses can continue to be made without MRI. Nonetheless, the use of MRI has highlighted the limitations of both radiography for imaging bone and ultrasonography for imaging soft tissue lesions. MRI has proven to be particularly helpful in patients in which the lameness has been localized to a certain area of the distal limb, but radiography and ultrasonography do not show an abnormality that explains lameness in a satisfactory manner.

GENERAL PRINCIPLES AND PHYSICS OF MRI

The generation of an MR image is based on the movement of hydrogen atoms generating a measurable electrical current in the body. MRI produces a grayscale image of tissue hydrogen protons by placing them in a strong magnetic field, exposing them to a radiofrequency (RF) pulse and measuring the magnetic resonance caused in the tissues in response to this pulse. A computer interprets the data and creates images that display the different resonance characteristics of different tissue types on a gray scale.

The resonance that is measured originates from the magnetic properties of the positively charged proton in the nuclei of hydrogen atoms in biological tissues. When exposed to a short RF pulse specific for hydrogen, which is emitted by the RF coil, hydrogen nuclei absorb this pulse and change their alignment within the main magnetic field. Following discontinuation of the RF pulse, the hydrogen nuclei resume their previous orientation of parallel alignment with the main magnetic field, thereby transitioning from a high-energy to a low-energy state. This transition results in energy release that is used to generate an electrical signal. The exchange of energy between different spin states is called the resonance and thus the name MRI. The time required for the hydrogen nuclei to resume equilibrium within the main magnetic field is the relaxation time, measured in milliseconds. The relaxation of hydrogen nuclei can be divided into two separate components, the longitudinal (T1) and transverse relaxations (T2). T2 is much shorter than T1. Most tissues can be characterized by their T1 and T2 signal properties. Protons are present in different densities and with different T1 and T2 properties between different tissues, thereby producing contrast differences between tissues on MR images. The degree to which the hydrogen protons are bound within a tissue determines their relaxation time. Protons in fluid or fat return to alignment quickly, whereas those in organized tendinous, ligamentous, or osseous tissue return slowly.

As fat and water both contain a large amount of mobile hydrogen atoms, the strength of the resonance signal also depends on the amount of fat and water in the tissue. High signal areas are white, and low-signal areas are black.

EQUIPMENT, HIGH- AND LOW-FIELD MAGNETS, AND RF COILS

The basic components of MR imaging are an RF shielded room, a body with mobile protons, a magnet generating a strong magnetic field, an RF coil to add energy to the protons to make them move, a receiver coil to collect the electrical signal generated by the moving protons, and a computer with acquisition software to time these events and generate an image from the electrical signals.

Magnetic field strength is expressed in tesla units. One tesla (T) is approximately 20,000 times the strength of the earth's magnetic field. High-strength magnetic fields measure in excess of 1T, low-strength fields less than 0.5T, and mid-strength fields between 0.5 and 1T. Superconducting, closed, cylindrical bore magnets (Figure 3.206) generate high-strength magnetic fields, while resistive or permanent, open magnets (Figure 3.207) produce low-strength fields. Signal strength is proportional to the strength of the magnetic field. Consequently low-field systems generate less tissue signal, require longer acquisition times, and produce lower-resolution images. The uniformity or homogeneity of the main magnetic field is higher in closed than in open magnets.

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Figure 3.206. A Siemens Magnetom Skyra 3T high-field magnet (Siemens, Malvern, PA) used for MRI of horses under general anesthesia. The horse is positioned in lateral recumbency with the lame limb lowermost. The region of interest in the limb, in this case the foot, is positioned in the isocenter of the magnet.



Figure 3.207. The Hallmarq Equine Limbscanner® (Hallmarq Veterinary Imaging, Guilford, UK) is an open, low-field magnet, mounted vertically at floor level and allows MRI to be performed on standing, sedated horses. *Source*: ©GuildfordPhotographer, courtesy of Hallmarq Veterinary Imaging.

Image quality and resolution increase with increasing magnetic field strength and increasing field homogeneity. Both high-field and low-field MRI systems are currently in clinical use for horses.

Though 1.5 T is still considered the standard for high-field imaging of horses (Figure 3.206), short bore 3.0 T magnets with large bore size (70 cm) and flared ends are now available for clinical imaging, some even reaching up to and including the stifle region. The area of the limb to be imaged must be positioned in or near the isocenter of the cylindrical bore of the magnet, which lies at the intersection of all three gradient coils and is

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Figure 3.208. The G-Scan Vet® (Universal Medical Systems, Inc. Solon, OH) is an open, 0.25-T low-field magnet that can be rotated from a horizontal to a vertical position to accommodate imaging of the horse's head, neck, and stifle more easily. *Source:* Courtesy of Dr. Martin Waselau.

the optimal position for imaging. This requires the horse to be recumbent and therefore under general anesthesia, which increases the cost and the risk of the procedure. Although high-field magnets are generally capable of imaging limbs of horses from the carpus and tarsus distally, this capability is limited by how far the horse can be pulled into the bore of the magnet. Therefore, not all high-field magnets are equal in this respect. Positioning in isocenter is more difficult in longer and narrower cylindrical bores than in some short bore magnets with flared ends. In addition, some high-field magnets have a much tighter imaging window around the isocenter than others, which makes it harder to pull areas of interest further proximal than the fetlock region into the imaging window.

Low-field MRI of horses is currently performed with permanent, open magnets with a field strength ranging from 0.20 to 0.31T. One open, low-field MRI scanner designed specifically for imaging distal limbs of standing, sedated horses (EQ2 – Hallmarq Veterinary Imaging, Guilford, UK) is mounted vertically at floor level (Figure 3.207). However, imaging of areas proximal to the fetlock region is prone to motion artifact with these units. Other low-field magnets (O-Scan Equine®, Vet MR Grande®, and G-Scan Vet®, Universal Medical Systems, Inc. Solon, OH) are oriented horizontally on a pedestal and require horses to be placed under general anesthesia. The O-Scan Equine® is a small magnet with an opening of 18 × 34 cm that is self-shielded within its own RF shielding cage with an internal thermostatic control system and can be used in any nonspecialized room. The G-Scan Vet® can be rotated from a horizontal to a vertical position to more easily accommodate imaging of the horse's head, neck, and stifle (Figure 3.208).

Low-field magnets produce a lower signal-to-noise ratio resulting in reduced image resolution and detail. Although many lesions have a higher conspicuity on high-field images, several studies have shown that low-field magnets are capable of producing diagnostic quality images of the distal limb, metacarpus/metatarsus, carpus, tarsus, and stifle. However, a high-field system is required to identify certain structures and lesions, especially those related to articular cartilage.[173,188,193]

Horses undergoing MRI must have all metal and ferrous material removed to avoid interference with the magnetic field and generation of susceptibility artifacts. This includes shoes, nails, metallic debris in the nail holes and sole, and the occasional metallic implant. Other ferromagnetic materials such as horse support tables and anesthetic equipment may interfere with the homogeneity of the magnetic field and should be avoided in the RF-shielded MRI room.

SEQUENCES AND PROTOCOLS FOR EQUINE MRI

MRI examinations rely on the use of several different acquisition sequences.[184] Each sequence name describes the RF pulse applied, the weighting of that pulse, and the associated magnetic field gradients. Different sequences used in conjunction to image a given anatomical area define the imaging protocol. It is necessary to use several sequences in multiple image planes within a protocol in order to identify pathological conditions accurately. The common categories of conventional MRI sequences are spin echoes (SE), turbo spin echoes (TSEs), gradient recalled echoes (GRE) and short tau inversion recovery sequences (STIR). The difference between these MR sequences lies in the method and timing of how the RF signals are pulsed into the tissues and how the resonance is collected to generate an image.

Fast spin echo (FSE) or TSE sequences are used as a more practical alternative to SE to reduce acquisition times while maintaining signal-to-noise ratio. The purpose

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of GREs is to decrease acquisition times further and to allow three-dimensional (3D) volumetric acquisitions with thin slices. This is especially useful in standing MRI where motion artifacts and relatively low signal-to-noise ratio are of concern. However, the advantage of fast 3D acquisition of gradient echoes comes at the expense of disadvantages like decreased soft tissue contrast, increased susceptibility to magnetic field inhomogeneities, and susceptibility artifacts. Most current clinical protocols in horses use a mixture of FSE and GRE sequences.

As many lesions tend to have increased signal intensity on MR images while at the same time fat retains high signal intensity in all sequences, eliminating the high signal intensity from fat (fat suppression) markedly increases the ability of recognizing high signal intensity from a lesion on an MR image. Fat suppression can be achieved by inversion recovery (STIR) or by a process of fat saturation.

As a result of fat suppression, adipose tissue appears black instead of white, making fluid the only remaining source of high signal on a fat-suppressed image, amidst dark bone, soft tissues, and fat. Fat suppression is commonly used in orthopedic imaging for the detection of abnormal fluid in bone (e.g. the navicular bone).

Depending on whether T1 or T2 relaxation is measured during acquisition, sequences are called T1 weighted, T2 weighted, or proton density (PD) weighted (between both T1 and T2). T1 and T2 relaxation times have characteristic values for each type of tissue and can be used in combination to describe the magnetic properties of all tissues (Table 3.2) (Figure 3.209). PD images display any change in the density of protons as a change in signal intensity.

An imaging protocol or series of different sequences is typically grouped together to image a region of interest. Both high- and low-field musculoskeletal protocols typically include PD spin echo, T2-weighted FSE, short tau inversion recovery (STIR) FSE, and T1- and T2*-weighted 3D GRE sequences.

PD images are easiest to evaluate ligament and tendon margins and symmetry. T2-weighted images are useful for looking at fluid in soft tissues thanks to their high fluid contrast. However, T2-weighted images show minimal shades of gray resulting in poor definition of soft tissue margins. STIR images show fluid in bone and soft tissues most easily because of their high fluid contrast, but they have a low resolution. T2*-weighted or spoiled T1-weighted GRE sequences are used in 3D acquisitions for evaluation of the fine anatomical detail in thin tissue slices.

The choice of protocol is determined by the type of magnet (high or low field), the manufacturer's sequence library, the region under scrutiny, and the preference of the attending clinician. Consequently, protocols may differ between hospitals and clinicians. With an endless number of combinations of sequences, weightings, and section planes available, it is tempting to continue scanning every patient until the clinician feels all questions have been answered. However, this approach lacks consistency and may cause problems. Sequences and section planes should be standardized to allow for consistent comparison between limbs and between horses. Moreover, excessive scanning times and prolonged recumbence within a confined magnet space with limited padding can result in post-anesthetic complications. A routine high-field MRI examination takes approximately 60–90 minutes to complete.

For optimal assessment of an anatomical region, images are generally obtained in three orthogonal planes, sagittal, dorsal, and transverse. Sagittal images serve as anatomical reference images for transverse sections. Transverse images are most useful to the identification of pathology, and the presence of asymmetry between the medial and lateral halves of the images may help identify lesions. Dorsal images are useful for definition of joint space width and cartilage thickness and also serve as reference images for transverse section planes.

ARTIFACTS OF MRI

MRI produces a wide range of artifacts and variations that can confuse the interpreter. In addition, MRI is susceptible to artifacts that are created by acquisition of images at oblique angles. Even slightly asymmetric positioning of image slices can create significant problems of image interpretation. Consequently, MRI is an imaging technique that can easily lead the examiner to overinterpret images as well as miss lesions.

Artifacts can be classified as motion artifacts, magnetic field heterogeneity artifacts, and digital imaging artifacts.

Motion artifacts generally result in ghosting, which results from displaced reduplications of the image in the phase-encoding direction, or in marked blurring of the image. Swaying of a standing sedated horse results in blurring of images, which may render the entire sequence nondiagnostic. STIR sequences are worst affected by this type of motion artifact. Respiration and blood flow can cause multiple motion ghosting in anesthetized patients (Figure 3.210).

Artifacts from magnetic field inhomogeneity lead to image distortion or alterations in signal intensity and are more common in low-field than high-field magnets. Temperature fluctuations, the presence of ferrous metal, and even some wound sprays or frog packing materials like blue Play-Doh may create field inhomogeneities. Magnetic susceptibility artifacts are a form of field inhomogeneity artifact as they are caused by the presence of materials with different susceptibility to magnetization, like ferrous metals or iron in hemoglobin and hemosiderin in tissues (Figure 3.211).[181,186] GREs are particularly susceptible to these artifacts. Feet should be radiographed routinely prior to MRI to ensure that all metal fragments and debris have been removed from the hoof wall.[185]

Chemical shift artifacts are caused by the presence of fat and water adjacent to each other. Protons from water and fat have different precession frequencies that causes the position of fat signal to shift in the frequency-encoding direction of the image relative to the position of water signal. The shifting of fat signal to a different location can results in spatial misregistration. This artifact is

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Table 3.2. The signal intensity of different tissues in different contrast weightings.

Sequence	T2	T1	Proton density	Inversion recovery
Cortical bone	Black	Black	Black	Black
Cancellous bone	Light gray	Light gray	Light gray	Light gray
Cartilage	Dark gray	Light gray	Gray	Gray
Tendon	Black	Black	Black	Black
Ligament	Black	Gray to black	Black	Black
Fat	Light gray	White	White	Black
Fluid	White	Dark gray	Light gray	White

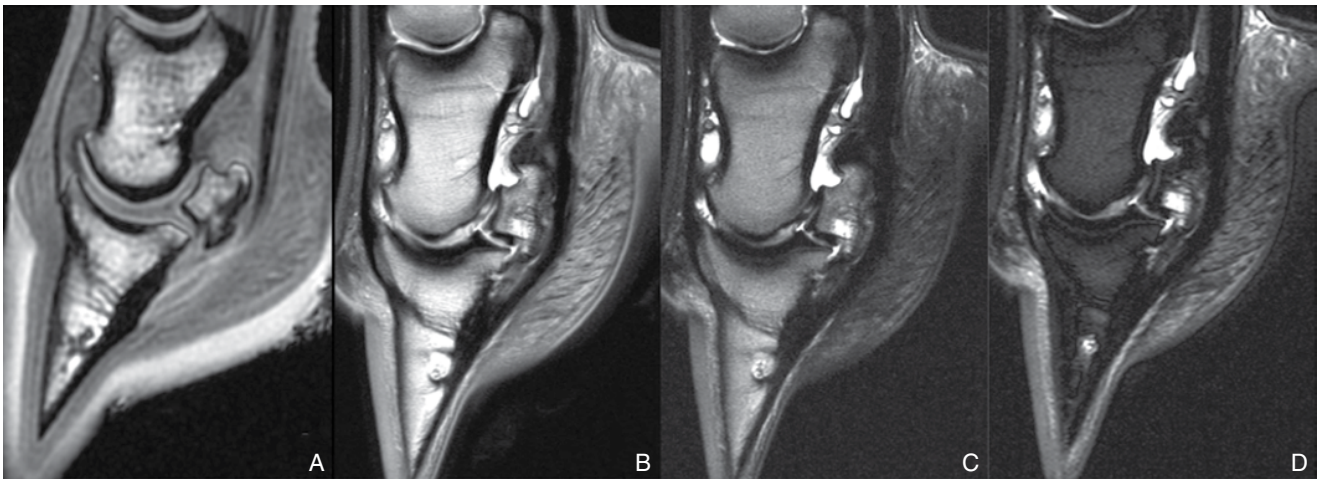


Figure 3.209. T1-weighted (A), proton density (B), T2-weighted (C), and short tau inversion recovery (STIR) sagittal images of the foot of a horse with navicular bone disease. Cortical bone, tendons, and ligaments are black on all sequences. On the T1-weighted image (A), fat is white, and fluid is dark gray. On the proton density image (B), fat is white, and fluid is light gray. On the T2-weighted image (C), fat and fluid are white. On the STIR image (D), fat is black, and fluid is white. In general, T1-weighted and PD images show anatomical detail well, while T2-weighted and inversion recovery images show less detail but have higher fluid contrast.

most obvious on high-field images. A specific fat/water phase cancelation artifact occurs in T2* images with echo time (TE) around 13 obtained with the Hallmarq MR system (Hallmarq Veterinary Imaging, Ltd. Guilford, UK).[189] In areas of medullary bone (e.g. in the navicular bone) with an equal amount of water and fat, signals from both fat and water cancel each other out and are replaced with an area of zero signal (black) in the medullary cavity of the affected bone. This can lead to an erroneous diagnosis of low signal in bone as sclerosis when it actually represents fluid.

Magic angle artifact causes a sudden increase in signal on T1, PD, and T2* sequences in tendons and ligaments where collagen is orientated at an angle of around 55° to the main magnetic field (Figure 3.212). This magic angle effect must be recognized as it can mimic lesions in tendons and ligaments. It is most obvious on high-field images of the distal part of the deep digital flexor tendon (DDFT) toward its insertion on the distal phalanx[31,176] or on low-field images of the lateral lobe of the DDFT where it courses palmar to the navicular bone.[177] Magic angle effect has also been described in low-field images of the superficial digital flexor tendon,[163] the oblique distal sesamoidean ligaments[171,172], and the collateral ligaments of the distal interphalangeal (DIP) joint.[74,171,178] The collateral ligaments of the DIP joint are also susceptible to magic angle artifact in high-field magnets, dependent on the orientation of the limb relative to the long axis of the bore of the magnet.[192,194] In a standing sedated horse with the limb abducted, the structures specifically at risk are the lateral collateral ligament of the DIP joint, the lateral lobe of the DDFT, and the medial oblique distal sesamoidean ligament. Magic angle artifact is particularly noticeable in sequences with a short TE (T1 and PD sequences) and is less evident on sequences with long TE (T2 images). Magic angle artifact can be reduced on T2-weighted images by increasing TE time.[194]

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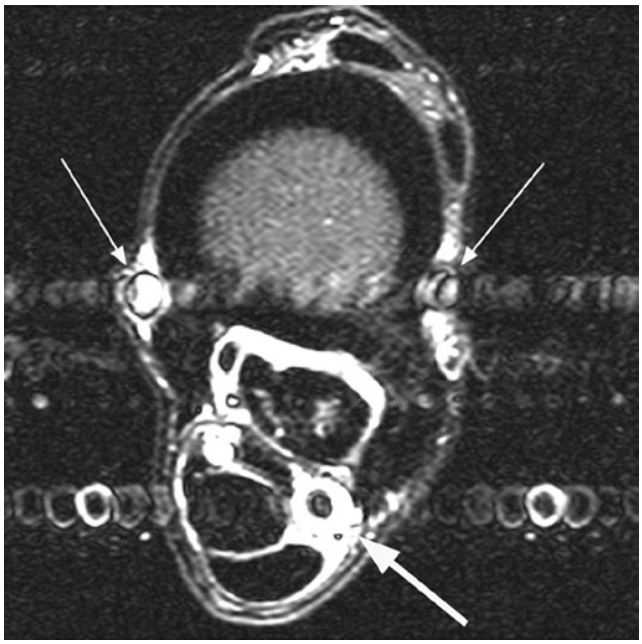


Figure 3.210. Transverse short tau inversion recovery (STIR) image of the right hind proximal metatarsal region. Multiple motion ghosting caused by blood flow results in displaced reduplications of the images of the lateral and medial dorsal metatarsal arteries (narrow arrows) and the medial metatarsal artery (broad arrow) in the phase-encoding direction.

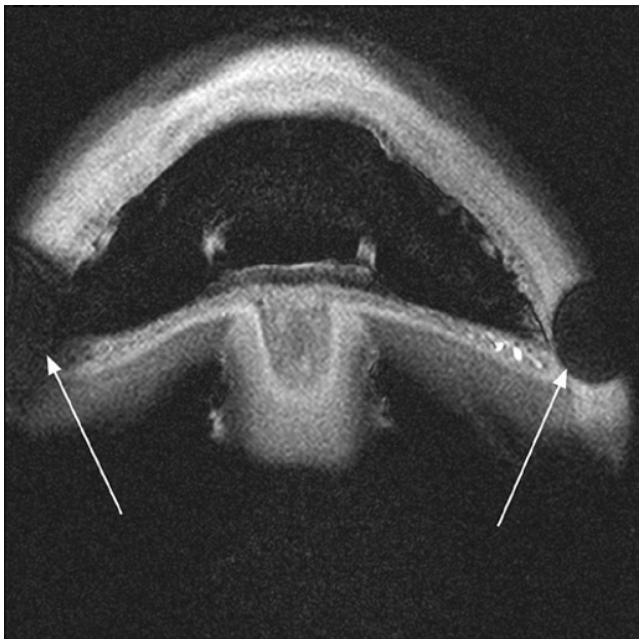


Figure 3.211. Transverse T1-weighted fast low-angle shot (FLASH) image with fat saturation of a foot at the level of the insertion of the deep digital flexor tendon to the distal phalanx. The presence of small amounts of metallic debris in nail holes has resulted in areas of zero signal and image distortion around the ferrous material (arrows).

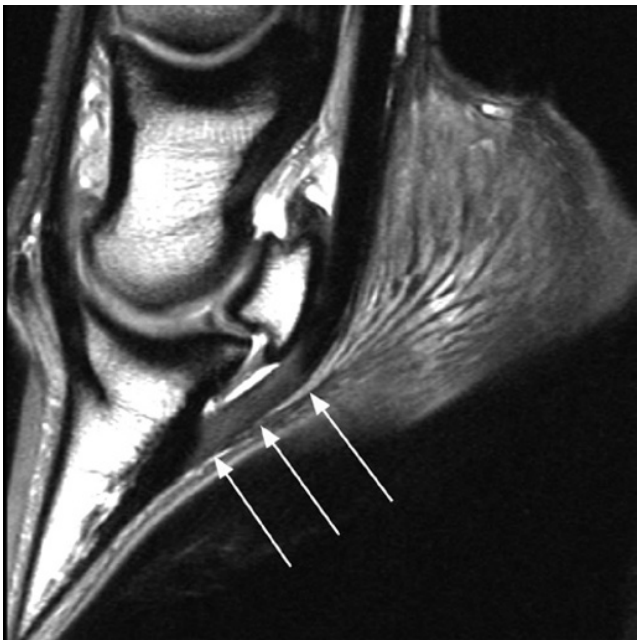


Figure 3.212. Sagittal proton density image of the central part of the foot of a forelimb. The magic angle effect causes an abrupt increase in signal intensity of the deep digital flexor tendon from the distal border of the navicular bone distally to its insertion to the distal phalanx (arrows).

Partial volume averaging artifacts occur when the different signal intensities of more than one tissue type within the same tissue slice are digitally averaged, resulting in a misleading shade of gray. This artifact results in blurring of the margins of structures, disappearing of small structures, and image inaccuracies. Any structure with an edge or curved surface crossing multiple voxels or small structures that partially cross or cut between two slices are susceptible to the volume averaging effect. For example, it may be impossible to distinguish the margin of articular cartilage when imaging the articular surfaces of the equine metacarpophalangeal joints (Figure 3.213). For curved articular surfaces, the artifact can be reduced by positioning individual slices perpendicular to the articular surface at multiple points.

Phase wrap artifacts occur when a portion of the object that lies outside the field of view is represented out of position in the image. Gibbs truncation artifacts or Gibbs ringing artifacts are seen when lines of bright signal are repeated parallel with an interface of abrupt signal change between two objects of markedly different signal intensity in the image. Temperature artifacts occur especially in inversion recovery sequences because the optimal inversion time for suppression of fat or fluid signal is affected by temperature. Abnormal temperature changes in tissues because of inflammation, alterations in vascular flow, or frozen storage may therefore result in insufficiently suppressed fat signal and give a false impression of pathology.[2,21,22] RF interference or zipper artifacts occur when electromagnetic energy from outside the magnet (e.g. electronic equipment in the scan room or an open door) finds access past Faraday's cage. It appears as a line of increased signal of one or two pixels wide through the image.

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Figure 3.213. Sagittal proton density image of the central part of the fetlock of a forelimb. The slice is positioned immediately adjacent to the sagittal ridge of the distal aspect of the third metacarpal bone. The articular cartilage layers of the third metacarpal bone and the proximal phalanx are poorly distinguishable from each other and from adjacent subchondral bone (arrow). The margins are blurred due to volume averaging effect of slices across the curved articular surfaces.

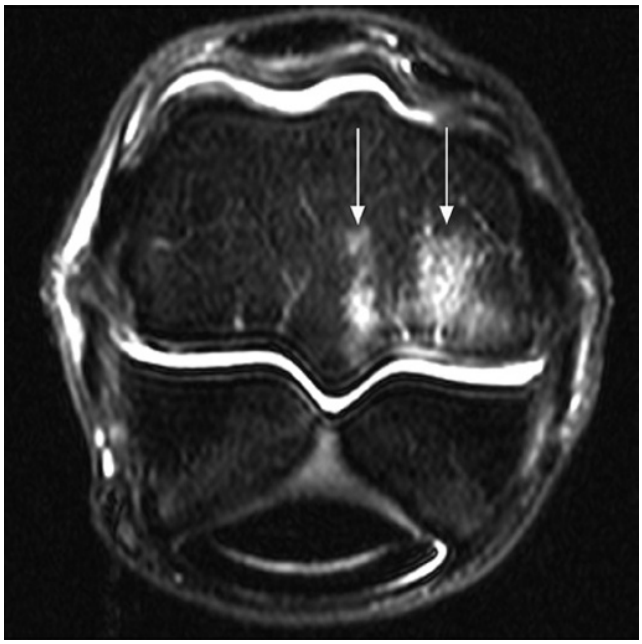


Figure 3.214. Transverse short tau inversion recovery (STIR) image of the distal aspect of the right third metacarpal bone at the level of the proximal sesamoid bones. There are hyperintense areas of abnormal intraosseous fluid in the palmar aspect of the lateral condyle and sagittal ridge of the third metacarpal bone (arrows).

INTERPRETATION OF MUSCULOSKELETAL MR IMAGES

"Signal intensity" describes the shade of gray of a specific tissue on an MR image (Table 3.2). Bright or white signal is described as hyperintense or as having high signal intensity. Dark or black tissues are hypointense or have low signal intensity. Isointense is used as a comparative term for two tissues that have similar signal intensity. Unlike other diagnostic imaging modalities, MRI is unique in that it produces images in which the same tissue may have a different appearance (signal intensity) depending on the sequence used for image acquisition.

Injury results in changes in biochemical composition and fluid content and therefore signal intensity of tissues. Bone sclerosis, increased tissue fluid, and presence of fibrous tissue can be readily detected because of alterations in signal intensity. For a complete assessment of injury, images must be analyzed in all pulse sequences and planes.

In general, fluid is hypointense on T1-weighted and hyperintense on T2-weighted and PD images. Increased protein or cellular content in fluid can lead to increased signal intensity of fluid on T1-weighted images. On T2-weighted images, the presence of blood leads to a relative decrease in signal intensity compared with fluid. Immature granulation tissue has high signal intensity on T2- and T1-weighted images, whereas mature fibrous tissue has low signal intensity on T2-weighted images but can retain higher signal intensity in T1-weighted images.[195]

Normal cortical bone is uniformly black on all sequences and has clearly defined margins. Cancellous bone is more heterogeneous gray because of the presence of adipose and connective tissues in addition to mineralized trabeculae. Bone remodeling can result in changes in bone contour, the presence of cancellous bone fluid and cancellous bone mineral densification. Changes in trabecular architecture are visible as loss in signal homogeneity and thickening or loss of trabeculae. The presence of abnormal fluid in bone is recognized as a combination of high signal on fat-suppressed and T2-weighted images and low signal on T1-weighted images (Figure 3.214). It may also produce a specific fat/water phase cancelation artifact in the standing low-field 0.27 magnet. Although this combination of signal changes is commonly referred to as a bone marrow lesion, bone edema, or bone bruising, it has been shown to reflect a wider range of possible pathologic changes including bone necrosis, hemorrhage, inflammation, replacement of trabeculae by fibrosis, fat necrosis, and bone edema.[148,200] Even so, the ability to detect the presence of fluid in bone makes MRI extremely sensitive in the early diagnosis of bone injury. Generalized osseous fluid with markedly increased STIR signal throughout the entire spongiosa may also be one of the earliest signs of osteomyelitis or may accompany septic synovitis of an adjacent synovial cavity (Figure 3.215).[62,67,191]

Areas of increased density or mineralization of cancellous bone (sclerosis) are characterized by low signal on both T2- and T1-weighted images (figure 3.216). Sclerosis is visible on MR images before it becomes visible radiographically. Damage to bone can also occur at the origin or insertion of ligaments and tendons and

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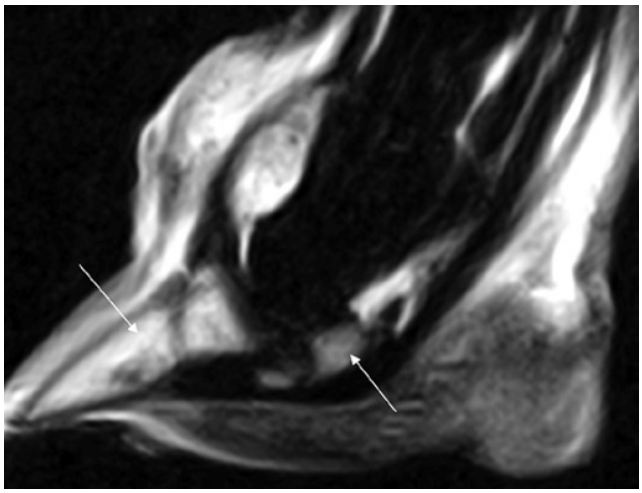


Figure 3.215. Sagittal low-field STIR FSE image of the left front foot of a show jumper with septic synovitis of the distal interphalangeal joint and navicular bursa following a nail puncture of the sole. There is marked and extensive increased signal intensity (osseous fluid) throughout the spongiosa of both the distal phalanx and the navicular bone (arrows). *Source*: Courtesy of Dr. Marieke Zimmerman.

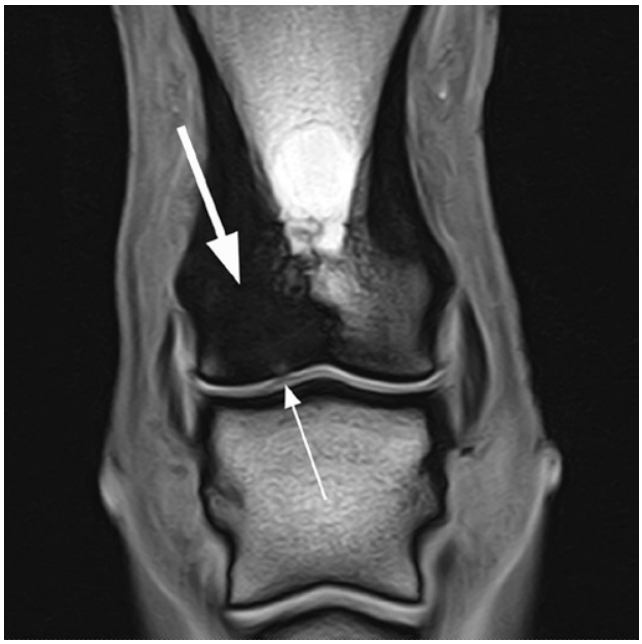


Figure 3.216. Dorsal T1-weighted spoiled gradient echo image of the proximal interphalangeal joint of the right forelimb of a horse with septic arthritis of 5 weeks' duration. There is a small, focal, hyperintense subchondral bone lesion (narrow arrow) surrounded by a wide area of extensive loss of signal from the cancellous bone of the laterodistal aspect of the proximal phalanx (broad arrow). When accompanied by a similar loss of signal in paired T2-weighted images, this indicates increased density or mineralization of cancellous bone (osteosclerosis).

may consist of localized osseous fluid, bone lysis, sclerosis, enthesophyte formation, or endosteal reaction. Occasionally an osseous cyst-like lesion can be observed at the attachment site of a ligament or tendon. Fractures are seen as defects in the bone contour and structure and

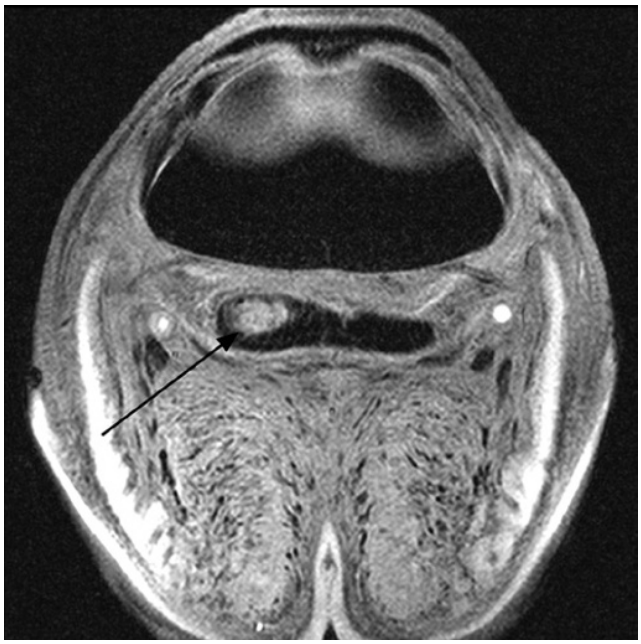


Figure 3.217. Transverse T1-weighted fast low-angle shot (FLASH) image with fat saturation of the right front foot at the level of the middle phalanx of a horse with acute onset foot lameness. There is abnormal signal hyperintensity in a large core lesion of the medial lobe of the deep digital flexor tendon (arrow).

as lines of high signal on T2-weighted and fat-suppressed images. There may be osseous fluid, sclerosis, or both in the parent bone. Incomplete stress fractures may be difficult to discern if they are limited to cortical bone only.[83]

Normal tendons emit zero signal on T1- and T2-weighted images. Normal ligaments produce more signal variation than tendons and vary from light gray to black because they are often composed of fiber bundles with different orientations, which makes them more susceptible to magic angle effects than tendons. The degree of signal variability is dependent on the specific ligament, the density of collagen bundles, and the sequence used for imaging. GRE sequences are inherently more prone the magic angle effect and produce less soft tissue contrast. Therefore T2-weighted FSE sequences should always be included when evaluating ligaments for possible injury.

In general, a localized increase in signal intensity in tendons or ligaments indicates tissue damage. Mild damage may be detected by the presence of periligamentous signal increase on T2-weighted images. More severe damage is characterized by either focal or diffuse intratendinous or intraligamentous increase in signal intensity (Figure 3.217). This may be accompanied by enlargement and shape changes. A partial tear may result in thinning, elongation, or a wavy or partially interrupted contour of a ligament or tendon. With complete tears, the contour is interrupted by a visible defect, and stumps may be present at the ligament or tendon ends. In the acute and subacute stages of tendon or ligament injury, signal increase is present in both T1- and T2-weighted images, due to the presence of fluid associated with acute collagen necrosis and inflammation.

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At later stages of fibrosis and healing, T2 signal progressively returns to normal, while T1 signal hyperintensity persists, often indefinitely.[19,78,88,160] Due to the persistence of signal abnormalities in injured tendons and ligaments, MRI may not be able to establish when a patient with a tendon or ligament injury can safely resume sporting activities. One study found that horses whose hyperintense tendon or ligament lesions had resolved on STIR images were significantly more likely to be sound than horses whose lesions persisted on STIR images.[78] On the other hand, tendon or ligament lesions that were visible only on T1 GRE images remained of uncertain clinical significance as a continued association with lameness was possible. In another study of clinical core lesions in the digital part of the DDFT, tendons with fascicular necrosis had signal increase on fat-suppressed images and T1-weighted images, whereas signal intensity in fat-suppressed images was normal in core lesions with fibroplasia rather than necrosis.[19] Similarly, the presence of increased signal on fat-suppressed images was associated with the most severe histological grades of collagen fiber disruption in one study of human Achilles tendinosis.[86]

Normal articular cartilage has medium signal intensity on PD images, medium to high signal intensity on T1-weighted images, and medium to low signal intensity on T2-weighted images. It can be clearly defined from hypointense subchondral bone and from synovial fluid, which is hypointense on T1-weighted images and hyperintense on T2-weighted images. 3D T1-weighted gradient and spoiled GRE sequences with fat suppression and thin slices (1–2 mm) have been found to be most useful for morphological evaluation, defining margins and thickness of equine articular cartilage in the DIP,[133,149] metacarpophalangeal,[135,136,143] and carpal joints[120] and in the navicular bursa.[159] Cartilage damage can be visualized directly as a change in signal, thickness, and/or surface contour of cartilage. Surface irregularities may indicate the presence of superficial fibrillation, but more substantial lesions (erosions and wear lines) can be visualized as defects in the cartilage surface contour. These cartilage defects allow pooling of synovial fluid, which results in focal T2 and STIR signal hyperintensity and T1 signal hypointensity inside the normal cartilage contour (Figure 3.218). Full-thickness cartilage defects are sometimes (but not always) accompanied by signal changes of subchondral bone, including thickening and irregularity of the borders of the subchondral bone plate and focal bone edema, that may draw attention to the less obvious cartilage signal changes. Although MRI has been reported as a good imaging modality for evaluation of articular cartilage, accurate MRI evaluation of degenerative and traumatic cartilage lesions in distal limb joints of horses can remain difficult,[80,130,136,143,174,175,180] especially in the absence of concurrent subchondral bone abnormalities.[193]

Cartilage imaging difficulties arise mainly from the thickness (lack thereof) of equine distal limb cartilage that is frequently too thin for the spatial resolution of clinical MRI systems. In addition, the articular surfaces of equine distal limb joints are markedly curved. Both factors promote partial volume averaging across image slices resulting in blurring of cartilage margins.



Figure 3.218. Dorsal short tau inversion recovery (STIR) image of the right fore fetlock of a horse with acute right fore fetlock lameness. There is abnormal signal hyperintensity at the level of the articular cartilage proximal to a small subchondral bone irregularity in the proximomedial articular surface of the proximal phalanx (arrow). There is mild signal hyperintensity in the subchondral bone adjacent to the articular cartilage hyperintensity. Necropsy confirmed the presence of a full-thickness cartilage injury at this site.

This artifact can be reduced by the use of thin, high spatial-resolution.[149]

New sequences aimed at improving morphological evaluation of articular cartilage are constantly evolving and under investigation. Even so, recent studies highlight the ongoing difficulties with MRI evaluation of cartilage morphology, even with current high-field systems.[80,95]

Quantitative MRI of articular cartilage aims to identify molecular alteration in the matrix before morphological changes occur.[81,129,130] Techniques like T2 mapping[196] and delayed gadolinium-enhanced MRI of cartilage (dGEMRIC)[15,33,34] have no clinical application in the horse at present but continue to be investigated for future use. Specifically, dGEMRIC[81,98,129] may prove to be helpful as an indicator of early degenerative changes associated with loss of proteoglycans from the extracellular matrix.

Normal synovial fluid has high signal intensity on T2-weighted images and low signal intensity on T1-weighted images. Joint capsule has medium to low signal intensity on all sequences. Joint effusion results in displacement of the joint capsule margins. Increased protein or hemorrhage in the synovial fluid may cause an increase in T1 signal and a decrease in T2 signal intensity of the fluid. Chronic hemarthrosis may result in the presence of small susceptibility artifacts on GRE images due to the presence of hemosiderin in the synovial membrane.

Blood in the lumen of a normal artery or vein may appear as low intensity, high intensity, or mixed intensity signal depending on the imaging parameters and blood

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flow characteristics. Hemorrhage can be differentiated into acute and chronic stages on MR images.[117] In the acute stage, hemorrhage is like other fluids, hypointense on T1-weighted images and hyperintense on T2-weighted images. After approximately one week, methemoglobin release leads to increased T1 signal intensity. In the chronic stage, hemosiderin will cause zero signal areas to appear due to susceptibility artifacts especially on GRE images.[20] Susceptibility artifacts may persist indefinitely at the site of previous hemorrhage due to injection, surgical intervention like arthroscopy, or injury.[181] The center of old hemorrhage may remain hyperintense on T1-weighted and T2-weighted images due to persistence of methemoglobin.

CONTRAST MRI TECHNIQUES

MRI contrast medium was developed to enable differentiation between two tissues with similar MR characteristics by differentially increasing their signal intensity in the presence of injury. Gadolinium is a paramagnetic contrast agent that enhances T1 relaxation and thus shortens T1 relaxation times, resulting in increased signal in tissues containing this element on T1 images.

Post-contrast fat-saturated T1-weighted sequences are obtained following intravenous administration of gadolinium diethylenetriaminepentaacetic acid (0.02 mmol/kg IV)[64] or gadopentetate dimeglumine (0.1 mL/kg IV).[85] Local intravenous delivery of gadopentetate by regional limb perfusion with a tourniquet in the mid-metacarpal region may provide similar results with lower dosages of contrast agent (5 mL gadopentetate diluted with 5 mL of physiological saline per distal limb).[1] Gadopentetate dimeglumine leaks into the extracellular compartment to become detectable on MR images following tissue injury with inflammation and capillary breakdown.[63] In horses' feet, significant contrast enhancement has been observed in tendinopathy lesions of the deep digital flexor, desmopathy lesions of the distal sesamoidean impar ligament and the collateral sesamoidean ligaments, synovitis (also septic) of the DIP joint, and palmar erosions of the flexor cortex of the navicular bone (Figure 3.219).[85,154] In the brain, gadolinium causes significant enhancement of inflammatory and neoplastic lesions because of their increased vascularity and ability to break down the blood–brain barrier.[64,109]

Direct MRI arthrography with a saline–gadopentetate dimeglumine mixture (1.0 mL gadopentetate dimeglumine/250 mL saline; 2–4 mmol/L) has been used in the assessment of complex intra-articular soft tissue structures in human patients. With respect to cartilage imaging, MRI arthrography has reportedly resulted in at least 10% improvement in chondral lesion detection relative to conventional MRI studies.[97,181] Higher concentrations cause signal blackout and nondiagnostic images due to strong magnetic susceptibility effects. Better cartilage contrast may be achieved following direct MRI arthrography with saline than with gadolinium,[35,100,102,159] as saline (hypointense) results in better image contrast between cartilage (medium high to high signal) and synovial fluid than gadolinium (hyperintense) on T1 images.[159] Distension of the navicular bursa or DIP joint with 0.9% saline revealed adhesion formation, DDFT injury, and fibrocartilaginous damage to the navicular bone.[16,100,102]

Indirect MRI arthrography is performed following intravenous injection of paramagnetic MRI contrast media as indicated above. Similar sensitivities and specificities have been reported for indirect MRI arthrography when compared with direct MRI arthrography in the diagnosis of intra-articular soft tissue injuries like tears of the rotator cuff, glenoid labrum, and menisci.[201]

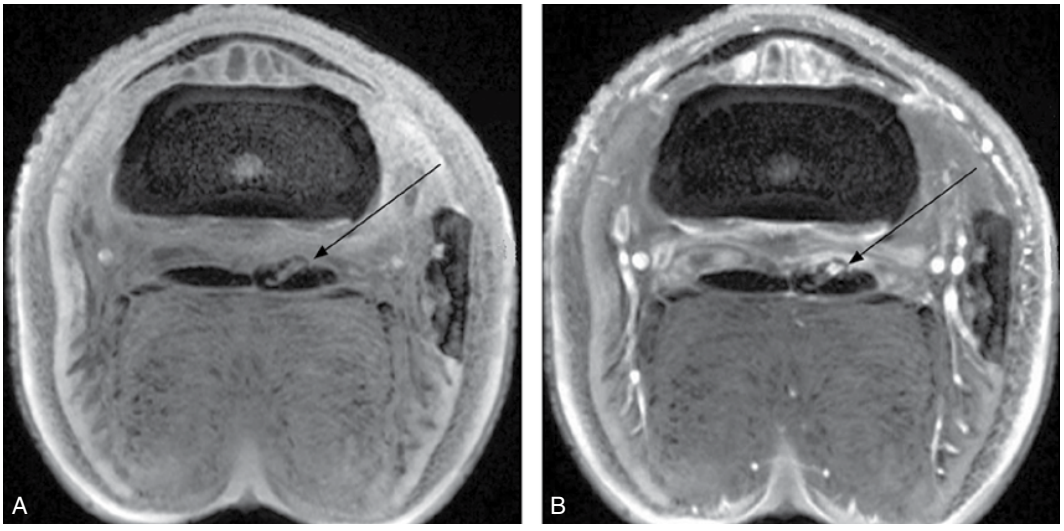


Figure 3.219. Transverse volumetric interpolated breath-hold examination (VIBE) images at the level of the middle phalanx of the right front foot of a horse with chronic foot lameness, before (A) and after (B) intravenous administration of gadolinium contrast material. There is an area of abnormal signal hyperintensity in the lateral lobe of the deep digital flexor tendon at the level of the proximal border of the navicular bursa (arrow). This lesion is enhanced by the administration of intravenous contrast as the relative signal intensity of the core lesion is increased in Figure B (arrow). Source: Courtesy of Dr. Carter Judy.

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Since cartilage has limited ability to repair itself, it is useful to determine its biochemical composition early on in clinical disease. One study showed that naturally occurring osteoarthritis significantly affects cartilage relaxation times as determined by dGEMRIC in the DIP joints of horses.[15]

HOW TO READ AN EQUINE MRI STUDY

A standard MRI protocol produces hundreds of high-detail images, each of which may contain important information. In order to turn this information into a useful diagnosis, a methodical and consistent approach should be used to reading the MRI study. Any suspect abnormality should be defined by signal intensity, size, shape, and contour in comparison with normal. Throughout the evaluation process the clinician should cross reference any suspected signal abnormality in two ways, i.e. by anatomical cross referencing and by contrast cross referencing. Anatomical referencing is performed by comparing the appearance of a suspected lesion with its appearance in other image planes of the same contrast weighting. Contrast cross referencing refers to the process of comparing the signal intensity of a suspected lesion in a PD or T1-weighted image with its signal intensity in the corresponding T2-weighted and fat-suppressed images. As a general rule, an abnormality should be identifiable in at least two different imaging planes and two different contrast weightings for it to be considered a "true" lesion. If an abnormality can only be seen in one pulse sequence in one orientation, then there is a high likelihood that the finding is an artifact.

Frequently more than one "true" lesion is identified during an MRI examination, causing a diagnostic dilemma, as not all apparent abnormalities are equally associated with pain and lameness and "normal" signal variation may occur. The clinician needs to decide on the likely hierarchy of clinical significance of lesions encountered, in light of the clinical exam findings and current knowledge of which MRI lesions are most common.

INDICATIONS, CASE SELECTION, ADVANTAGES, AND DISADVANTAGES OF MRI

MRI is indicated when a lameness problem has been localized to an anatomical area and other imaging modalities have failed to provide an unequivocal diagnosis. Lameness should first be localized to an anatomical area because unlike nuclear scintigraphy, MRI is not a screening technique. Accurate knowledge of the localization of the cause of lameness, as well as the pitfalls encountered with diagnostic analgesia is indispensable when interpreting MR images. These rules apply, even in the face of newer, more powerful 3 T magnets with faster scanning times now allowing routine screening of the foot, pastern, and fetlock regions in horses with distal limb lameness with only short general anesthesia times.

MRI is particularly useful in anatomical areas where conventional imaging modalities have limitations, like the foot, the palmar/plantar soft tissues, and the joints of the distal limbs. Ultrasonography is limited by the hoof capsule in lameness of the foot. Ultrasonographic findings may be equivocal in lameness associated with the palmar/plantar soft tissues of the distal limb. Radiography is incapable of detecting subtle or early cartilage or subchondral bone abnormalities in joint lameness. Radiographic abnormalities are also absent in many forms of osseous trauma (e.g. bone contusions, bone edema, occult subchondral bone loss). Although MRI is the only imaging modality that can assess all tissues in a single examination, the availability of MRI should not result in the omission of radiographic and ultrasonographic examinations. MRI should not replace but rather complement radiographic and ultrasonographic findings. Information obtained from radiographic, ultrasonographic, and scintigraphic examinations helps in the interpretation of MRI findings and a full set of diagnostic images always provides a better basis for an accurate diagnosis. Radiography in particular has better bone versus soft tissue contrast when compared with MRI and is therefore more sensitive to subtle bone contour changes like osteophytes, enthesophytes, and small osteochondral fragments. Ultrasonography gives a better representation of fiber pattern in tendons and ligaments and is not plagued by magic angle and flow artifacts that occur in structures like the suspensory ligament branches or distal sesamoidean ligaments, especially during standing MRI.

There are numerous advantages of MRI over other imaging modalities. MRI does not use ionizing radiation. It has high intrinsic contrast and resolution, particularly for soft tissues, resulting in good anatomic separation between different tissues. Next to anatomic information, MRI also displays information that is pathophysiologic. As a 3D cross-sectional imaging modality, MRI is able to scan an object in any image plane.

The main disadvantages of MRI are its cost (installation and running costs), its still limited availability, its limited accessibility mostly restricted to comfortable examination of the distal limbs for standing magnets, its poor suitability as a screening technique (unlike CT), the need for general anesthesia with high-field magnets, the relatively lower tissue signal and interference of patient movement with low-field magnets, and the need for dedicated specialist training. Image quality can be influenced by many different parameters, including time, signal-to-noise ratio, size of the object of interest, slice thickness, field of view, and other imaging specifications. In addition, MRI gives rise to a number of unfamiliar imaging artifacts that may mimic the presence of lesions or render a scan nondiagnostic. It is important to know how signal characteristics are influenced by all of the abovementioned parameters, so that the clinician can assure high image quality. The large number of images generated with each study also makes interpretation time consuming.

MAGNETIC RESONANCE IMAGING OF THE FOOT AND PASTERN

Introduction

MRI has become the gold standard of diagnosis in horses with foot lameness, because of its higher sensitivity and specificity than radiography, ultrasonography,

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and scintigraphy for lesions responsible for lameness, resulting in a more accurate, early diagnosis and prognosis and allowing for the selection of the most appropriate specific treatment protocol. Many of the causes of foot lameness we know today thanks to the use of MRI, can only be identified conclusively with MRI.

One study, using cadaver limbs of horses with distal limb lameness, found that anatomical structures appeared similar in both high-field and low-field systems but that the margins of some structures were less clearly defined with low-field MRI likely due to partial volume effect.[122] It was concluded that the level of detection of moderate to severe lesions in the foot was similar between high- and low-field systems. However, small tendon, articular cartilage, ligament, and bone lesions were better detected using a high-field system.[122]

There have been several reports on the incidence of injuries diagnosed with both low- and high-field MRI systems in horses with foot lameness. Some of these[24,46,48,75,103,113,157,168,179] are summarized in Table 3.3. Overall, injury of the DDFT was the most prevalent lesion, while navicular bone lesions were the next most common diagnosis followed by injuries of the collateral ligaments of the DIP joint. Abnormalities in the DIP joint and navicular bursa were diagnosed frequently in low-field studies but less commonly in high-field studies. Injuries to all other structures occurred with markedly lower frequency, generally in less than 10% of horses. The absence of significant abnormalities was mentioned in up to 14% of horses in four studies in which an MRI diagnosis was not made. Although MRI is the most sensitive and specific diagnostic modality for the diagnosis of injuries of a3l tissues in the horse's foot,[121] not all causes of foot lameness can be readily identified (Table 3.3).

MRI Abnormalities in the Foot

Tendinopathy of the DDFT

The normal DDFT can be clearly recognized as a well-delineated, bilobed, double elliptical structure of homogeneous low signal (black) with a hyperintense midline septum. From the navicular bone distally, the midline septum is no longer visible, and the tendon becomes progressively flatter to finish in a crescent-shaped distal insertion on the distal phalanx. A generalized signal increase in the portion of the tendon comprised between the distal border of the navicular bone and its insertion to the distal phalanx can be caused by the magic angle effect (Figure 3.212). The appearance of the normal DDFT has a strong left to right limb as well as medial to lateral lobe symmetry in both signal intensity and cross-sectional area measurements.[119] The tendon contains a dorsal layer of high T1 and PD signal both at the level of the navicular bone and proximal to the navicular bone.[17,159]

Tendon lesions are characterized by focal or linear, marginal or central, intratendinous signal increase on both T1 and T2 images, variably accompanied by some enlargement of the affected lobe in the acute stage of injury.[43] Lesions may occur distal to the navicular bone at the insertion on the distal phalanx, at the level of the flexor surface of the navicular bone, at the level of the collateral sesamoidean and T-ligaments in the proximal recess of the navicular bursa, or at any combination of these three levels at the same time. Lesions are usually restricted to one lobe but may change their morphology while transitioning from one level to another. There is a good correlation between the MRI appearance of tendon lesions and their pathological classification into core lesions (Figure 3.220), parasagittal plane splits and tears (Figure 3.221), insertional lesions (Figure 3.222), and dorsal surface erosions and fibrillations (Figure 3.223).[18,32,121,156,158] Generalized T1 signal increase in one tendon lobe may constitute an artifact rather than diffuse tendinopathy.[177] Insertional enthesopathy can result in cortical irregularity, caused by focal bone resorption or enthesophyte formation at the insertion of the tendon to the flexor surface of the distal phalanx, and in diffuse osseous signal change in the palmar aspect of the distal phalanx, caused by osseous fluid or sclerosis. Dorsal lesions may be accompanied by adhesions between the dorsal surface of the tendon and the palmar surface of the collateral sesamoidean and impar ligaments within the navicular bursa. Torn collagen fibers arising from the tendon's damaged dorsal surface may recoil proximally in the navicular bursa, resulting in the dorsal protrusion of a tendon granuloma into the proximal recess of the navicular bursa.[170] Adhesions are suspected when one or more bands of hypointense (T2) or hyperintense (T1) material are present between the dorsal surface of the DDFT and the collateral sesamoidean ligaments, obliterating the normal synovial fluid signal in this space. Sometimes lack of separation extends across the entire surface of both structures in the proximal recess of the navicular bursa. Other signs of navicular bursitis accompanying lesions of the dorsal surface of the intrabursal portion of the tendon include fluid distension of the proximolateral and proximomedial pouches of the bursa (proximolateral pouch is always larger) and thickening and proliferation of the bursal synovium in the proximal recess of the navicular bursa. Thickening of the collateral sesamoidean and distal impar ligaments may also be seen. Resorptive lesions of the flexor cortex of the navicular bone are frequently accompanied by fibrous adhesions between the eroded bone surface and the dorsal surface of the tendon. Unfortunately, adhesion detection is difficult in the absence of normal synovial fluid volume between the opposing structures in the navicular bursa, as may occur in chronic bursitis with synovial proliferation. Distension of the navicular bursa or DIP joint with 4–6 mL of 0.9% saline is useful as it should cause separation of the tendon from the collateral sesamoidean ligaments and the navicular bone if no adhesions are present.[100,102,159] Lack of separation after injection is indicative of fibrous adhesions between adjacent structures.

It may be possible to use T1-to-T2 signal differences to estimate the age or stage of healing of a tendon lesion.[160] Early tendinopathy has been characterized by increased signal in T2, STIR, and T1 images. In the chronic stages of tendon healing by fibrosis, signal intensity in core lesions generally decreases in T2 images but remains high in T1 and PD images.[160]

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Table 3.3. Incidence of MRI findings in seven retrospective MRI studies of foot lameness (Mair et al.,[107] Sherlock et al.,[164] Dyson et al.,[54] Dyson and Murray,[49] Boswell et al.,[24] Mitchel et al.,[113] Schramme and Redding,[157] Gutierrez-Nibeyro et al.,[76] and Stockl et al.[179])

MRI lesions % total incidence	*N* = 35	*N* = 41	*N* = 199[a]	*N* = 347[a]	*N* = 170	*N* = 98	*N* = 172[a]	*N* = 79	*N* = 120[b]
DDFT tendinopathy	49	29	60	48	52	64	30	54	43
Navicular bone disease	31	32	19	29	8	77	50	78	69
CL desmopathy DIP joint	6	7	31	43	23	21	16	39	12
Navicular bursitis	—	17	—	—	—	49	1	57	60
Collateral sesamoidean desmopathy	14	10	—	10	—	13	8	24	4
Distal sesamoidean impar desmopathy	9	—	10	15	1	4	6	30	15
DIP joint synovitis/OA	23	37	3	2	9	68	9	53	47
Bone bruising middle or distal phalanx	—	5	7	4	6	2	3	24	—
Distal sesamoidean desmopathy	6	—	1	2	1	—	2	—	—
Distal digital annular desmopathy	—	—	—	—	—	—	4	14	—
Ungular cartilages								10	
Multiple injuries	NS	15	17	33	4	NS	24	94	17
No abnormalities detected	14	12	NS	NS	6	NS	10	NS	6

DDFT, deep digital flexor tendon; CL, collateral ligament; DIP, distal interphalangeal; OA, osteoarthritis; and NS, not specified.
The total incidence of each lesion is represented, rather than only the incidence of lesions considered as the primary cause of lameness. Several horses had simultaneous incidence of more than one lesion.
[a] High-field MRI system.
[b] Incidence in both lame and contralateral limbs.

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Figure 3.220. Transverse T1-weighted fast low-angle shot (FLASH) image with fat saturation of the right front foot at the level of the middle phalanx of a horse with acute onset foot lameness. The lateral lobe of the DDFT is enlarged and contains a core lesion characterized by a central, circular area of signal hyperintensity (arrow) surrounded by normal, low intensity, tendon signal.

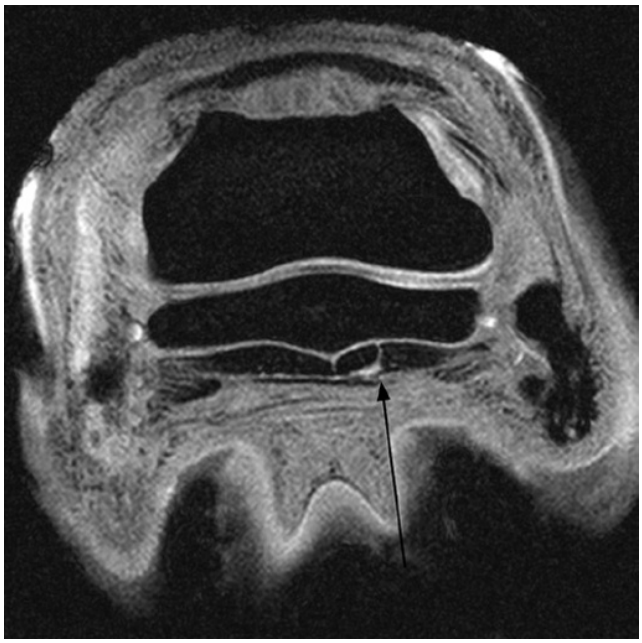


Figure 3.221. Transverse fast low-angle shot (FLASH) image with fat saturation of the right foot at the level of the navicular bone of a horse with acute onset foot lameness. A linear signal hyperintensity arises from a defect in the dorsal surface of the deep digital flexor tendon and progresses in a dorsopalmar direction, resulting in a full-thickness parasagittal split of the lateral lobe (black arrow).

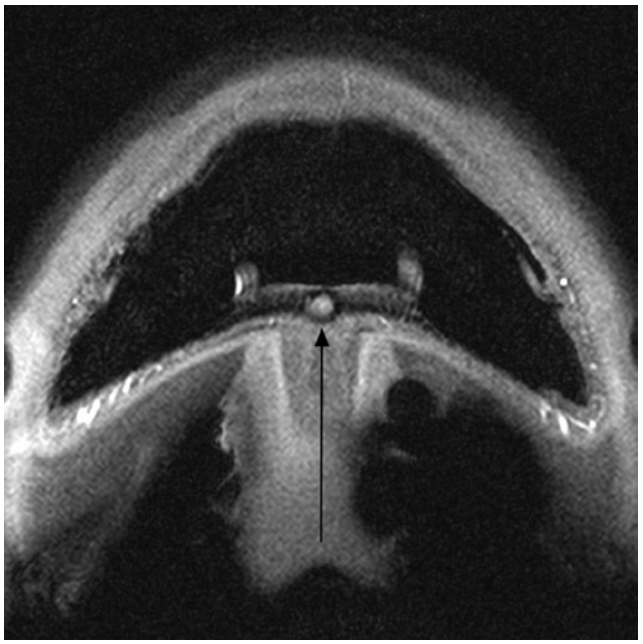


Figure 3.222. Transverse fast low-angle shot (FLASH) image with fat saturation of a foot at the level of the insertion of the deep digital flexor tendon to the distal phalanx of a horse with foot lameness. A hyperintense core lesion is present in the central part of the tendon (arrow) and extends distally to the bone–tendon interface.

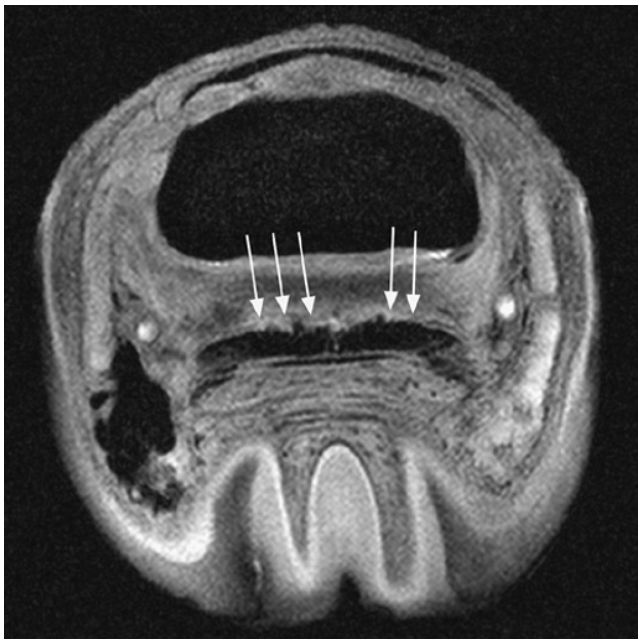


Figure 3.223. Transverse fast low-angle shot (FLASH) image with fat saturation of the foot at the level of the middle phalanx of a horse with foot lameness. The dorsal surface of the deep digital flexor tendon is irregular due to the presence of fibrillations and short, incomplete sagittal splits disrupting the smooth dorsal contour of the tendon (arrows).

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Lesions of the Navicular Bone

MR images of the navicular bone in horses with navicular bone disease may show one or more of three main abnormalities: remodeling changes in the medulla, degenerative changes of the flexor border, and osteochondral fragmentation of the distal border.[49] The most common type of abnormality seen in the navicular bones of horses with recent onset navicular syndrome was STIR signal hyperintensity in the medullary cavity of the navicular bone with or without additional areas of T2 and PD signal hypointensity[152] (Figure 3.224), but this STIR signal increase is more reliably assessed on FSE than on GRE images.[138] It has been speculated that osseous fluid in the medulla may be an acute inflammatory or post-traumatic finding in horses with recent onset navicular syndrome.[152] However, in pathological studies, MRI evidence of "bone marrow lesion" was associated not only with acute inflammation but also with evidence of hemorrhage, interstitial edema in medullary fat, prominent capillary infiltration in marrow fat, thinning of trabeculae and widening of intertrabecular spaces, medullary fibrosis, chronic osteonecrosis, and, in some horses, adipose tissue necrosis.[17,32,58] In one pathological study, all but one horse with medullary STIR signal hyperintensity had concurrent chronic degenerative changes of the flexor surface of the navicular bone.[158] On the other hand, pathological studies have not included horses with recent onset lameness, and it therefore remains possible that medullary STIR hyperintensity can represent acute inflammatory fluid in the spongiosa of the navicular bone.[152] This possibility is supported by the frequent observation of osseous fluid signal in the spongiosa of the navicular bone without concurrent abnormalities of the distal border or flexor surface on MR images. In such horses, it is difficult to differentiate between abnormal osseous fluid of an acute post-traumatic nature and osseous fluid accumulation associated with a chronic degenerative process on a single examination. As acute, post-traumatic osseous fluid (e.g. a bone contusion) tends to resolve with rest, in contrast to the persistence of degenerative osseous fluid, this distinction requires one or more follow-up MRI examinations to be performed.[190]



Figure 3.224. Sagittal short tau inversion recovery (STIR) image of the foot of a horse with lameness that is abolished by anesthesia of the palmar digital nerves. There is marked STIR hyperintensity of cancellous bone in the medullary cavity of the navicular bone (arrow) indicating the presence of abnormal medullary fluid, medullary fibrosis, or medullary fat necrosis.

Medullary STIR signal hyperintensity may be focal near the distal or palmar border of the navicular bone, or extend from the distal border in a vertical band along the palmar cortex to the proximal border of the bone, or spread diffusely throughout the medullary cavity. Based on the extent and intensity of STIR signal increase, medullary bone edema can be graded from mild to severe. Lower grades of medullary STIR signal hyperintensity have been encountered in non-lame control limbs, but severe medullary edema was strongly associated with the presence of lameness.[121,152] Areas of signal hypointensity may also be seen in the medullary cavity of the navicular bone and may be focal or diffuse. Loss of T1 signal may either represent replacement of medullary fat by fluid or bone densification, while loss of T2 and PD signal indicates the presence of bone densification and sclerosis or even replacement of medullary trabecular bone by compact lamellar bone, usually as a consequence of marked trabecular thickening in response to degenerative changes of the flexor surface of the navicular bone.[4,158]

In horses with chronic navicular syndrome, the most common MRI abnormality was the presence of abnormal signal hyperintensity at the level of the flexor border of the navicular bone (Figure 3.225).[158] This can be a subtle, focal increase, caused by synovial fluid pooling at a site of palmar fibrocartilage loss and thinning, best seen on sagittal PD or T2-weighted images. MRI bursography with saline has been shown to improve the conspicuity of fibrocartilage lesions.[159]

A normal shallow, smooth depression is present in the middle third of the palmar sagittal ridge of up to 50% of normal navicular bones.[197] This is also characterized by pooling of synovial bursal fluid but should not be confused with a degenerative lesion of the palmar fibrocartilage. Even so, this normal synovial depression may be the site of early bone degeneration in some horses with navicular bone disease. The presence of osseous fluid signal on STIR images within the flexor cortex and spongiosa adjacent to the synovial depression in the sagittal ridge is a likely sign of early bone degeneration. Signal increase at the flexor surface can also be more extensive and extend deeper within the cortical bone of the flexor cortex when cortical bone erosion is present (Figure 3.225). Focal bone loss from the flexor surface is best seen on fat-suppressed images in high-field studies and transverse high-resolution T1 images on low-field standing MR images. These flexor cortex lesions may not be easily detected radiographically.[165] In affected horses, MR images usually show concurrent irregularity of the normally smooth endosteal surface of the flexor cortex, especially opposite the site of focal signal increase of the palmar surface. Fibrocartilage and cortical bone loss at the palmar aspect of the flexor surface of the navicular bone is

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Figure 3.225. Sagittal proton density image of the foot of a horse with chronic navicular bone degeneration. There is localized signal hyperintensity in the distal third of the flexor border of the navicular bone due to loss of cortical bone and the presence of fibrous tissue and bursal synovial fluid in the defect (arrow). There is diffuse loss of signal in the medullary cavity of the navicular bone due to osteosclerosis. There is loss of separation between the palmar surfaces of the thickened collateral sesamoidean and distal sesamoidean impar ligaments on the one hand and the dorsal surface of the deep digital flexor tendon on the other, suggestive of abnormal adherence between these structures.

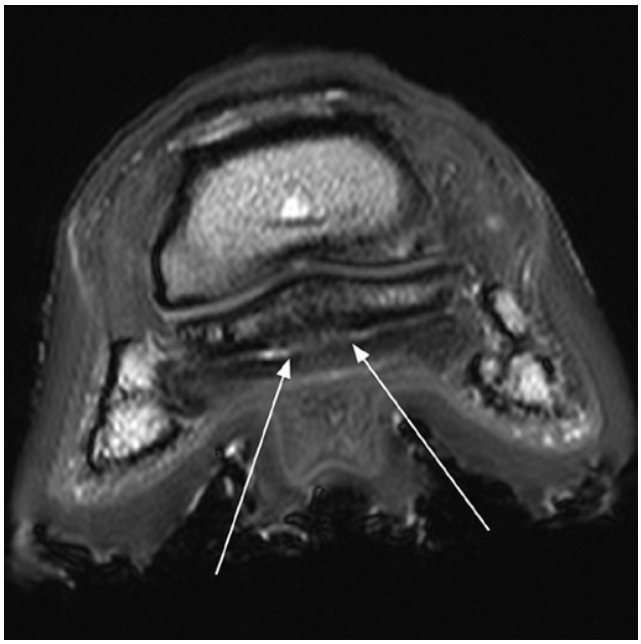


Figure 3.226. Transverse T2* gradient echo image of the foot of a horse with chronic navicular bone disease. The palmar border of the navicular bone is irregular due to degenerative erosion of the flexor cortex. There is sclerosis of the medullary cavity of the navicular bone. The normal hyperintense synovial fluid layer separating the palmar border of the navicular bone from the dorsal surface of the deep digital flexor tendon is interrupted by hypointense fibrous adhesions between both surfaces (arrows).

frequently accompanied by fibrillation of the dorsal aspect of the DDFT. Areas of advanced fibrocartilage and cortical bone loss from the flexor surface of the navicular bone are prone to adhesion formation between the dorsal surface of the DDFT and the exposed area of bone (Figure 3.226).

Another form of degenerative navicular bone disease may be caused by the presence of osseous fragments at the distal border of the navicular bone (Figure 3.227).[8–10,50] Especially large fragments may induce a focal remodeling response in the distal margin of the navicular bone, resulting in localized osseous fluid with STIR signal hyperintensity, irregular thickening of cortical bone, and formation of an osseous cyst-like lesion.[9] A distal border fragment accompanied by focal medullary bone edema, an osseous cyst-like lesion, or thickened cortical bone at the distal border of the navicular bone is likely to be associated with lameness. Even so, distal border fragments may be asymptomatic,[198] especially when they are smaller and if there is no MRI evidence of remodeling of the distal border of the navicular bone adjacent to the fragment.

In horses with a complete navicular bone fracture, MRI can demonstrate the fracture configuration on 3D images and elucidate if fracture fragments have lacerated the surface of the DDFT, prior to possible attempts at surgical repair. Fractures must be distinguished from bipartite navicular bones for the purpose of prognosis and treatment planning.[76]

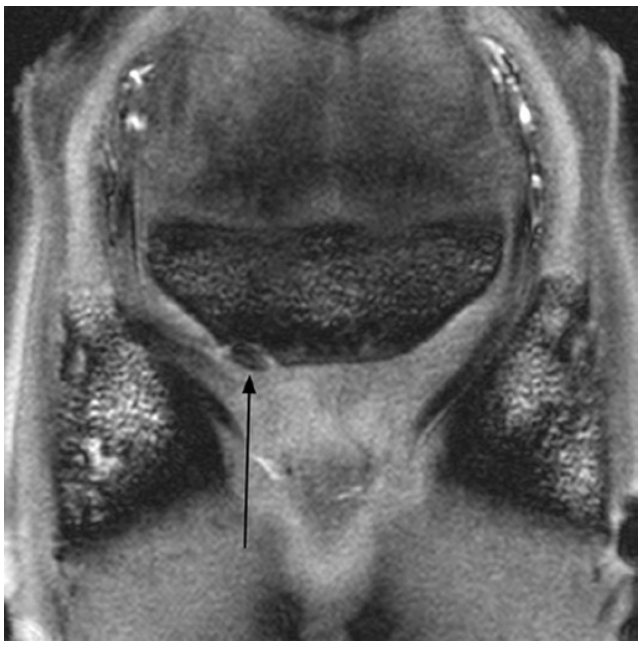


Figure 3.227. Dorsal T1-weighted fast low-angle shot (FLASH) image at the level of the navicular bone, oriented parallel with its flexor surface. The narrow slice thickness and orientation of this sequence allow for identification of an osseous fragment at the lateral angle of the distal border of the navicular bone (arrow).

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Lesions of the Distal Sesamoidean Impar Ligament

The distal sesamoidean impar ligament is outlined by hyperintense fluid signal from the DIP joint dorsally and the distal recess of the navicular bursa distally. It is composed of individual fiber bundles with synovial interdigitations of the DIP joint and small branches of the palmar digital arteries. The heterogeneous structure with multiple fluid–fiber interfaces in this small ligament results in a high susceptibility to partial volume averaging. Therefore, focal high signal within the impar ligament must be interpreted with caution because of normal signal variation. One study found no significant correlation between the MRI appearance of the impar ligament itself and the presence of histological abnormalities.[50] Moreover, the ligament is thicker and its palmar border more intimately apposed to the dorsal border of the DDFT axially than abaxially, which may confound identification of ligament thickening and adhesion formation. A diagnosis of desmitis of this ligament is rare and rarely considered the primary cause of lameness, except in the presence of distal border fragments or cystic lesions in the distal border of the navicular bone.[50]

Unequivocal MRI signs of impar ligament injury include marked thickening, extensive adhesion of the palmar surface of the ligament to the dorsal surface of the DDFT with loss of normal fluid space in the distal recess of the navicular bursa, and osseous signal abnormalities at the insertion of the ligament to the distal phalanx. The latter include focally increased fluid signal, increased mineralization, entheseous new bone production, and focal osteolysis with formation of a cyst-like lesion (Figure 3.228).[199] Generalized thickening of the impar ligament and adhesions between the impar ligament and the DDFT occur most commonly in association with navicular bursitis accompanying marked pathology of the DDFT or the navicular bone and may be an integral part of secondary generalized bursal inflammation.

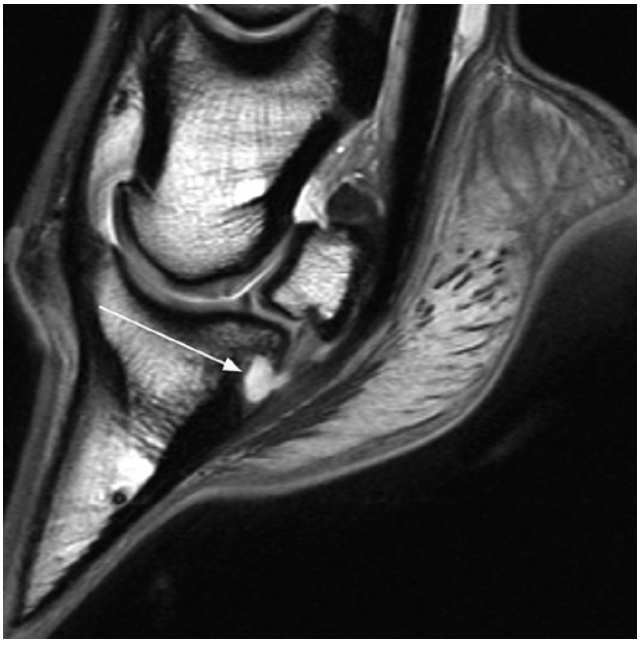


Figure 3.228. Sagittal proton density image of the central part of the foot of a horse with chronic foot lameness. There is localized signal hyperintensity in an osseous cyst-like lesion at the insertion of the distal sesamoidean impar ligament to the distal phalanx indicating chronic enthesopathy of this ligament (arrow).

Lesions of the Collateral Sesamoidean Ligaments

It has been reported that the paired collateral sesamoidean ligaments have uniform low intensity signal in all image sequences and are symmetrical in thickness medially and laterally.[152] However, the presence of signal increase is a common normal variation on all contrast weightings, especially near the insertion to the proximal border of the navicular bone.[17,176] The dorsal and palmar borders of the ligaments are clearly demarcated by high fluid signal in the palmar recess of the DIP joint and the proximal recess of the navicular bursa.

Collateral sesamoidean ligament injury may be evident as an altered shape with focal or diffuse signal increase in the body of the ligament. Asymmetric thickening is most easily identified when the paired ligament is normal (Figure 3.229). Generalized thickening of the ligament with loss of separation from the dorsal surface of the DDFT, possibly with adhesions, occurs most commonly in association with chronic navicular bursitis secondary to tendinopathy or degenerative navicular bone disease.

Primary collateral sesamoidean ligament injury is rare except in one report of horses with hindfoot lameness.[11]

Lesions of the Navicular Bursa

Pooling of hyperintense fluid can normally be observed in the proximolateral and proximomedial pouches of the navicular bursa and to a lesser extent between the DDFT and the distal impar ligament distally. There should be visible separation between the dorsal surface of the DDFT and the palmar surfaces of the collateral and distal impar sesamoidean ligaments in normal navicular bursae, apart from the areas of attachment between these structures by the normal proximal and distal synovial membrane reflections in the sagittal midline of the navicular bursa.

Navicular bursitis results in effusion with enlargement of the proximolateral and proximomedial pouches of the bursa and sometimes in dorsal deviation of the central part of the collateral sesamoidean ligaments or the distal impar ligament. The proximolateral recess of an inflamed navicular bursa is always more distended than the proximomedial recess. Fibrous scar tissue can occur in the proximal and distal recesses of the bursa and is indicative of chronic bursitis. Lack of separation between the dorsal surface of the DDFT and the palmar surface of the collateral sesamoidean ligaments may be suggestive of adhesion formation.[79] Simple distension of the navicular bursa is a frequent nonspecific finding in many horses irrespective of the primary injury site.[46] It is rarely considered the primary cause of lameness.[152]

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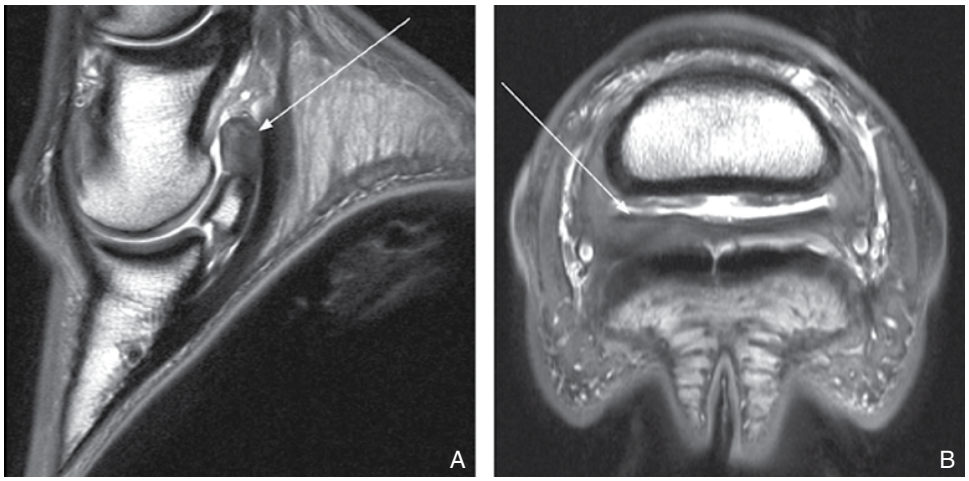


Figure 3.229. Sagittal proton density image lateral to the sagittal midline (A) and transverse proton density image at the level of the proximal aspect of the navicular bursa (B) of the left foot of a horse with chronic lameness that can be eliminated by anesthesia of the palmar digital nerves. The lateral collateral sesamoidean ligament contains areas of increased signal intensity and is markedly thickened (arrows) in comparison with the medial ligament. Swelling has resulted in loss of separation between the palmar surface of the lateral collateral sesamoidean ligament and the dorsal surface of the lateral lobe of the deep digital flexor tendon. The presence of adherence between both structures cannot be ruled out.

Lesions of the Collateral Ligaments of the DIP Joint

Normal collateral ligaments can be seen as well-delineated elliptical structures of homogeneous to mildly heterogeneous signal with smooth osseous margins at the origin and insertion on most transverse MR images. Focal or diffuse signal increase in collateral ligaments is a common sequel of magic angle artifact, especially in T1, T2*, and PD images and results in a high incidence of signal variation in these structures.[74,171,177,178,192] The lateral collateral ligament is most commonly affected by this artifact in standing horses. The fiber arrangement and curvature within the periphery of the proximal segments of the collateral ligament may give rise to a central region of nonuniform low signal intensity with a rim of intermediate to high signal intensity at the level of the middle phalanx due to magic angle effect that can be confused with a desmopathy on both low- and high-field images.[90,192] Therefore, suspected signal changes in a collateral ligament must always be evaluated in transverse T2-weighted FSE images, if possible with a long echo time, in order to verify if signal increase is truly caused by tissue damage and not by magic angle artifact. High signal on a T1-weighted GRE sequence that is not accompanied by high signal on the corresponding T2-weighted and STIR images is not a reliable indicator of injury.

On dorsal images obtained in an image plane parallel with the direction of the collateral ligaments and perpendicular to the solar surface of the foot, the collateral ligaments appear as curved, banana-shaped bands of homogeneous, low T2 signal.

Lateromedial size and shape differences between paired ligaments are possible due to anatomical variation and adaptive change. Signal asymmetry at the proximal aspect of the collateral ligaments may also occur due to uneven length or thickness of collateral ligaments.[44,53,117]

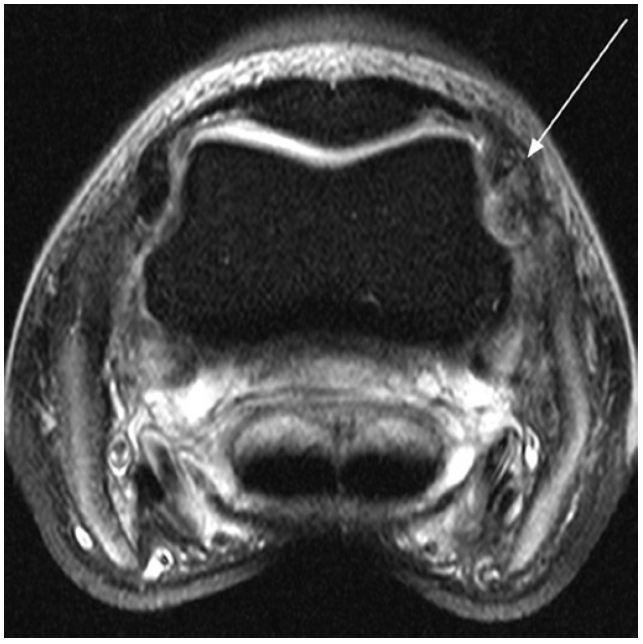


Figure 3.230. Transverse proton density image with fat saturation oriented parallel with the solar surface of the foot of a horse with collateral ligament injury of the distal interphalangeal joint. The affected collateral ligament is enlarged, and its margins are irregular (arrow). There is loss of architecture, and irregular areas of signal hyperintensity are dispersed throughout the cross section of the ligament (arrow).

Desmopathy is characterized by increased cross-sectional area, irregular contour, and increased signal intensity of the collateral ligament (Figure 3.230).[44,73] The medial collateral ligament is more frequently affected than the lateral.[44,53] Abnormal signal increase can be diffuse or focal and is best recognized on transverse T2 FSE

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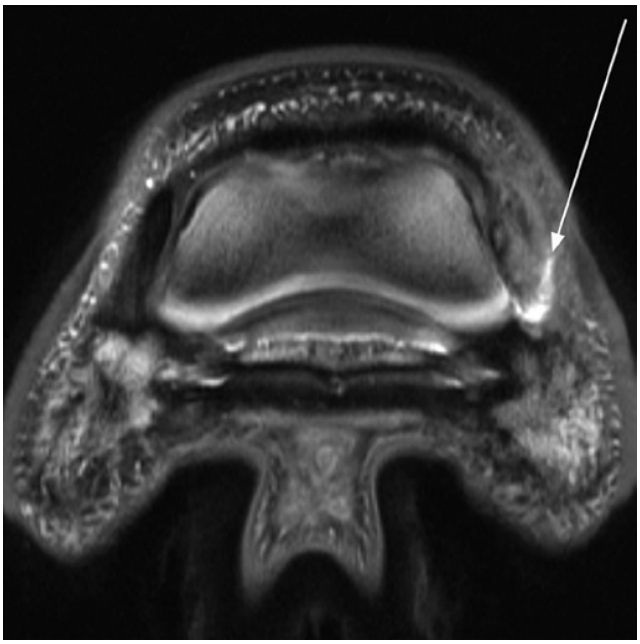


Figure 3.231. Oblique transverse proton density image of the foot of a horse with collateral ligament injury of the distal interphalangeal joint. Slice direction is perpendicular to the flexor surface of the navicular bone. There is pooling of hyperintense synovial fluid in a defect resulting from avulsion of collateral ligament fibers from their insertion on the distal phalanx (arrow). There is enlargement, loss of architecture, increased signal intensity, and loss of margination of the affected ligament. The hyperintense fluid outlines the distal stump of the torn collateral ligament (arrow).



Figure 3.232. Sagittal short tau inversion recovery (STIR) image of the central part of the right foot of a horse with lameness that can be abolished with intra-articular anesthesia of the distal interphalangeal joint. There is a focal signal hyperintensity within the hyaline cartilage layer of the distal articular surface of the middle phalanx (arrow). This focal signal increase is caused by pooling of synovial fluid within an articular cartilage defect. An oblique full-thickness cartilage cleft was identified at necropsy.

or STIR images. Desmopathy may be accompanied by thickening of periligamentar structures, one of which is the chondrocoronal ligament. Osseous damage at the origin or, more commonly, at the insertion of a collateral ligament causes signal alteration in the adjacent bone. In 45% of cases of collateral ligament disease, osseous abnormalities were identified on MRI.[39] These osseous abnormalities can consist of entheseous new bone, cortical and endosteal irregularities or defects, osseous cyst-like lesions, and increased mineralization or osseous fluid occupying cancellous bone in the palmar process adjacent to the insertion of the collateral ligament. Partial or complete avulsion of a collateral ligament from its insertion on the distal phalanx results in pooling of hyperintense joint fluid in the defect, outlining the distal stump of the torn collateral ligament (Figure 3.231). Collateral desmitis of the DIP joint may occur in conjunction with ossification of the collateral cartilages.[55,56,105] Osteoarthritis or misalignment of the DIP joint caused by subluxation associated with collateral ligament injury may occur but are rare. Marked widening of the joint space may indicate joint instability associated with severe collateral ligament injury, but this sign should be assessed with caution as asymmetric weight-bearing or positioning of the limb also results in an asymmetric joint space.

Due to the highly common normal signal variation, it may be difficult to establish the clinical significance of mild signal changes in collateral ligaments.

Lesions of the DIP Joint

The presence of joint distension can be readily deduced from enlargement of the dorsal and palmaroproximal outpouchings of the DIP joint with fluid. Distension may cause palmar deviation of the axial part of the collateral sesamoidean ligaments. Distension of the dorsal joint pouch is generally more prominent in standing horses than in recumbent horses. Distension of the DIP joint is a frequent nonspecific finding in many horses irrespective of the primary injury site.[46,152]

The DIP joint usually has smoothly curved articular surfaces that make it difficult to assess the articular cartilage accurately due to partial volume averaging, so that focal cartilage defects may be missed. Thinner slice thickness is helpful in reducing this artifact and improving cartilage definition. Dorsal and sagittal T1 high spatial resolution slices are used for cartilage assessment with the 0.27T low-field magnet on standing horses.[149] On these high-resolution images, the articular cartilage layers are clearly distinguishable as two distinct layers of high signal separated by a thin line of intermediate signal representing synovial fluid in the joint space.[133,149] Careful slice-per-slice comparison with the contralateral normal limb is necessary.

Generalized cartilage disease in osteoarthritis may be observed as loss of cartilage surface definition, loss of visual distinction between the two cartilage layers, cartilage surface irregularity, or generalized thinning of the

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articular cartilage layer. The resulting narrowing of the joint space width may be an early sign of osteoarthritis,[149] but this sign is only reliable if the horse is either bearing weight symmetrically in a low-field magnet or the foot is supported with the limb held straight in a high-field magnet. In MRI of standing horses bearing weight evenly, generalized loss of articular cartilage may result in malalignment between the middle and distal phalanges.[149]

Focal cartilage lesions are recognized as focal increase (T2, PD, or STIR images) or decrease (T1) in signal intensity caused by pooling of synovial fluid in a cartilage defect (Figure 3.232). Not all focal cartilage defects cause lameness, and many focal cartilage defects occur without signal alteration in the adjacent subchondral bone.[179] Nonetheless, altered thickness of the subchondral bone plate, increased STIR signal in subchondral bone, and endosteal irregularity may be useful indicators for the presence of an adjacent area of cartilage damage.

Focal osseous cyst-like lesions confluent with an articular cartilage and subchondral bone defect and containing increased T1, T2, and STIR signal can be present in the central part or close to the palmar border of the weight-bearing surface of the distal phalanx, where they cannot be detected radiographically. There may be a variable amount of osseous fluid in the trabecular bone of the distal phalanx peripheral to the osseous cyst-like lesion. Small osseous cyst-like lesions or focal depressions in the subchondral bone with a corresponding cartilage defect may occasionally be seen in the central portion of the sagittal midline groove of the distal articular surface of the middle phalanx. These lesions are frequently an incidental finding, unrelated to foot lameness.

Osteophytes may be visible as small spur formations on the extensor process of the distal phalanx, the palmar margin of the middle phalanx, and the dorsoproximal border of the navicular bone. They are most easily identified on T2* images, but radiographs are generally more sensitive for the identification of osteophytes than MR images.[149]

Septic arthritis can cause specific MRI abnormalities that may allow early and accurate diagnosis of synovial sepsis.[62,67,191] Most striking is generalized diffuse STIR signal increase in the distal phalanx, middle phalanx, and/or navicular bone. Other changes include heterogeneous synovial fluid signal, capsular thickening, synovial proliferation, cartilage loss, focal subchondral bone lysis, and early sequestrum formation. Post-gadolinium imaging may highlight fibrin deposition and result in synovial membrane enhancement.

Lesions of the Distal and Middle Phalanges

Osseous trauma to the phalanges results in more or less extensive osseous fluid. Signal intensity may initially also be high on T2-weighted sequences and gradually diminishes as reactive mineralization (sclerosis) occurs in the area of the bone bruise. There is usually associated increase in radiopharmaceutical uptake on scintigraphic images. Bone bruises of the phalanges occur mostly in the region of the palmar process of the distal phalanx[46] or the dorsodistal aspect of the middle phalanx.[134,189] Bone bruises of the palmar processes of the distal phalanx can be associated with irregularity of the cortical margin and disruption of the adjacent laminar architecture. Chronic bone mineral density increase of a palmar process of the distal phalanx may be the result of a chronic bone bruise, or may be associated with collateral ligament injury. Bone bruising of the dorsodistal aspect of the middle phalanx[134,189] (Figure 3.233) usually involves a well-circumscribed area of cancellous bone adjacent to the dorsal half of the DIP joint and can be located axially or abaxially in the middle phalanx. The presence of osseous fluid in this location may be associated with concurrent increased osseous fluid in the spongiosa of the navicular bone.

Osseous fluid in the distal phalanx may also be secondary to primary injuries of the foot like collateral desmopathy, osteoarthritis of the DIP joint, extensive ossification of the cartilages of the foot, osseous cyst-like lesions,[107] and space-occupying lesions.

Generalized osseous fluid in the distal and/or middle phalanges with markedly increased STIR signal throughout the entire spongiosa may also be one of the earliest signs of osteomyelitis (e.g. associated with a puncture wound). However, a similar pattern of generalized osseous fluid in the spongiosa of one or both phalanges may be seen in the presence of severe regional inflammation as caused by a joint flare or septic arthritis of the DIP joint.[62,67,191]

MRI may identify incomplete or complete fractures of the phalanges that are not visible radiographically because the fracture plane does not coincide with the direction of the standard radiographic projections. A predilection site for fractures of the distal phalanx has been described at the base of an ossified ungular cartilage

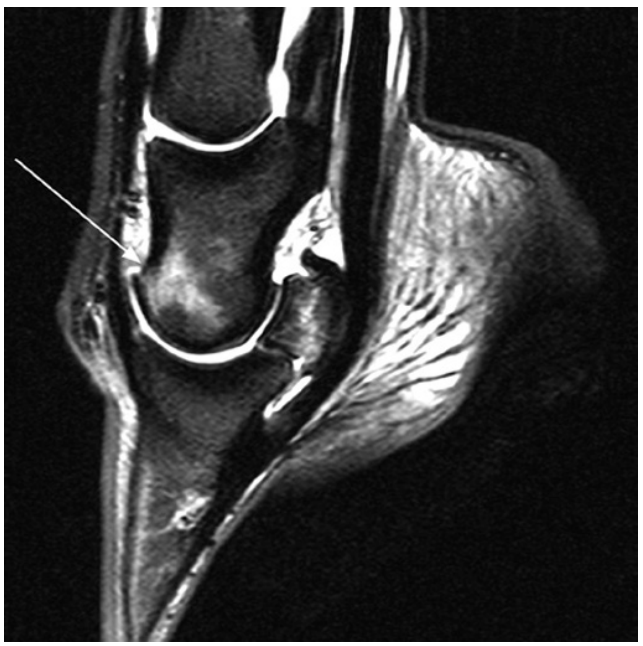


Figure 3.233. Sagittal short tau inversion recovery (STIR) image of the central part of the foot of a horse with acute onset foot lameness. There is an area of marked signal hyperintensity at the dorsodistal aspect of the middle phalanx adjoining the articular surface of the distal interphalangeal joint (white arrow). This appearance is suggestive for the presence of bone edema or a localized bone bruise of the middle phalanx.

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(sidebone).[162] These stress fractures may be incomplete or complete and are usually accompanied by extensively increased bone mineral density (sclerosis) of the palmar process and the ossified ungular cartilage. They are non-articular, simple, and non-displaced fractures but frequently involve the fossa of the collateral ligament of the DIP joint. They have a predictable proximodistal course with a palmar to dorsal and axial to abaxial orientation. Enlargement and signal abnormalities were seen in the ipsilateral chondrocoronal, chondrosesamoidean, collateral sesamoidean, and collateral ligaments of most horses and thought to contribute to the pain causing lameness.[162]

Lesions of the Ungular Cartilages

Primary injuries of unossified ungular cartilages and their related chondrocoronal and chondrosesamoidean ligaments are uncommon. Diagnosis depends on MRI that reveals a markedly thickened, hypervascular cartilage with irregular contours and diffusely increased STIR signal intensity.[45] Occasionally the thickened, inflamed cartilage may appear malaligned and displaced relative to its attachment site on the palmar process of the distal phalanx.

Ossification of one or both cartilages of the foot is usually characterized by continuity of normal bone structure from the proximal portion of the palmar process through the ossified cartilage with a hypointense outer rim of cortical bone surrounding a hyperintense spongiosa. However, trauma to an ossified cartilage may result in increased bone mineral density (sclerosis) combined with focal or diffuse osseous fluid and is characterized by increased STIR signal and reduced T1 and T2 signal either at the base of the ossified cartilage or at the junction between two separate centers of ossification. Increased mineralization may also be an incidental finding but usually then without osseous fluid. Trauma to an ossified cartilage may be accompanied by thickening and signal abnormalities in the chondrosesamoidean and chondrocoronal ligaments. Collateral desmitis of the DIP joint may also occur in conjunction with severe ossification of the collateral cartilages.[45,55,105]

Fracture results in a clear linear hyperintensity at the base of an ossified cartilage surrounded by osseous modeling, including osseous fluid, although clear differentiation between trauma to the junction between separate centers of ossification and a fracture is not always easy.[55,162]

Quittor or infection of an ungular cartilage is characterized by thickening of the infected cartilage and increased STIR signal. There may be evidence of fluid tracts or fluid pooling with focal high or heterogeneous T2 and STIR signal intensity within the cartilage and surrounding soft tissues. MRI examination may also reveal variable involvement of other soft tissue and osseous structures of the foot in addition to infection of the ungular cartilages. Preoperative MRI was shown to facilitate and guide the surgical approach to and postoperative management of quittor in patients.[112]

Lesions of the Hoof Wall and Space Occupying Lesions

MRI has revealed soft tissue changes of the dorsal hoof wall in acute and chronic phases of laminitis.[3,72,118] High-field MR images (4.7T) of the developmental stage of laminitis were able to show loss of normal corium architecture, loss of lamellar architecture, increased signal intensity in the dermis including in the lamellae, and focal areas of separation between the epidermal and dermal lamellae in the stratum lamellatum, resulting in an increased ratio (>0.7) between the width of the stratum lamellatum and the total dermal width (stratum lamellatum + stratum reticulare).[3] Similar but more pronounced findings were seen in chronic laminitis due to displacement of the distal phalanx with more obvious separation between dermal and epidermal lamellae and the formation of a triangular wedge of lamellar regeneration.[89,118] Additional findings were circumscribed areas in the hoof wall of low intensity representing gas, circumscribed areas of high signal intensity representing abscess formation, irregularity of the cortical outline of the distal phalanx, increased osseous fluid palmar to the dorsal cortex of the distal phalanx, and increased size and number of vascular channels in the distal phalanx.[89,118]

Mild focal disruption of interlamellar alignment or focally increased dermal fluid may be seen as an incidental finding not related to lameness. Focal or linear hemosiderin accumulation in the dermis from old trauma may cause a signal void.

Space-occupying masses in the stratum medium or lamellatum of the hoof wall, containing a variable amount of keratin, may be a cause of lameness or an incidental finding on MR images. MRI reveals a smoothly demarcated hoof wall lesion replacing the normal architecture of the dermis of the hoof wall or sole with deformity of the adjacent surface of the distal phalanx. The signal characteristics of these space-occupying masses vary from an area void of signal or with hypointense signal on any sequence to an area of heterogeneous mixed signal intensity in T1- and T2*-weighted images and hypointensity in STIR sequences (Figure 3.234).[101,104,106] This signal variation is likely to reflect the degree of keratinization in the mass. While true keratomas usually result in a well-circumscribed hypointense circular area protruding into the dermis and osseous defect, the signal intensity of granulomas and fibrous or fibrovascular masses tends to be more heterogeneous and mixed.[7,101,106] Adjacent intermediate or high STIR signal intensity in the trabecular bone of the distal phalanx is indicative of reactive inflammatory osteitis. The tomographic slicing produced by MRI has been useful for precise anatomical localization and surgical planning of hoof wall mass removal.[68,106]

Puncture Wounds of the Sole

MRI has been shown to be an excellent modality for diagnosing penetrating foot injury in the horse.[20,92] Injuries from penetrating foreign objects in the foot often result in focal areas of hypointense signal traversing the soft tissues on the palmar aspect of the foot in all image sequences. Signal hypointensity of these tracts is explained in acute stages as a signal void due to air or ferrous material and in chronic stages due to hemosiderin,[20] which may persist indefinitely as a sign of previous penetrating injury. In one large, retrospective study of horses with synovial sepsis following a penetrating injury, MRI was able to identify injury to the DDFT, the

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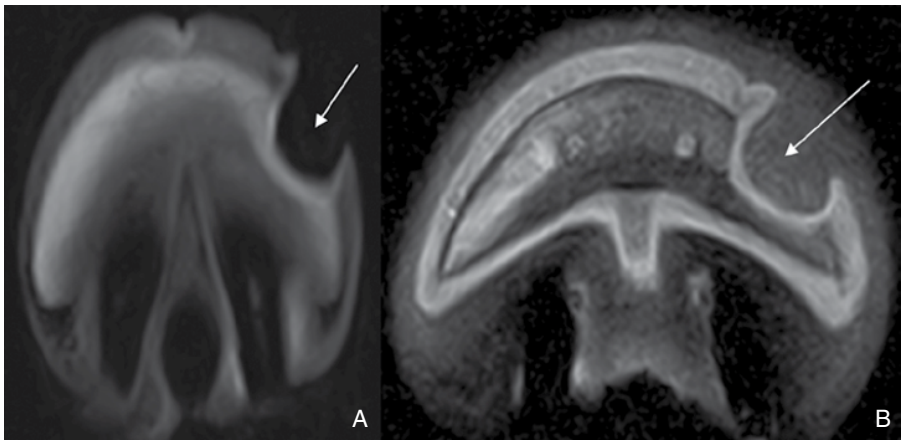


Figure 3.234. Low-field transverse horizontal T2 FSE (A) and transverse oblique T1 GRE (B) images of a horse with a keratoma of the hoof wall. There is an extensive circular area of T2 signal void (left arrow) and T1 signal hypointensity intruding from the hoof wall into the dermis and corresponding large but smooth osseous defect (right arrow).

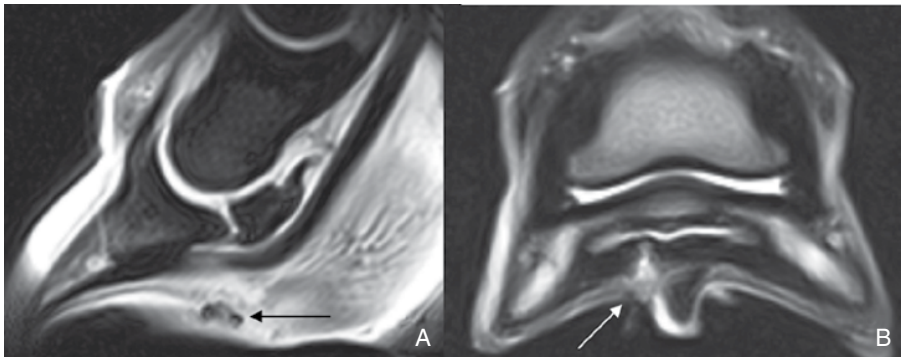


Figure 3.235. Low-field sagittal T2* GRE (A) and dorsal T2 FSE (B) images of horse with a nail puncture of the lateral sulcus of the frog. There are susceptibility artifacts in the frog tissue caused by either hemosiderin or metallic debris in the penetrating tract (black arrow). Linear T2 signal hyperintensity suggests there is fluid in the tract that continues into a parasagittal breach of the lateral lobe of the deep digital flexor tendon (white arrow).

navicular bone, and the DIP joint.[65] MRI not only allows assessment of tract direction and depth but is the only imaging technique that permits early recognition of tendon disease (Figure 3.235), osseous injury, and involvement of surrounding soft tissue structures. Another study highlighted the importance of using T2 FSE sequences to look at tendon boundaries and determine tendon involvement because signal voids created by ferrous material or hemosiderin can preclude full assessment of the tendon on GRE sequences.[155,181]

Generalized osseous fluid in the distal phalanx and navicular bone with markedly increased STIR signal throughout the spongiosa of these bones may be one of the earliest signs of synovial sepsis of the navicular bursa and/or DIP joint or of osteomyelitis.[191] This appearance of generalized "bone edema" should alert the clinician to the likelihood of penetration of at least one synovial cavity (Figure 3.215).

Gadolinium contrast fistulography may be useful to improve the identification of fistulous tracts in the foot.[66]

Lesions of the Proximal Interphalangeal Joint

Lesions in the proximal interphalangeal joint are rare. Occasionally focal subchondral bone damage with osteolysis and associated osseous fluid may be a cause of lameness. Osseous cyst-like lesions may be an incidental finding. Osteophytes may be visible at the joint margins, but radiography is generally more sensitive for this finding of osteoarthritis than MRI.

Lesions of the Digital Annular Ligaments

Isolated cases of desmopathy of the distal and proximal digital annular ligaments,[36] the axial and abaxial palmar ligaments of the proximal interphalangeal joint, the proximal ligament of the digital cushion, the chondrocompedal ligament, and the chondrosesamoidean ligament have also been observed. MRI characteristics of desmopathy are focal or diffuse thickening, focal or diffuse areas of signal hyperintensity within the ligament at the level of the proximal interphalangeal joint, and occasionally adhesion formation.[36]

Lesions that are Poorly Detectable with MRI

Some tissue abnormalities in the foot do not show up well on MR images. Abnormalities of the hoof and sensitive laminae of the solar and heel regions of the foot may not result in obvious signal abnormalities. Although some marked subsolar abscesses or bruises may result in dermal and osseous signal increase in the solar region of the foot, many horses with solar pain responsive to application of hoof testers have unremarkable foot MR images. Similarly, many horses with poor dorsopalmar foot balance resulting in palmar heel pain produce unremarkable MRI scans. Early hyaline or fibrocartilage degeneration of the DIP joint or navicular flexor surface as well as mild fibrillation of the dorsal surface of the DDFT in the navicular bursa may also be difficult to identify.[158]

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MAGNETIC RESONANCE IMAGING OF THE FETLOCK REGION

Introduction

Conventional imaging techniques have limitations in the fetlock region. Radiography and scintigraphy are not capable of detecting early cartilage loss and subchondral bone injury without marked structural bone damage or demineralization.[54,166,202] Ultrasonographic evaluation lacks sensitivity for injuries of the suspensory branches and the straight and oblique distal sesamoidean ligaments.[152,172] Hence MRI is being used routinely to diagnose the causes of lameness in the fetlock region of horses. The primary indication for MRI is pain localized to the fetlock region with diagnostic analgesia, without sufficient radiological or ultrasonographic abnormalities to explain the degree of lameness. The increasingly frequent use of MRI to elucidate distal limb lameness has revealed that lameness caused by pain in the fetlock region may be abolished not only by an abaxial sesamoid nerve block in the majority of horses[35,51] but also, though less frequently, by a palmar digital nerve block.[37] Unfortunately, this regularly causes confusion as to which region of the distal limb should be evaluated first with MRI in horses that are presented with a history of lameness resolution following either of these nerve blocks. Experience with such patients teaches that horses presented with a history of a negative response to a palmar digital nerve block but a positive response to an abaxial sesamoid nerve block are much more likely to have a primary fetlock lesion than a foot lesion responsible for the lameness.[35]

The normal MRI anatomy of the fetlock joint has been described, using both high- and low-field images (MRI abnormalities in the fetlock region).[173] Several studies have documented the incidence of MRI diagnoses in the fetlock region (Table 3.4).[70,90,144] Types and incidence of lesions in the fetlock differ depending on horse breed and athletic activity. In a study of 40 horses from a variety of breeds and disciplines, half the horses

Table 3.4. Frequency of MRI abnormalities in three retrospective studies of horses with lameness caused by pain localized to the fetlock region (Gonzalez et al.,[71] King et al.,[91] and Powell[144]).

MRI lesions % incidence	$N = 40$[a]	$N = 232$[a]	$N = 131$[a,b]
Subchondral bone injury	47.5	13	71.5 (55 POD; 11.5 DOD; 4 P1)
Distal sesamoidean desmitis	32	52 (31 ODSL; 21 SDSL)	4 (3 ODSL; 1 CDSL)
OA and cartilage injury	20	9	—
Suspensory branch desmitis	20	8	4
Osteochondral fragmentation	18	2	2
Proximal sesamoid bone injury	18	—	5
Intersesamoidean desmitis	10	3	—
DDFT tendonitis within DFTS	10	1	—
Collateral desmitis	7.5	7	—
SDFT tendonitis	5	2	—
Enostosis-like lesion	5	—	—
Palmar annular desmitis	2	3	—
Proximal digital annular desmitis	2	—	—
Dystrophic mineralization of LDET	2	—	—
Short incomplete P1 fractures	—	1	14.5
Incomplete MCIII/MTIII condylar fissures/fractures	—	—	20

OA, osteoarthritis; DDFT, deep digital flexor tendon; DFTS, digital flexor tendon sheath; AL, accessory ligament; SDFT, superficial digital flexor tendon; LDET, long digital extensor tendon; P1, proximal phalanx; MCIII, third metacarpal bone; MTIII, third metatarsal bone; POD, palmar osteochondral disease; DOD, dorsal osteochondral disease; ODSL, oblique distal sesamoidean ligament; SDSL, straight distal sesamoidean ligament; and CDSL, cruciate distal sesamoidean ligament.

Several horses had simultaneous incidence of more than one lesion.

[a] High-field MRI system.

[b] Racing Thoroughbreds only.

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had subchondral bone abnormalities of the distal metacarpus and one third lesions of the distal sesamoidean ligaments.[70] However, in a different MRI study of 232 horses, also from a variety of breeds and disciplines, subchondral bone abnormalities were very uncommon, while more than half the horses were lame because of distal sesamoidean ligament injuries.[90] On the other hand, an MRI study of 131 racing Thoroughbreds with fetlock lameness reported almost exclusively osseous injuries, including subchondral bone trauma and incomplete stress fractures.[144] In both mixed breed studies,[70,90] distal sesamoidean ligament desmitis was the most common soft tissue injury, while other soft tissue injuries of the suspensory branches, collateral ligaments, intersesamoidean ligaments, and flexor tendons also occurred but with much lower frequency. Cartilage lesions, osteoarthritis, and osteochondral lesions were also reported. Combinations of injuries were common in the fetlock region, with 63% of horses in one study being identified with multiple MRI abnormalities.[70] Most common was the combination between subchondral bone lesions and injuries involving components of the suspensory apparatus.[70] Abnormalities of the subchondral bone and lesions of the distal sesamoidean ligaments have also been documented in other MRI reports.[54,151,172] Although subchondral bone injuries have been diagnosed with standing low-field MRI,[166] soft tissue abnormalities are much less commonly identified using standing low-field MRI.[144] The most common MRI diagnosis in racing Thoroughbreds was palmar/plantar osteochondral disease (POD) of the MCIII/MTIII, present in more than half of the horses examined.[144] Fractures were also much more commonly diagnosed on MR images of the fetlock of racing Thoroughbreds compared with other disciplines.[140,141,144,183] Soft tissue injuries were identified in less than 10% of racehorses with fetlock lameness,[144] although another more recent study found evidence of periarticular soft tissue injuries in 21% of racing Thoroughbreds and 37% of racing Standardbreds, but these were rarely considered the main cause of lameness (Table 3.4).[139]

MRI Abnormalities in the Fetlock Region

Subchondral Bone Abnormalities of the Metacarpal/Metatarsal Condyles

The normal subchondral bone thickness of the distal aspect of the MCIII/MTIII varies from dorsal to palmar and from abaxial to axial, being thinnest axially and thickest in the middle of each condyle, especially toward the palmar aspect.[47] Subchondral bone thickness of the distal aspect of the MCIII/MTIII is likely to change as it adapts to the type of exercise the horse performs. The subchondral bone thickness of the proximal phalanx increases slightly toward the palmar aspect of each condyle. There is reasonable lateromedial symmetry in subchondral bone thickness of both the MCIII/MTIII and the proximal phalanx. The increased use of MRI has highlighted the importance and high prevalence of bone sclerosis and bone marrow lesions in subchondral bone injury in the fetlock of horses.[42,51,69,139]

Abnormal MRI signal in POD lesions of the condyles of the MCIII/MTIII is manifested as diffuse or focal signal increase in fat-suppressed images consistent with bone marrow lesion or bruising and diffuse T1, PD, and T2 signal decrease consistent with trabecular thickening and osteosclerosis (Figure 3.236). Osseous fluid appears more commonly in the acute stage of injury, while

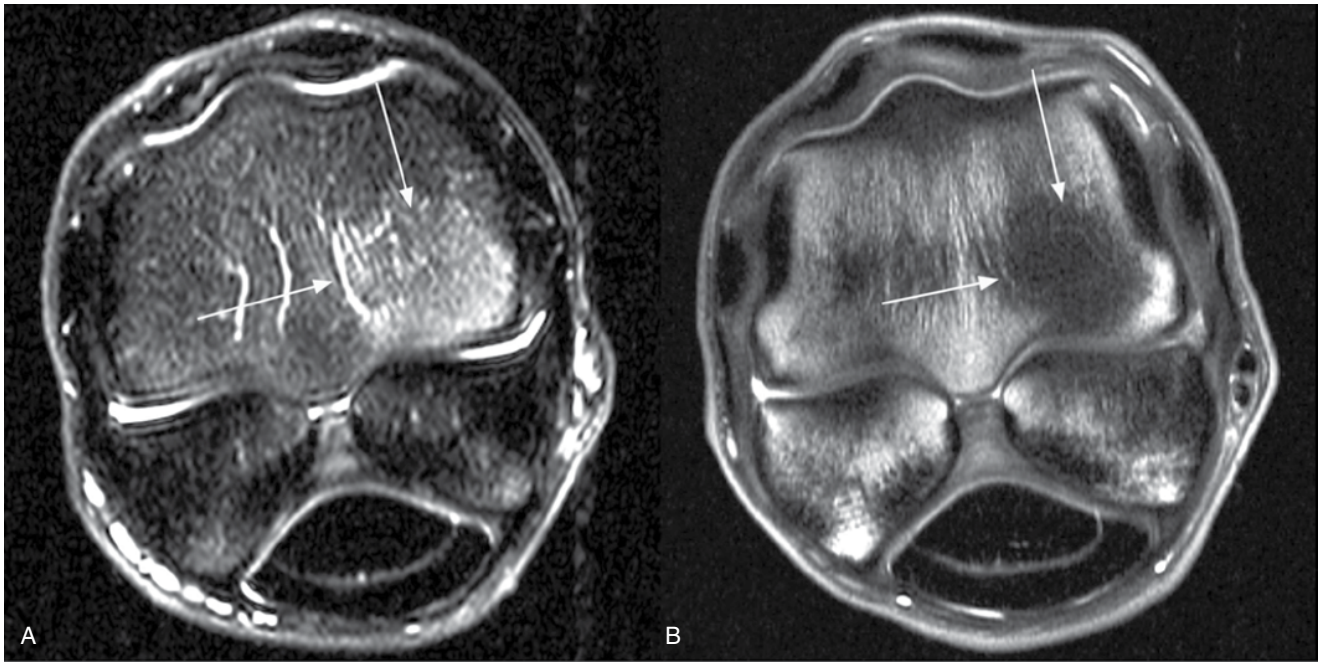


Figure 3.236. Transverse short tau recovery (STIR) image (A) and transverse proton density image (B) at the level of the proximal sesamoid bones of the right fore metacarpophalangeal joint of two different 3-year-old racehorses with fetlock lameness and abnormal MR signal in the palmar aspect of the lateral condyle of the third metacarpal bone. STIR signal hyperintensity suggestive of abnormal bone fluid is present in Figure A (arrows), while an area of signal hypointensity indicates the presence of palmar osteosclerosis in Figure B (arrows).

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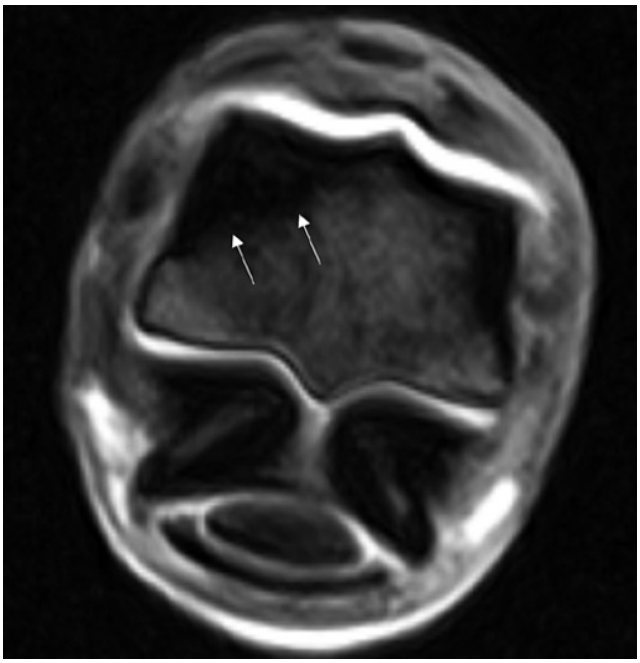


Figure 3.237. Transverse low-field T2* GRE image of the right metacarpophalangeal joint of an 9-year-old Warmblood. There is broad triangular area of low signal intensity containing a central zone of mixed signal intensity in the dorsal half of the medial condyle (arrows) of the third metacarpal bone, an appearance characteristic of dorsal osteochondral disease with mixed osseous fluid and sclerosis.

sclerosis reflects more chronic bone damage with reactive remodeling. Focal signal hyperintensity on all sequences within the sclerotic subchondral bone, adjacent to the joint margin of the affected condyle, is caused by trabecular necrosis in the center of the POD lesion.[176] When located close to the articular surface, osteonecrosis may lead to secondary articular cartilage loss, subchondral bone collapse, and joint contour deformity.

Location, severity, and extent of subchondral bone changes in the fetlock differ between Thoroughbreds, Standardbreds, and sport horses.[42,139,144] In racing Thoroughbreds and racing Standardbreds, bone signal changes were predominantly located in the palmar/plantar aspect of the distal condyle of the MCIII/MTIII (POD lesion), most commonly with biaxial distribution.[54,139,144,166,202] An MRI-based grading scale from 0 (normal) to 5 (markedly abnormal) to assess the severity of POD lesions has been proposed recently based on the abovementioned signs of disease caused by high-magnitude cyclical loading.[140,141]

Osseous changes (fluid and/or osteosclerosis) in the dorsomedial aspect of the condyles or dorsal sagittal ridge of the MCIII/MTIII occur much less frequently in racing Thoroughbreds[144] but have a high prevalence in racing Standardbreds[139] and sport horses[42] (Figure 3.237). In sport horses, especially show jumpers, dorsal bone marrow lesions of the metacarpal condyles are very common, though not always related to lameness.[42] Even though the authors did not find an association,[42] it is generally felt that the higher-grade lesions with extensive dorsomedial sclerosis and sometimes severe and extensive osseous fluid are more likely to be clinically significant than lower-grade lesions in this location.

Normal, parallel, linear areas of increased signal intensity on T2 and STIR images and decreased signal intensity on T1 images may be present in the palmar region of the distal metaphysis and epiphysis of the MCIII/MTIII. This "tiger-stripe" appearance is due to hypervascularity or congestion of the nutrient vessels that appear as well-defined hyperintense lines emanating from the palmar aspect of the metaphyseal region coursing in a dorsodistal direction into the condyles of the MCIII/MTIII. An increase in number, size, and conspicuity of these vessels may occur in the presence of chronic remodeling/sclerosis of the distal condyles or capsulitis with distension of the joint.

The ability of MRI to predict which horses are at risk of developing complete condylar or proximal sesamoid bone fractures has been studied extensively.[140,141,147,182,183] Despite conflicting information on a clear association between sclerosis and the ability to predict condylar fracture formation, there was an overall higher grade of osseous fluid (bone marrow lesion) and bone densification (sclerosis) in the condyle of bones with a condylar fracture, than in the condyles of the limbs of control horses.[140,141,182,183] There was also a higher grade of bone marrow lesion in the fractured condyle than in the corresponding condyle of the contralateral limb.[141] Recent studies have emphasized the important role of MRI in early detection of bone marrow lesions (osseous fluid) as a prodromal sign of condylar fracture.[141,144] Short unicortical fissures of the condylar groove have also been recognized as a prodromal pathology of condylar fractures that may be present for weeks to months, and MRI has been shown to be helpful in their early detection.[147]

Osseous Trauma of the Proximal Phalanx

Proximal phalangeal subchondral bone injuries diagnosed on MRI most often involve the sagittal groove area in all types of horses. The MR appearance of sagittal groove injuries has included osseous fluid, bone contusions, osteosclerosis, focal bone loss due to osteonecrosis, short incomplete midsagittal fractures, and osseous cyst-like lesions.[51,69,139,144] It is uncertain whether a continuum exists between these different injuries in nonracehorses as differences in characteristics have been suggested between lesions occupying the dorsal half of the sagittal groove (incomplete fracture) and those located in its center (sagittal groove trauma), even though the latter may also but rarely progress to fracture.[30,51,69,99]

Variable standing low-field MRI characteristics of acute or chronic presentations of sagittal groove trauma in mature Warmblood horses[69] (Figure 3.238) have been described as high signal on fat-suppressed images (osseous fluid), low signal on all sequences (sclerosis), ill-defined subchondral bone loss, focal subchondral bone defects, and short incomplete linear hyperintensity extending through the subchondral bone into the trabecular bone of the sagittal groove. In addition, all horses had abnormal bone mineral densification of the dorsal aspect of the sagittal ridge of the MCIII/MTIII and the majority also had signs of concurrent

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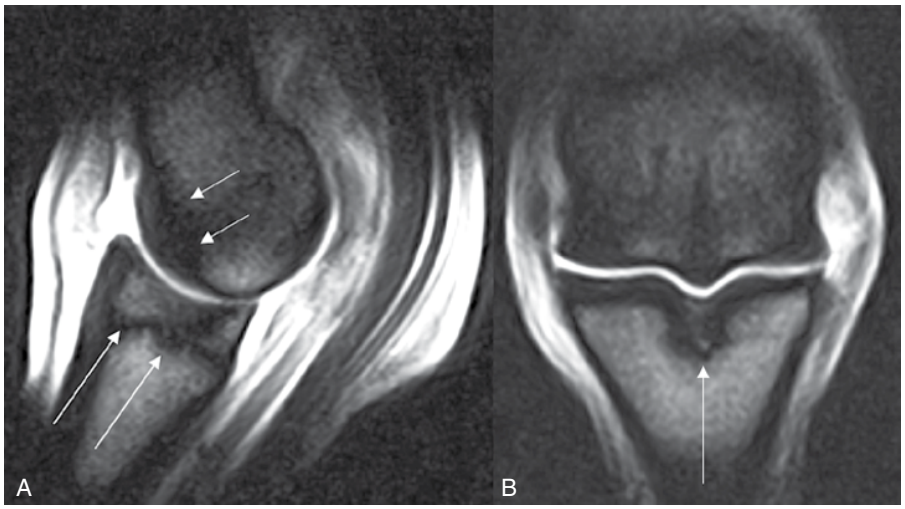


Figure 3.238. Sagittal (A) and dorsal (B) low-field T2* GRE images of the left metacarpophalangeal joint of an 11-year-old Warmblood with lameness due to sagittal groove trauma of the proximal phalanx. There is a large area of high signal intensity with a margin of low signal extending in a dorsopalmar direction along the base of the sagittal groove (lower left arrows). A similar signal abnormality can be seen on the dorsal image (right arrow). This fat/water cancelation artifact is characteristic for abnormal osseous fluid accumulation (bone contusion). Notice also the area of low signal (sclerosis) occupying the dorsal aspect of the sagittal ridge of the third metacarpal bone (upper left arrows).

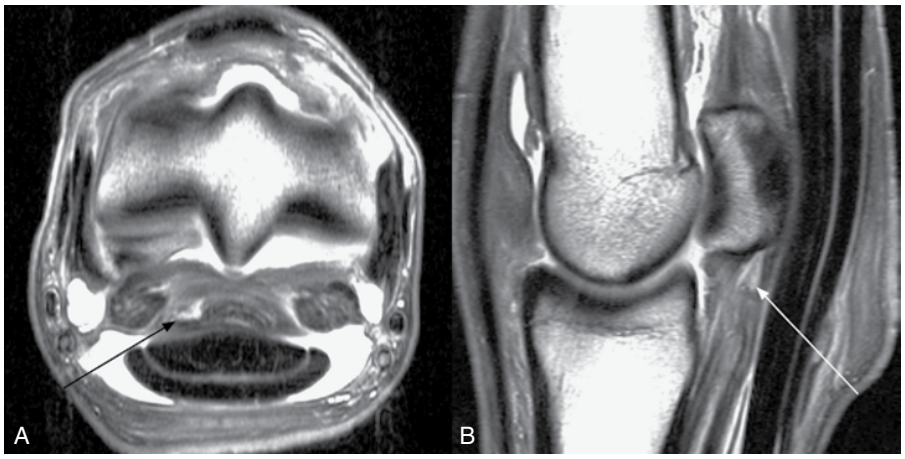


Figure 3.239. Transverse and sagittal proton density images of the metacarpophalangeal joint of a horse with chronic fetlock lameness. There is abnormal focal signal hyperintensity within the proximolateral aspect of the straight distal sesamoidean ligament indicative of local fiber disruption (black arrow). The lesion originates at the distal border of the lateral proximal sesamoid bone and extends 11 mm distally (white arrow).

osteoarthritis (osteophytes and synovitis).[69] Abnormal bone signal persisted at the sagittal groove of all horses with recheck MRI examinations.

Incomplete sagittal fractures of the proximal phalanx have been described mainly in racehorses and more recently also in sport horses.[58,69,91,99,144] MRI reviews of sagittal groove trauma in sport horses report incomplete sagittal fractures in 20,[69] 28,[99] and 50%[51] of patients, characterized by linear increased signal intensity in the subchondral bone in T1- and T2-weighted images.

Bone marrow lesions (osseous fluid) with or without loss of bone mineral density, extending distally from the joint surface through the subchondral bone of the medial glenoid of the proximal phalanx, have also been observed as a cause of fetlock lameness.[70,137,139,144]

Distal Sesamoidean Ligament Injuries

The straight distal sesamoidean ligament has a heterogeneous MRI appearance with multiple high and low signal areas spread throughout most of its length.[172] A normal, small homogeneous triangular signal hyperintensity exists at the insertion of the straight distal sesamoidean ligament onto the middle phalanx. The oblique distal sesamoidean ligaments have heterogeneous signal intensity throughout their entire length to the insertion. The lateral oblique distal sesamoidean ligament is frequently larger and more hyperintense than the medial ligament.[172] In a standing horse, images of the proximal third of the oblique distal sesamoidean ligaments are susceptible to magic angle effect, especially medially, resulting in increased signal intensity in the ligaments, thereby confounding image interpretation.[171] The signal heterogeneity of the distal sesamoidean ligaments varies between individual horses.[172] Normal signal heterogeneity should not be confused with the presence of a lesion. Abnormal signal intensity in a lesion of the distal sesamoidean ligaments commonly covers a larger cross-sectional area than the focal signal hyperintensities caused by normal signal variation.[172] Lesions within the oblique and straight distal sesamoidean ligaments can result in discrete or diffuse areas of signal hyperintensity within the body or along the edge of the affected ligament. One or multiple small core lesions with focal signal increase may be observed in affected ligaments, extending from 5 to 30 mm in a proximodistal direction (Figure 3.239). Enlargement of a ligament may also occur with or without abnormal signal increase, but this finding is less consistent. In one study, 30% of distal sesamoidean ligaments affected with desmitis were not enlarged.[70] Lesions may occur biaxially or bilaterally.

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The location of lesions within distal sesamoidean ligaments and the distribution of lesions varies between studies.[70,90,151,172] Straight sesamoidean desmitis occurs more commonly in the distal part of the ligament, proximal to its insertion on the middle phalanx,[151,172] although proximal lesions near the origin were most common in another study (Figure 3.239).[70] Oblique distal sesamoidean desmitis can occur proximally or throughout the entire length of the ligament.[70,90,151,172] Cruciate distal sesamoidean desmitis is very rare.[144,172]

Reports suggest that distal sesamoidean desmitis is frequently regarded as the primary cause of lameness,[70,90,151] although one author considered lesions to be the sole cause of lameness in only 2 of 58 horses with evidence of desmitis.[172] The majority of horses with oblique or straight distal sesamoidean desmitis diagnosed with MRI do not have a palpable enlargement, nor do they show any ultrasonographic abnormalities.[70,151,172]

Suspensory Ligament Branch Injuries

The suspensory ligament branches are paired triangular structures of low signal intensity that flatten in a dorsopalmar direction and widen lateromedially as they move distally toward their insertion on the proximal sesamoid bones. The margins of the branches are sharply delineated. Close to the insertion, the branches become D shaped on cross section, and faint, linear, high intensity, dorsopalmar striations appear near the ligament–bone interface, possibly associated with the presence of adaptive fibrocartilaginous metaplasia at the insertion. A small hyperintense indentation may be present in the palmar border of the normal suspensory branch immediately proximal to its insertion.

Generally, there is a good correlation between the presence of (even mild) MRI abnormalities in a suspensory branch and the ultrasonographic appearance of fiber abnormalities in that branch. Lesions are characterized by an intraligamentous focus of signal hyperintensity in PD, T2, and STIR images, usually near the palmar/plantar border of the affected branch, with or without enlargement of that branch (Figure 3.240). The clinical significance of mild signal changes in the suspensory branches is not always clear, but they may be the only finding in an otherwise normal MRI examination of a horse whose lameness was abolished by a low four-point nerve block. During standing low-field MRI, false signal increase in one or both suspensory branches may be caused by vascular flow artifact or magic angle artifact, even on transverse T2 FSE images.

Injuries of the Proximal Sesamoid Bones

Abnormal MRI signal in the proximal sesamoid bones includes osteosclerosis, STIR signal hyperintensity consistent with osseous fluid or contusion, and focal trabecular bone loss. Standing low-field MRI has been helpful in identifying osseous cyst-like lesions at various locations in the proximal sesamoid bones, involving the articular surface, the axial border, the abaxial surface, or the base.[108] Enthesopathy at the damaged attachment site of a sesamoidean ligament may result in trabecular bone loss leading to an osseous cyst-like lesion of the proximal sesamoid bone (Figure 3.241). Bone mineral

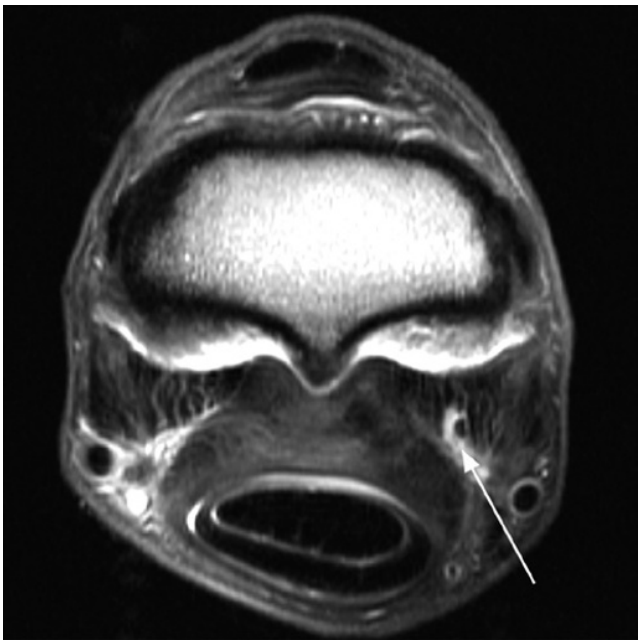


Figure 3.240. Transverse proton density image of a metatarsophalangeal joint of a horse with lameness localized to the fetlock region. There is a small, linear hyperintensity reflecting the presence of a tear in the plantar border of the lateral branch of the suspensory ligament (arrow).

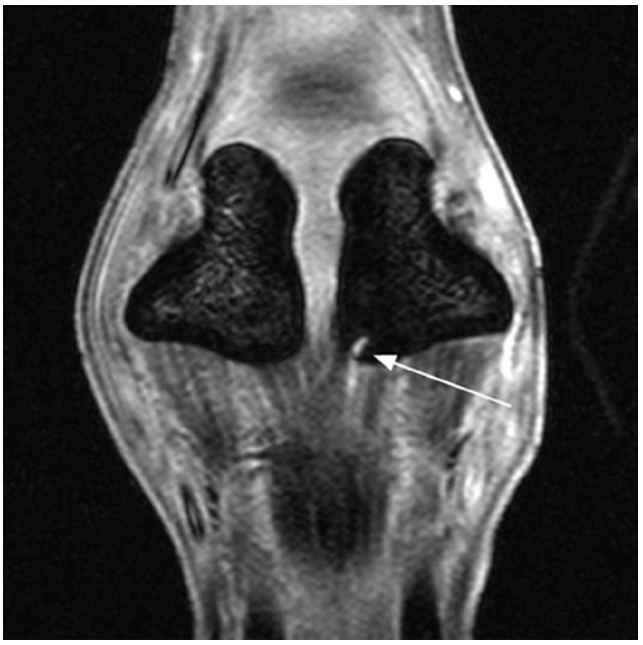


Figure 3.241. Dorsal fast low-angle shot (FLASH) image with fat saturation of the proximal sesamoid bones of a horse with lameness localized to the metacarpophalangeal joint. There is focal and intense signal hyperintensity at the base of the medial proximal sesamoid bone (arrow) indicative of a small osseous cyst-like lesion associated with a small vertical tear of the straight distal sesamoidean ligament.

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densification of 75% or more of a proximal sesamoid bone or continuous dorsal to palmar bone mineral densification, as identified on standing low-field MR images, was considered to be associated with an increased risk of catastrophic fracture of the proximal sesamoid bones.[140]

Injuries of the Intersesamoidean Ligament

Intersesamoidean desmopathy results in a large or small, focal, central area of signal hyperintensity within the damaged portion of the intersesamoidean ligament in T1, T2, PD, and STIR images (Figure 3.242). Hyperintense lesions associated with focal trabecular bone loss may be found along the axial margin or at the apex of the proximal sesamoid bones in association with intersesamoidean ligament injury. Injuries may also result in small avulsion fracture fragments of the axial margin. Intersesamoidean ligament injury can affect one or both proximal sesamoid bones.

Axial osteitis of the proximal sesamoid bone and necrosis of the intersesamoidean ligament characterized by hyperintense signal may also be a complication of focal sepsis of hematogenous or unknown origin, and the distinction between septic and traumatic lesions may be difficult to make, even on MR images.

Osteophyte formation at the proximal and distal margins of the sesamoid bones may be present in association with osteoarthritis of the fetlock joint.

Osteoarthritis and Articular Cartilage Abnormalities

The distal articular surface of the MCIII/MTIII in the fetlock joint is the most difficult to image with MRI because of its curvature and the thin articular cartilage layer. The ability to assess cartilage injuries in this joint challenges the limits of MRI systems and the capability to resolve fine detail.[193] A focal, irregular island of fluid hyperintensity associated with pooling of synovial fluid in a chondral defect can sometimes be recognized on transverse PD, T2, or STIR images that are located exactly parallel with and through the affected articular surface (Figure 3.243). Cartilage defects have been identified on all articular surfaces in the fetlock joint.[70] Overall, high-field MRI tends to underestimate the number of cartilage lesions, the size of the lesions, and the extent of cartilage loss in the fetlock joint compared with arthroscopic findings, gross examination, or histology.[70,132,136] The sensitivity of MRI is particularly decreased when chondral lesions are linear in nature.[136,193] There is a major effect of field strength on detection of articular cartilage lesions in the fetlock joint, with markedly decreased accuracy of standing low-field MRI.[193] In an *ex vivo* study comparing the effect of sequence selection and field strength on detection of osteochondral defects in the metacarpophalangeal joint, experimentally created articular cartilage defects were not identified on low-field images.[193]

A specific focal cartilage defect at the dorsodistal abaxial aspect of the medial metacarpal condyle has been described as a chondral delamination injury of the equine distal metacarpus caused by calcified cartilage fracture.[110] The lesion is characterized by intense focal STIR signal increase in the subchondral bone subjacent to the cartilage defect and by pooling of synovial fluid inside the contours of the cartilage defect on MRI images.

Osteophytes may be seen as contour changes of the proximal and distal articular margins of the proximal sesamoid bones and the dorsoproximal, lateral, and medial margins of the proximal phalanx. Osteophytes may be more easily recognized on radiographs due to the radiographic summation effect and the better radiographic contrast between cortical bone and soft tissue attachments at the joint margins.[70] However, osteophyte scores were significantly higher on MR images than on radiographs in a study comparing imaging modalities for the assessment of noncartilaginous changes in equine metacarpophalangeal osteoarthritis.[135,137] The authors reported a particular central subchondral osteophyte type that could only be diagnosed with high-field MRI or computed tomography.[135] Central subchondral osteophytes were described as focal hypointense protuberances projecting from the subchondral plate of the palmarodistal aspect of the metacarpal condyle into the overlying articular cartilage.

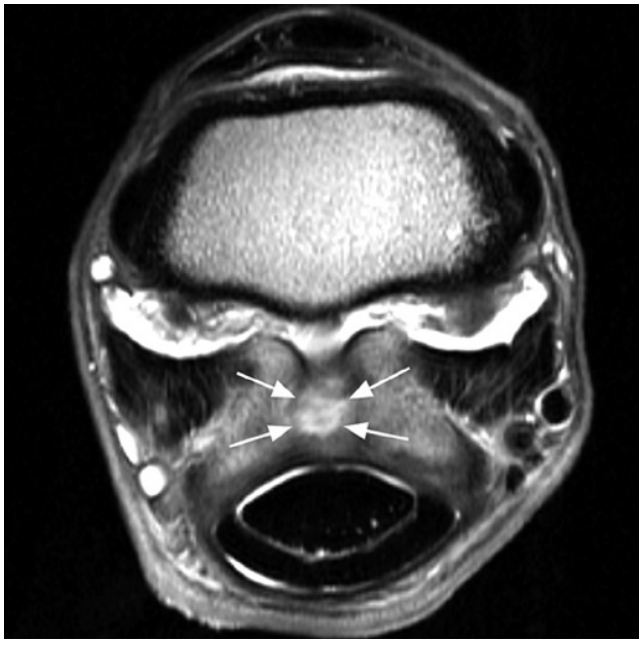


Figure 3.242. Transverse proton density image at the level of the apices of the proximal sesamoid bones of a horse with acute severe metacarpophalangeal joint lameness. There is a circular area of signal hyperintensity indicative of fiber disruption and desmitis in the intersesamoidean ligament (arrows).

Osteochondral Fragmentation

Some osteochondral fragments from the dorsoproximal margin of the proximal phalanx and the basilar margin of the proximal sesamoid bones that are not visible radiographically may be recognized as focal areas of osseous hypointensity separated from parent bone on all sequences. Osseous fragments may be difficult to distinguish from end-on blood vessels near joint margins as both may appear hypointense on MR images. Avulsion fragments are always difficult to differentiate from the

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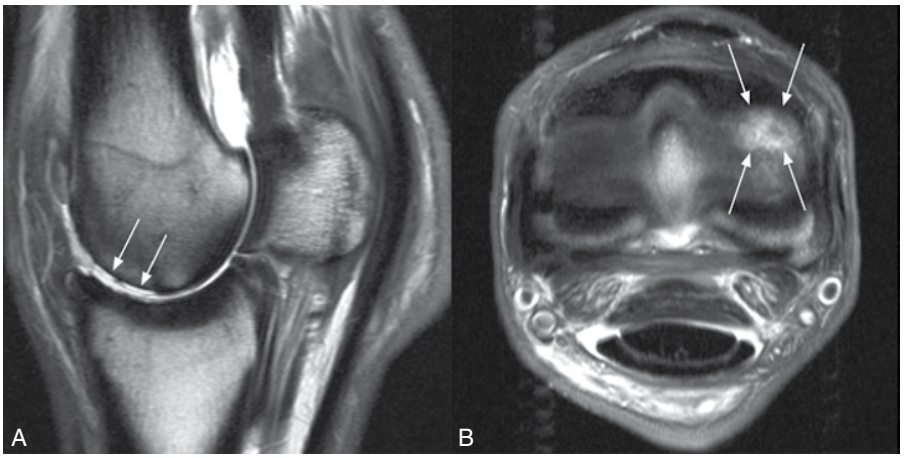


Figure 3.243. Sagittal and transverse proton density images of the fetlock of a horse with chronic metacarpophalangeal joint lameness. There is an elliptical area of full-thickness cartilage loss on the dorsodistal aspect of the medial condyle of the third metacarpal bone (arrows). This is characterized by replacement of the normal hypointense cartilage layer with pooling of hyperintense synovial fluid in the cartilage defect (arrows). A wavy, thin, hypointense line overlying the cartilage defect may indicate the presence of pannus tissue along with synovial fluid (A). An irregular island of fluid hyperintensity associated with pooling of synovial fluid in the chondral defect can be recognized on the transverse PD image that runs through the affected joint surface (B).

tendon or ligament in which they are embedded, due to the similarities in signal intensity between bone, tendon, and ligaments.

Abnormalities of the Digital Flexor Tendon Sheath

Injuries of the digital flexor tendons in the digital flexor tendon sheath may be recognized as dispersed small, focal areas of signal hyperintensity, distinct hyperintense core lesions, thickening of the affected lobe(s), and/or longitudinal parasagittal or frontal splits of the lateral or medial border of the tendon with partial separation of the tendon margins (Figure 3.244). Lesions of the DDFT within the digital flexor tendon sheath may continue distally into the navicular bursa and the insertion on the distal phalanx. Lesions of the superficial digital flexor tendon within the digital flexor tendon sheath may extend into one of the branches of the tendon and its insertion on the middle scutum. Areas of signal hyperintensity and contour changes of the flexor tendons within the digital sheath are most obvious in transverse fat-suppressed T1 spoiled gradient-echo (SPGR) images and frequently not visible ultrasonographically. Increased fluid distension and intrathecal soft tissue proliferation of the digital flexor tendon sheath and thickening and signal change in the palmar annular ligament of the fetlock have also been recognized.[70]



Figure 3.244. Transverse fast low-angle shot (FLASH) image with fat saturation immediately distal to the base of the proximal sesamoid bones of a horse with chronic lameness localized to the digital flexor tendon sheath. There is a small linear hyperintensity (arrow) that was revealed to be a longitudinal tear of the lateral margin of the deep digital flexor tendon extending 3.5 cm proximodistally into the fetlock canal. The detached lateral margin of the tendon is slightly displaced. This lesion was treated successfully by tenoscopic removal.

Collateral Ligament Injuries

Collateral desmitis is characterized by enlargement of the superficial or deep part of the collateral ligament relative to the contralateral limb and by the presence of signal hyperintensity in T2 and PD images in the affected part of the ligament. Signal increase may be difficult to recognize in the deep part of the collateral ligament as this structure frequently appears hyperintense on PD and T1-weighted MR images, due to the presence of magic angle effect caused by the oblique fiber orientation in the deep part of the collateral ligament. Evidence of endosteal irregularity may be present at the origin of a collateral ligament. A small avulsion fragment at the base of the epicondylar fossa in association with collateral desmitis has been described.[70]

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MAGNETIC RESONANCE IMAGING OF THE METACARPAL AND METATARSAL REGIONS

Introduction

Lameness caused by pain associated with the proximal palmar metacarpal and plantar metatarsal regions is common in equine athletes. Some of the causes of lameness in this region are difficult to diagnose accurately because affected horses frequently have no or equivocal radiological or ultrasonographic signs of disease. It may thus not always be possible to distinguish between normal anatomy, imaging artifacts, and structural abnormalities of the suspensory ligament using ultrasonography.[52] The tomographic nature of MRI allows assessment of size and signal changes within the complex anatomy of this region and results in good soft tissue contrast and detail without superimposition.

There have been several reports with widely varying results on the incidence of injuries diagnosed with both low- and high-field MRI systems in the metacarpal and metatarsal regions (Tables 3.5 and 3.6).[5,6,28,93,128] In general, transverse MRI slices that are aligned perpendicular to the long axis of the cannon bone (parallel with the carpometacarpal/tarsometatarsal joint) are most useful for diagnosis.

In the forelimb (Table 3.5), the reported distribution of MRI abnormalities appears to depend on field strength of the system used. A very low incidence of soft tissue injury in the proximal metacarpal region has been reported in a low-field study,[128] while high-field studies consistently indicate much higher numbers of injuries of the accessory ligament of the DDFT and the suspensory ligament.[5,28] This may be explained by easier recognition of bone signal change than soft tissue change in standing low-field MR systems, because motion artifact in standing patients can lead to poor recognition of anatomical detail as well as of signs of injury in soft tissue structures. Motion artifact results in blurring of signal and loss of contour definition of the muscle and adipose tissue bundles within the suspensory ligament. Inability to accurately define these muscle and adipose tissue bundles hampers the ability to distinguish these bundles from areas of fiber damage in the collagenous part of the suspensory ligament. Lesion occurrence in forelimbs is also heavily influenced by the horse's discipline and age. A recent high-field study showed a much higher prevalence of osseous abnormalities of the third metacarpal bone in young Quarter horses used for cutting than previously reported in studies of mixed breed sport horse populations.[5,28]

As the only retrospective MRI studies of the metatarsal region available have been performed with high-field magnets,[6,28,93] a comparison between low- and high-field injury prevalence is not possible for the hindlimb (Table 3.6). However, given that motion artifact tends to be more pronounced in standing MRI of the hindlimb than the forelimb, the same concerns apply. By far the most common injury in the proximal metatarsal region is desmopathy of the proximal part of the suspensory ligament with or without an enthesis injury of the third metatarsal bone at the attachment of the suspensory ligament. The high occurrence of MR abnormalities of the distal tarsal region in horses with lameness localized to the proximal plantar metatarsal region with diagnostic anesthesia illustrates the concern that local anesthetic techniques lack specificity for accurate localization of pain causing lameness in this area.[6,93]

MRI Abnormalities in the Proximal Metacarpal/Metatarsal Regions

Proximal Suspensory Desmopathy

The normal MRI anatomy of the fore and hind suspensory ligaments has been described in detail.[14,59,94,124,127] The normal suspensory ligament is a low signal intensity structure of hypointense dense collagen fibers, interspersed with two centrally located high signal intensity tissue bundles, that each contain sparse muscle fibers surrounded by a layer of loose and adipose connective tissue and neurovascular structures. Normal cross-sectional areas have been defined for the proximal part of the suspensory ligament in the forelimb and vary from 1.61 to 1.90 cm^2 in one study[5] and from 1.94 to 2.32 in another.[127] There is considerable variation between horses in the MRI appearance of the proximal portion of the forelimb suspensory ligament,[124] due to the variation in the amount, shape, and signal intensity of the muscle and fat tissue bundles.[127] Within these bundles, high signal intensity areas correspond to adipose tissue and intermediate signal intensity areas to muscle tissue. The large variability in the MRI appearance of the proximal portion of the forelimb suspensory ligament in non-lame horses should be borne in mind when interpreting MR images of lame horses.[127] Therefore, comparison with the contralateral limb or with published reference images may be useful to help with interpretation of suspected MRI changes.[124]

In hindlimbs, the suspensory ligament starts as a narrow triangular band, evolves into a right-angled triangle that is wider on the lateral aspect than medially, and contains a dorsal central cleft dividing it in two lobes. At 4 cm distal to the tarsometatarsal joint, it becomes oval to bean shaped and separates away from the plantar cortex of the third metatarsal bone to continue distally, while both lobes merge into one oval ligament.[161] Normal cross-sectional areas have been defined for the proximal part of the suspensory ligament and have varied from 1.12 to 2.23 cm^2.[161] Injuries of the proximal part of the suspensory ligament may result in enlargement, contour changes, focal and diffuse signal increase, and periligamentous fibrosis.

Abnormalities are best seen in transverse PD images. Lesions can be graded as mild, moderate, or severe, depending on the abnormal signal intensity and the proportion of total cross-sectional area of the ligament affected.

Enlargement of the cross-sectional area of the suspensory ligament may vary from mild to severe, but because of the variation in size within limbs and between horses, this feature should be interpreted with caution especially when the enlargement is mild.[5,93,127,161] Even though marked enlargement may result in a decrease of the space between the dorsal margin of the suspensory ligament and the palmar or plantar surface of the bone, periligamentous fibrosis and adhesion formation are uncommon features of proximal suspensory desmopathy.

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Table 3.5. Frequency of MRI abnormalities in three retrospective studies of horses with lameness caused by pain localized to the proximal metacarpal region (Brokken et al.[28] and Barrett et al.[5]) or proximal metacarpal and distal carpal regions (Nagy and Dyson[128]).

MRI lesions % incidence	N = 29 horses[a]	N = 58 limbs	N = 44 limbs[a,b]
Bone injury proximal MCIII	3	10	Sclerosis: 96 Focal resorption: 73 Osseous fluid: 56
PSD	14	—	Enlargement: 75 Muscle/ligament architecture: 25 Dorsal fiber irregularity: 68 Increased PD signal: 57
Concurrent PSD and bone injury proximal MCIII	21	9	NR
Desmitis AL DDFT	55	—	—
Tendinopathy DDFT	7	—	—
Medial sclerosis carpal bones and/or metacarpal bones	—	50	
Syndesmopathy MCII-MCIII	—	17	—
Osseous and soft tissue abnormality between C2-C3-C4 and MCII-MCIII	—	10	—
Osseous reaction MCII, MCIV	—	—	45
OA CMC joint	—	3	12
OA ICJ		—	11
No obvious abnormalities	—	—	—

OA, osteoarthritis; DDFT, deep digital flexor tendon; AL, accessory ligament; MCII, second metacarpal bone; MCIII, third metacarpal bone; MCIV, fourth metacarpal bone; C2, second carpal bone; C3, third carpal bone; C4, fourth carpal bone; CMCJ, carpometacarpal joint; ICJ, intercarpal joint; PSD, proximal suspensory desmopathy; and NR, not reported.

Several horses or limbs had simultaneous incidence of more than one lesion.

[a] High-field MRI system.

[b] Western cutting horses only.

Contour changes or loss of a distinct border are common[28] and usually accompany areas of focal or diffuse signal increase in the dense collagenous regions of the ligament. Focal or diffuse areas of signal increase reflect disruption of the dense hypointense collagenous tissue.[28,93] Signal increase has been described predominantly in the central and centrolateral dense collagenous portions of hind suspensory ligaments (Figure 3.245).[93] In one report, lesions extended on average 13.2–51.1 mm distal to the tarsometatarsal joint with lesion length varying from 4.3 to 107 mm.[93] In the forelimbs of young Quarter horses used for cutting, signal increase was seen predominantly at the dorsal margin of the proximal portion of the ligament, either confined to the medial bundle or involving the entire dorsal margin.[5] Although signal changes due to fiber disruption mainly affect the dense collagenous part of the suspensory ligament, muscle and fat bundles may be included in the disease area in severe cases, where the area of signal increase exceeds 30% of the cross-sectional area of the ligament with loss of normal architecture.[5,6,93] Decrease in size or signal of the central normal muscle and fat bundles may be caused by relative enlargement of the peripheral collagenous areas of the ligament.[28] Recently, an association has been suggested between proximal suspensory pain and signal changes and histological abnormalities of the fat and muscle bundles without involvement of the dense collagenous tissue in the proximal portion of the suspensory ligament of hindlimbs.[60,61] However, given the normal signal variation of these fat and muscle tissue bundles, the study's bias toward limbs with moderate to severe ultrasound changes, the lack of agreement between microscopic lesions of the muscle bundles and moderate to severe echogenicity changes of the collagen fiber pattern, and the lack of agreement between documented microscopic lesions of muscle and fat bundles and previous high-field MRI descriptions of proximal suspensory desmopathy,[6,28,93] a better understanding of these findings, their prevalence, and significance is required.

In many cases, evidence of enthesis bone injury at the level of attachment of the suspensory ligament to the

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Table 3.6. Frequency of MRI abnormalities in three retrospective high-field studies of horses with lameness caused by pain localized to the proximal metatarsal region (Brokken et al.[28] and Labens et al.[94]) or proximal metatarsal and distal tarsal regions (Barrett et al.[6]).

MRI lesions % incidence	*N* = 16 horses	*N* = 48 limbs of 39 horses	*N* = 125 limbs of 103 horses
PSD	50	25	53
Bone injury proximal MTIII	0	8	Enthesopathy: 32 Osseous fluid: 23
Concurrent PSD and bone injury proximal MTIII	10	19	NS
Distal tarsal OA	—	6	DITJ: 42 TMTJ: 51
Splint bone injury with focal suspensory desmitis/adhesion	—	4	—
Tarsal bone cyst	—	2	18
Slab fracture	—	-	3
Enthesopathy/desmopathy intertarsal ligaments	—	2	27
Bone marrow lesion tarsal bone(s)	—	4	13–22
Medullary injury MTIII	—	2	—
Dorsal cortical injury MTIII	—	2	—
Tendinopathy DDFT	6	2	—
Tendinopathy SDFT	—	2	—
Effusion of the distal tarsal sheath	6	—	—
No obvious abnormalities	6	22	—

NS, not specified; OA, osteoarthritis; DDFT, deep digital flexor tendon; MCIII/MTIII, third metacarpal/metatarsal bone; MCIV/MTIV, fourth metacarpal/metatarsal bone; TMTJ, tarsometatarsal joint; and DITJ, distal intertarsal joint.
Several horses or limbs had simultaneous occurrence of more than one lesion.

proximal palmar/plantar aspect of the cannon bone is seen concurrently with proximal desmopathy (Figure 3.246). The concurrent presence of bony and ligamentous changes appears to be more common in forelimbs than hindlimbs, especially in young Quarter horses used for cutting.[5]

Adhesions of the Suspensory Ligament

In normal forelimbs, a space of loose connective tissue with high signal intensity exists between the medial border of the suspensory ligament and the second metacarpal bone.[124,127] The lateral border of the suspensory ligament is overall closer to the fourth metacarpal bone and may in some areas contact the bone. The same applies in the hindlimb, where the suspensory ligament is positioned more laterally on the plantar aspect of the cannon bone than in forelimbs and the lateral border of the ligament contacts the fourth metacarpal bone between 0 and 2 cm and again between 8 and 12 cm distal to the tarsometatarsal joint.

The tomographic nature of MRI allows for visualization of the margins of the entire suspensory ligament. This has enabled a more definitive diagnosis of adhesions between ligament margins and exostoses of the second and fourth metacarpal and metatarsal bones,[203] where ultrasonography is unable to determine the existence of abnormality. Adhesions lead to suspensory desmitis through tearing of the adhered ligamentous fibers with resulting inflammation and lameness. Loss of normal space between the suspensory ligament and a splint bone, in conjunction with signal or contour changes of the suspensory ligament and bone proliferation on the surface of that splint bone, is strongly suggestive of adhesion formation. Adhesions may be visible as a tissue band of low signal connecting the ligament to an area of osseous proliferation on the axial surface of a splint bone. However, these findings should be interpreted with caution because normal fibers of the suspensory ligament have been shown to originate from the proximal part of the fourth metatarsal bone and the lateral margin of the normal suspensory ligament is overall

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closer to and may contact the fourth metacarpal and metatarsal bones.

Osseous Injury of the Third Metacarpal/Metatarsal Bone

The normal proximal palmar/plantar metacarpal/metatarsal cortex has a uniform thickness and smooth periosteal and endosteal surfaces, although mild endosteal and periosteal irregularity may be seen in some horses.[124] The smoothness of the palmar/plantar cortex at the origin of the suspensory ligament is difficult to evaluate, because both the cortex and the suspensory ligament have low signal intensity and cannot be distinguished from one another. Between 3 and 5 cm distal to the carpometacarpal joint, the metacarpal cortex is slightly thicker medially than laterally.

Bone injury at the site of attachment of the suspensory ligament not only occurs most frequently in combination with proximal suspensory desmitis but also can be seen as an isolated injury.[5,28,93,127] Abnormalities indicative of enthesis bone injury at the origin of the suspensory ligament include abnormal medullary signal hypointensity on transverse PD and T2-weighted images indicative of increased bone density (sclerosis) (Figure 3.246), medullary signal hyperintensity on STIR images and fat/water cancelation artifact on T2* images compatible with the presence of abnormal bone fluid, or a combination of both sclerosis and fluid. Horses with chronic suspensory ligament lesions tend to have more sclerosis often with thickening of the proximal palmar/plantar cortex and an irregular endosteal or periosteal contour due to new bone formation (Figure 3.246). The palmar/plantar cortex may further contain focal high intensity signal indicative of trabecular bone resorption resulting in an altered contour with a focal concave bone defect.

Primary bone injuries without suspensory ligament abnormalities can also occur in the proximal palmar/plantar aspect of the bone, with a similar range of MRI abnormalities indicating fluid, sclerosis, or both. The exact cause of these bone injuries is unknown, but a mechanism of repetitive cortical fatigue or stress injury seems likely,[115,151] especially in horses that perform strenuous exercise at high speed or over long distances, even though the injury has been documented in many different types and breeds.[115] MRI signs of traumatic bone bruising and remodeling appear predominantly on the medial palmar aspect of the proximal portion of the metacarpus but not limited to the attachment area of the suspensory ligament and have been described in young racing Thoroughbreds,[114,150] horses used for endurance racing,[29] and Western cutting horses.[5] In a more advanced form of this traumatic fatigue injury, incomplete vertical or oblique hairline fractures of up to 9 cm length may develop in the proximopalmar medial aspect of the third metacarpal bone.[82,114,115]

Isolated bone marrow lesions in the medullary cavity of the third metacarpal/metatarsal bones can occur in close relation to the nutrient foramen and intramedullary blood vessels as can focal enostosis-like lesions.

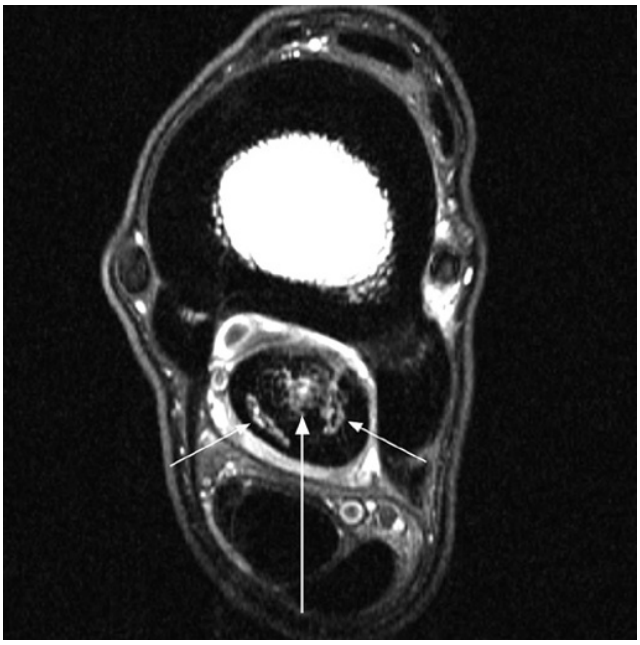


Figure 3.245. Transverse proton density image of the proximal metatarsal region of the right hindlimb of a horse with mild to moderate proximal suspensory desmitis. There is an abnormal area of diffuse signal increase in the central part of the suspensory ligament (long arrow). This must be distinguished from the two normal focal areas of high signal intensity associated with two muscle and fat tissue bundles in the suspensory ligament (short arrows).

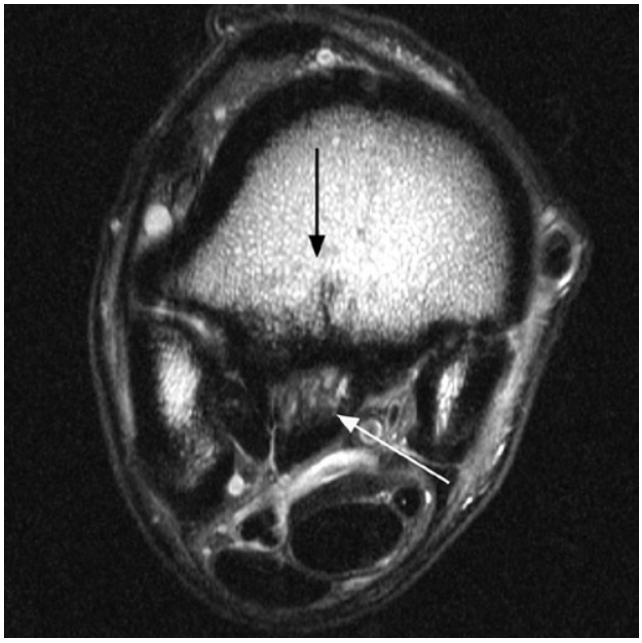


Figure 3.246. Transverse proton density image of the proximal metatarsal region of the left hindlimb of a horse with marked proximal suspensory desmitis and enthesopathy of the proximal plantar metatarsal cortex. There is a large central area of abnormal signal hyperintensity in the suspensory ligament (white arrow). There are irregular areas of low signal in the medullary cavity of the third metatarsal bone reflecting the presence of osteosclerosis (black arrow). The plantar metatarsal cortex is thickened and has an irregular endosteal margin.

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MRI evidence of traumatic bone injury is not only limited to the proximal palmar/plantar aspect of the third metacarpal/metatarsal bones but also has been encountered in the dorsal third metacarpal/metatarsal bone, as well as in the proximal aspect of the splint bones.[29,93]

Desmopathy of the Accessory Ligament of the Deep Digital Flexor Tendon

The accessory ligament of the DDFT often has heterogeneous signal intensity when examined in either a high-field or a low-field magnet.[125] In a cadaver MR imaging study, the majority of accessory ligaments had low to intermediate signal intensity but contained oblique bands of high signal intensity crisscrossing through the ligament. This was thought to be caused by the presence of thick collagen bundles arranged in large crimps, crossing obliquely and randomly through this ligament, thereby causing extensive magic angle effects.[125] Moreover, the cross-sectional area of the accessory ligament varied widely ranging from 68.1 to 299 mm^2 in its most proximal 7 cm. In addition, the authors cautioned that the presence of normally attached fibrous bands and the large variability in signal intensity and size of the accessory ligament in non-lame horses should be borne in mind when interpreting MR images of lame horses.[125]

MRI signs of desmitis were size increase and focal to diffuse intraligamentous signal hyperintensity.[28] Lesions generally extend from 1 to 4 cm distal to the carpometacarpal joint. Comparison with the contralateral limb was considered essential to allow detection of subtle signal and size changes. In this study, desmitis of the accessory ligament was the most common MRI finding in horses with proximal metacarpal region lameness. However, this observation has not been supported by others,[5,128] who never mentioned desmitis of the accessory ligament when discussing injuries in the proximal metacarpal region.

Abnormalities of the DDFT or the suspensory ligament may accompany desmitis of the accessory ligament.[142] In horses with chronic desmitis, irregularity of the borders of the ligament and loss of the distinct high signal border between the accessory ligament and the DDFT may occur. Low signal scar tissue bridging the hyperintense space between the ligament and the flexor tendons may be indicative of adhesion formation but must be distinguished from the normal fibrous bands on the lateral aspect of the ligament.[125]

Other Abnormalities

Other injuries have been reported in horses with lameness localized to the proximal palmar/plantar metacarpal/metatarsal region with diagnostic analgesia with a much lower incidence than those listed above (Tables 3.6 and 3.7). Mostly these findings involve the distal tarsal and carpal joints (see "MRI Abnormalities of the Carpal Region and MRI Abnormalities of the Tarsal Region") and indicate the lack of specificity of diagnostic analgesia of the proximal metacarpal and metatarsal regions.[6,93]

Occasionally focal fiber disruption without thickening is recognized with MRI in the SDFT or DDFT in the absence of ultrasonographic abnormalities.[41,93,150] MRI is not used routinely for the diagnosis of injuries of the flexor tendons in the metacarpal region as this is mainly done with ultrasonography. However, MRI may be more sensitive than ultrasonography for monitoring tendon repair.[86,88,160] The T2 versus T1 signal differences in healing tendon may prove to be helpful in planning the rehabilitation of horses with tendon injuries more accurately.[160,190]

Osseous and soft tissue abnormalities of the second and fourth metacarpal bones (exostosis, synostosis, osteitis) and their interosseous ligaments (syndesmopathy) have been reported as very common in two studies of proximal metacarpal MRI.[5,128] These include irregularities of the periosteal and endosteal aspects of the cortex, osseous fluid, and/or sclerosis, increased intraligamentous STIR signal and enthesophyte formation at the origin, and insertion of the interosseous ligaments. Synostosis may lead to osseous fusion between the second or fourth and the third metacarpal bones. These abnormalities are common in young horses, especially endurance and race horses, and their clinical significance as a cause of lameness is not always clear.

MAGNETIC RESONANCE IMAGING OF THE CARPAL REGION

Introduction

Practical problems may arise when scanning limb areas proximal to the third metacarpal and metatarsal regions with both high-field magnets or low-field standing systems. It can be difficult to position the carpus or tarsus in the isocenter of most long, closed-bore, high-field magnets. When MR images are obtained on standing sedated horses with low-field open magnets, increased movement at these proximal levels can render diagnostic quality imaging difficult, in spite of advances in motion correction software. This problem does not appear to occur with the horse's limb positioned under general anesthesia in an open low-field magnet, provided that the magnet opening is sufficiently wide to accommodate the area of interest.

Most carpal injuries can be sufficiently evaluated using radiography, ultrasonography, and scintigraphy. Consequently, few clinical reports of *in vivo* MRI results in horses are available,[128,146,175] and cadaveric studies have been used to describe the normal low- and high-field MRI anatomy of and signal distribution in the carpus.[71,116,120,126] MRI was able to identify several ligaments that cannot be shown with other imaging techniques (except CT arthrography), including the transverse intercarpal ligaments, the radiocarpal ligament, the short palmar carpal ligaments, and the carpometacarpal ligaments.[71,126] Transverse MRI images are typically most useful to evaluate the soft tissues of the carpal region.

Even though diagnosis of carpal lameness can usually be made with conventional imaging modalities, MRI of the carpus may be useful when no imaging abnormalities are present, when the degree of lameness does not

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correlate with imaging findings, or when the pattern of diagnostic anesthesia is unclear due to confusion between the responses to subcarpal nerve blocks and intercarpal joint anesthesia.

Lesions detected with MRI in horses with carpal lameness have included bone sclerosis and/or osseous fluid, osteochondral pathology, fractures, ligament injuries, and injuries of the carpal canal.[116,128] The most commonly detected abnormality in the carpus in one low-field study was decreased T1 and T2 signal intensity (sclerosis) in the medial aspect of the carpal bones. Less common were signal abnormalities between the second and third carpal bones.[128]

MRI Abnormalities in the Carpal Region

Osseous and Osteochondral Pathology

Carpal bone sclerosis is well recognized in racing Thoroughbreds and Standardbreds, but its clinical significance is not always clear. The third and radial carpal bones are most commonly affected.[128] Sclerosis without osseous fluid may represent adaptive remodeling changes following repetitive loading, or may be a cause of lameness,[128] especially when the area of sclerosis is extensive. Sclerosis with osseous fluid represents nonadaptive bone damage and is more closely associated with lameness. Extensive sclerosis may lead to focal trabecular bone resorption and necrosis resulting in a focal subchondral area of signal increase representing bone resorption within the low intensity sclerotic area.

Extensive sclerosis may also be associated with osteochondral damage of the adjacent articular surface of the intercarpal or radiocarpal joints, and the articular cartilage should be assessed carefully for thickness and signal changes. Cartilage damage should be considered in the presence of subchondral bone edema or focal subchondral bone loss.

Carpal bone chip fractures are seen as a focal area of low signal intensity separated from the parent bone by a line of higher signal representing fluid in the fracture gap. There may be osseous fluid, sclerosis or both in the parent bone. The tomographic nature of MRI may help identify complex or hidden fracture lines that are not readily visible on conventional radiographic projections. This information may help in planning for surgical treatment of palmar fragments or comminuted fractures.

Soft Tissue Injuries

A variety of ligaments exist within and around the carpal joints. Injuries of intercarpal ligaments cannot be identified ultrasonographically, and diagnosis relies on arthroscopic or tomographic imaging with CT arthrography or MRI.[71,116] Intercarpal ligament damage may be seen as loss of the linear structure of the ligament with either increased signal intensity within the ligament on both T1- and T2-weighted images, or low signal intensity on T1-weighted images with high signal intensity on T2-weighted images because of ligament disruption and infiltration of synovial fluid into the defect. The diagnosis is facilitated if there are enthesis abnormalities of the intercarpal ligaments' attachment sites to the carpal bones, like osseous fluid, osseous resorption, or osseous proliferation. Damage to collateral ligaments is best assessed ultrasonographically because of their superficial location. Generalized signal increase in a ligament may reflect fiber disruption, but signal heterogeneity may exist in normal ligaments.

Injuries of the flexor tendons or the accessory ligament of the DDFT may be seen within the confines of the carpal sheath.[145] However, areas of injury with intratendinous signal increase must be distinguished from persistent muscular tissue, which may require contralateral comparison.[126] Injuries of the accessory ligament are sometimes limited to its origin on the palmar aspect of the third carpal bone, causing osseous irregularities of the attachment site as well as signal and size changes of the ligament itself.

MAGNETIC RESONANCE IMAGING OF THE TARSAL REGION

Introduction

MRI of the tarsus can be useful because of the limitations of other imaging modalities for the diagnosis of early or focal osteoarthritis, cartilage erosions, focal subchondral bone lesions, sclerosis, bone contusions, fractures, osseous cyst-like lesions, and intertarsal ligament injuries.[40,123]

The same limitations apply to scanning the tarsal region as apply to the carpal region, i.e. motion artifact from swaying is (even more) common on standing low-field images of the upper hindlimb and positioning near the isocenter may be impossible in closed high-field magnets.

Equine tarsal MRI anatomy has been described in detail using both low- and high-field systems.[12,16,26,27,57,94] In the normal tarsus, articular cartilage of the distal tarsal joints is very thin, which does not allow for distinction of proximal and distal cartilage layers in these joint spaces.[7] Subchondral bone plates of the distal tarsal bones and the third metatarsal bone have homogeneous, low signal intensity with a regular osteochondral junction and a smooth but undulating deep border.[26,27] In sport horses that undergo high intensity training, subchondral bone thickness is greater medially in the distal intertarsal joint and laterally in the tarsometatarsal joint.[25] This repeatable thickness pattern of subchondral bone is lost in horses with distal tarsal lameness.[25,26]

Ligaments of the tarsus typically have low signal intensity in all sequences; however, multiple areas of increased signal intensity have been identified at specific locations (like in the short and long medial collateral ligaments) and were attributed to focal magic angle effects associated with variable orientation of collagen fibers within the ligaments.[12] The talocalcaneal ligament has heterogeneous signal intensity in all sequences, due to infiltration of a mixture of joint fluid and fat between the collagen fibers of the ligament.[12] The accessory ligament of the DDFT varies in size and signal intensity. The long plantar ligament comprises a number of related parts, separated by lines of high signal intensity corresponding with fibrous septa. The plantar aspect of this ligament has uniformly low signal intensity in all

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sequences, but the dorsal half is more heterogeneous with multifocal spots or lines or bands of higher signal intensity.[59]

MRI of the tarsal and proximal metatarsal regions is frequently performed together, as diagnostic anesthesia is insufficiently specific for accurate localization of pain causing lameness in this area.[6,93] In one study, 47% of horses with a positive response to subtarsal perineural diagnostic anesthesia were found to have a primary lesion in the distal tarsal joints, while 33% of horses with a positive response to intra-articular anesthesia of the distal tarsal joints were diagnosed with proximal suspensory desmitis.[6] Therefore, MRI protocols should be adopted to allow for complete evaluation of both anatomic regions in horses where pain has been localized to this area and conventional imaging techniques produce negative or equivocal results.

Several clinical reports and one large case series of *in vivo* tarsal MRI have become available in recent years.[6,13,23,33,40,41,57,93,96,131,167,169] The prevalence of MRI lesions of the distal tarsus detected with a high-field magnet was reported for 125 limbs of 103 horses with lameness originating from the distal tarsal/proximal metatarsal area. Osteoarthritic changes of the distal intertarsal and tarsometatarsal joints were the most common findings, including sclerosis of the third and central tarsal bones in 54% and 100% of limbs, respectively, osteophytes at the margins of the distal intertarsal or tarsometatarsal joints in 51% and 70% of limbs, respectively, and articular cartilage damage or subchondral bone lysis of the same joints in 42% and 51% of limbs, respectively. Degenerative changes of osteoarthritis (osteolysis and loss of joint space) were also found in 65% limbs between the central and fourth, central and second, third and second, and third and fourth tarsal bones. Suspensory ligament desmopathy was found in 53% of limbs in this study, suggesting that multiple abnormalities of the distal tarsal joints and the proximal part of the suspensory ligament were frequently identified simultaneously in this population of predominantly Western performance horses. No retrospective MRI studies are available to document the prevalence of injuries of the proximal tarsal region.

MRI Abnormalities in the Tarsal Region

Osteoarthritis

The superiority of MRI over other imaging modalities to detect early changes of osteoarthritis in the tarsus has been clearly demonstrated.[6,26,40,96] Periarticular new bone formation, joint space narrowing, subchondral sclerosis, and subchondral bone lysis may be seen in the tarsometatarsal, distal intertarsal, and rarely proximal intertarsal joints. In a study of 38 Icelandic horses, the most commonly detected MRI lesions observed in 42 distal intertarsal joints classified as osteoarthritic were mineralization front defects, joint margin lesions, and articular cartilage lesions.[96] An association has been reported between MRI abnormalities of the intertarsal interosseous ligament region and osteoarthritis of the distal intertarsal joint.[6,169]

MRI changes of osteoarthritis may also occur in the vertical contact areas between the various tarsal bones (Figure 3.248) and in the talocalcaneal joint (Figure 3.247). However, mild subchondral bone contour irregularities, sclerosis, osteophytes, and intertarsal ligament changes may be seen in non-lame horses, and their presence should not be overinterpreted as clinical disease.



Figure 3.247. Sagittal proton density image of the tarsus of a horse with osteoarthritis of the talocalcaneal joint. There is localized loss of joint space and subchondral bone margins in the center of the joint. This lesion is surrounded by a wide irregular area of signal loss reflecting reactive osteosclerosis in both the talus and the sustentaculum tali of the calcaneus (arrows).

Osseous Trauma

Osseous trauma related to trabecular microfracture, bone edema, or hemorrhage characterized by high STIR signal in bone can occur in the central, third, and fourth tarsal bones[6,13](Figure 3.249) as well as in the talus or calcaneus.[93,202] Osseous fluid in the vicinity of the intertarsal foramen may represent an intertarsal ligament enthesopathy.

The most common finding on MR images of the tarsus of horses with tarsal pain is sclerosis of the central and/or third tarsal bones, characterized by low signal intensity in the medulla and/or subchondral bone region.[6,40]

Fractures

Incomplete or complete vertical fractures of the central (or third) tarsal bone are usually stress fractures and may not always be detected radiographically.[6,40] These fractures are identified on MR images as a line of high signal intensity in most imaging sequences but especially on T1 images, where they are generally surrounded by an area of low signal intensity representing osseous fluid surrounding an acute fracture or sclerosis surrounding stress fractures or chronic fractures.[6] MRI may also be useful for identification of non-displaced

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fractures of the talus or calcaneus that are not readily visible radiographically.[33]

Osseous Cyst-like Lesions

Osseous cyst-like lesions occurred in 22% of limbs in one MRI study of horses with distal tarsal region lameness and were approximately equally distributed between the third and central tarsal bones.[6] They may also occur in the fourth tarsal bone or in the distal tibia at the apex of the parasagittal grooves that articulate with the trochlear ridges of the talus. Osseous cyst-like lesions have intermediate to high signal intensity surrounded by low signal intensity of sclerotic bone. They may be incidental findings in the tarsal bones, especially if there are no signal alterations in the surrounding bone. Reactive signal changes that represent more significant bone damage are osseous fluid or extensive sclerosis in surrounding bone. Osseous cyst-like lesions are usually associated with an articular surface defect.

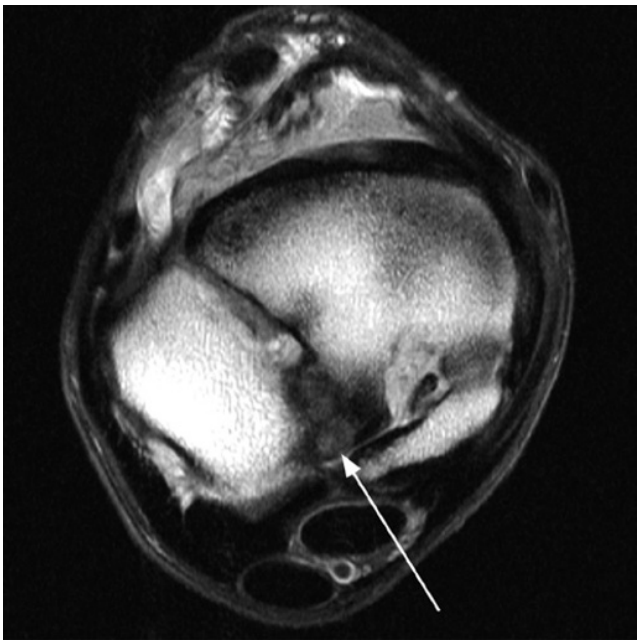


Figure 3.248. Transverse proton density image through the proximal row of tarsal bones of the left tarsus of a horse with focal osteoarthritis of the articulation between the central and fourth tarsal bones. There is loss of joint space and subchondral bone outline surrounded by a margin of irregular osteosclerosis of the fourth and central tarsal bones (arrow).

Intertarsal Ligament Injury

Lesions of the intertarsal interosseous ligament occurred in 33% of horses with distal tarsal region lameness.[6] Lesions have been described as either signal increase[57] or signal decrease[6] in the ligament with loss of normal ligament architecture. Changes of the ligament fossa include thickening and irregular contour of the subchondral bone and narrowing to even total obliteration of the fossa by new bone production. Osseous fluid and/or sclerosis may be present in the third and central tarsal bones either side of the ligament fossa (Figure 3.249). Abnormalities of the ligament and the ligament fossa typically occur concurrently. Severe changes of desmopathy and enthesopathy are invariably accompanied by degenerative changes of the distal intertarsal joint.[6]

Other Soft Tissue Injuries of the Tarsus

Care is required when interpreting signal increase because several ligaments in the tarsus are subject to magic angle artifact, both in standing and recumbent MRI, which can overlap with certain types of injury.[12] Anecdotal reports have illustrated medial and lateral collateral ligament injuries.[167] Hyperintense core lesions of the long plantar ligament may also occur, and

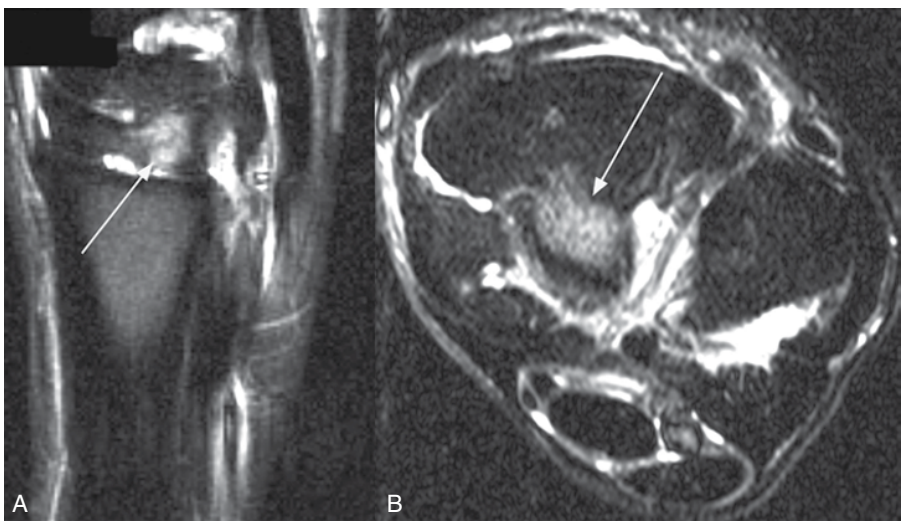


Figure 3.249. Sagittal (A) and transverse (B) short tau inversion recovery (STIR) images of the right tarsus of a horse with acute onset hindlimb lameness. There is abnormal intraosseous signal hyperintensity (osseous fluid) in the plantar aspect of the third tarsal bone (arrows), compatible with bone edema or a bone bruise.

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lameness may be abolished by anesthesia of the deep branch of the lateral plantar nerve.

Injuries of the tarsal sheath portion of the DDFT (lateral digital flexor) have been reported at the level of the distal tarsus[41] and at the level of the sustentaculum tali.[123] Chronic calcaneal bursitis may be accompanied by focal cortical bone loss from the tuber calcis with generalized reactive osseous fluid throughout the proximal portion of the calcaneus.

MAGNETIC RESONANCE IMAGING OF THE STIFLE REGION

Introduction

MRI examination of the stifle has enormous potential for improved diagnosis of injuries of this complex joint as many of the soft tissue structures of the joint cannot be evaluated comprehensively with any other imaging modality. MRI provides a more complete evaluation of the stifle than exploratory arthrosocopy.[87]

MRI of the stifle has rarely been possible in live horses. However, recent equipment improvements both in low- and high-field magnet design have made the stifle accessible to routine MRI examinations under general anesthesia. Clinical MRI of the stifle in selected horses has been possible in ultrashort to short, wide bore (70 cm) high-field magnets (1.5-T Siemens Magnetom Espree™; 3.0-T Siemens Verio™ Open-Bore system, Siemens Medical Solutions Inc., Malvern, PA, USA).[84] Moreover, technical innovation allowing for rotation of open magnets has now made stifle MRI accessible to all horses under general anesthesia with a 0.25-T low-field MRI system (Figure 3.208; Rotating Vet MR Grande XL, Esaote, SpA, Genova, Italy), with the exception of particularly short-legged horses or ponies.[111]

Different stifle MRI protocols have been suggested for high-field[38,77,87] and low-field magnets.[153,186] Contrast enhancement with gadopentetate dimeglumine administered intravenously has also been useful to clarify further lesions in the stifle.[87] Various imaging planes with the joint in flexion and extension have been evaluated.[38]

The MRI anatomy of the stifle has been described in cadaver limbs using both high-field and low-field magnets.[38,77,153] Both systems allowed for detailed evaluation of all soft tissue structures of the stifle. In addition, MR images provided excellent resolution of articular cartilage and subchondral bone.[38,77] The unique shape and fiber orientation of the menisci predisposed to the effects of magic angle, specifically between the deep external and shallow internal margins and at the junction between the horns and the respective meniscotibial or meniscofemoral ligaments.[38] In another study using a 0.25-T low-field open magnet, MR imaging findings in 10 fresh cadaver stifles of asymptomatic horses correlated well with gross and histopathological findings. Irregular areas of signal increase in all patellar ligaments corresponded to adipose tissue infiltrating between the collagen bundles. The authors concluded that the study supported the use of low-field MRI for detection of stifle joint lesions in horses, even though the pathologies observed had been subclinical.[153]

The incidence of MRI lesions of stifles examined in an 0.25-T low-field open magnet was recently reviewed in two studies.[111,187] In one study of 61 horses with stifle lameness, meniscotibial ligament degeneration, fibrillation, and tearing were the most common abnormality (87%), closely followed by osteoarthritis (74%).[111] Degeneration or tearing of a meniscus (56%) or cruciate ligament (59%) were also common. Less frequently observed were bone marrow lesions or bone contusions (30%), tibial enthesopathy of meniscotibial ligament insertions with cystic bone degeneration (18%), subchondral bone defects or osseous cyst-like lesions of the tibial or femoral condyles (21%), and collateral desmopathies (10%). Another study, including 70 horses with stifle lameness without conventional imaging abnormalities, identified meniscal lesions as the most common abnormality (95%).[187] Cartilage fibrillation or defects (66%), bone contusions (50%), and cruciate ligament abnormalities (43%) were also commonly encountered. Although these observations suggest that many of these lesions were not only very common but also present concurrently, the clinical significance of some low-field MRI abnormalities in the stifle has been questioned previously.[153]

MRI Abnormalities in the Stifle Region

Meniscal and Meniscal Ligament Injuries

The obvious advantage of MRI is that it can evaluate the entire meniscus including areas not visible arthroscopically or ultrasonographically, as well as internal lesions that do not reach the articular surfaces. An increase in internal signal or anatomical disruption of the normally hypointense triangular structure of the meniscus is indicative of damage (Figure 3.250). Intrasubstance signal increase without signs of synovitis or cartilage damage is considered a degenerative change.[111] However, it should be remembered that menisci are also prone to magic angle effects as a cause of signal increase.[38] Meniscal injuries are best recognized on PD or T2 sagittal or frontal images.[87] Damage to the meniscotibial or meniscofemoral ligaments can occur in isolation or with concurrent meniscal damage. MRI changes include intra-substance signal increase or loss of architecture, loss of margin definition or fraying, and tears.[111] Osseous resorption and degenerative cyst formation can occur as a manifestation of enthesopathy at the tibial insertions of the meniscotibial ligaments cranial to and on either side of the medial intercondylar eminence of the tibia.

Cruciate Ligament Injuries

It has been stated that both cruciate ligaments should be uniformly hypointense on PD and T2 images and that the caudal cruciate ligament can be used as a reference structure to assess signal change in the cranial cruciate.[87] However, others have claimed that the cranial cruciate ligament has low to intermediate signal intensity in its proximal half, while its distal half has intermediate to high signal corresponding to twisting of the ligament fibers from proximal to distal and the presence of shallow striations and fanning of the ligament.[38]

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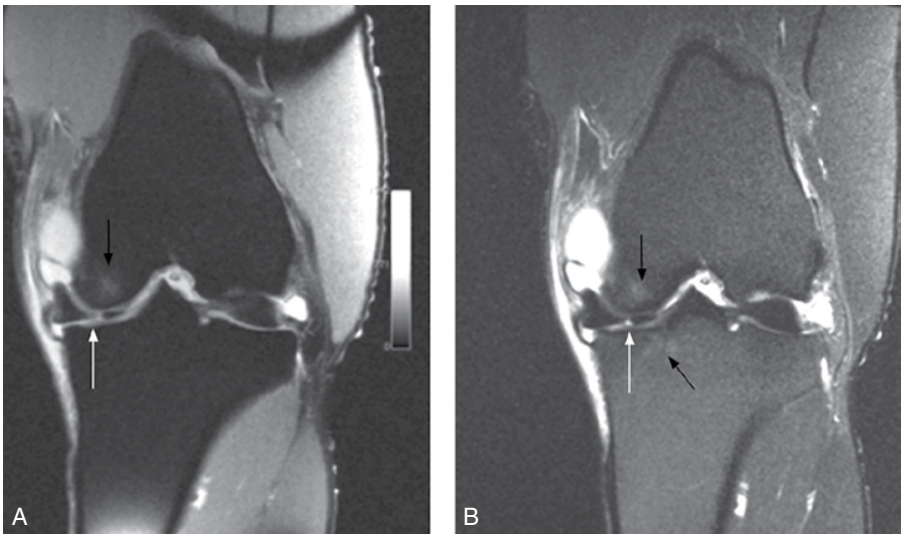


Figure 3.250. Dorsal proton density image with fat saturation (A) and dorsal short tau inversion recovery (STIR) image (B) of the left stifle of a horse with a tear of the cranial horn of the medial meniscus (white arrow). There is intraosseous signal hyperintensity in the medial femoral condyle and the axial margin of the medial tibial plateau suggestive of abnormal bone fluid (black arrows). *Source*: Courtesy of Dr. Carter Judy.

Moreover, the cranial cruciate ligament is supposed to be heavily subjected to magic angle artifact in closed high-field magnets with the patient in lateral recumbency.[87] Magic angle effect was also consistently seen in the cranial cruciate ligament with the horse in dorsal recumbency and the stifle positioned in extension in an open low-field 0.25-T magnet. Therefore, the internal signal of the ligament should never be assessed on T1 images only,[87] but evaluation should also include short TE sequences. Thickening of the cranial cruciate ligament with internal signal changes in T1 and PD images has been described as the most consistent finding in cranial cruciate ligament injury.[87] Increased STIR or T2 signal was rarely present, possibly because of the chronic course of the lameness in such patients before MRI was undertaken.[87] Other MRI findings associated with cruciate ligament injury include joint effusion and osseous fluid at the femoral origin and/or tibial insertion of the affected cruciate ligament.[87,111] Although one low-field study almost always found concurrent meniscal abnormalities in horses with cruciate ligament injury,[187] others have not shared this observation using high-field MRI.[87] Caudal cruciate ligament injuries are generally considered to be rare.[87]

Bone Contusions

Bone contusion, bone edema, or bone marrow lesions are a common finding in the femoral and tibial epiphyses, either as a primary abnormality or in association with other injuries of the stifle (cruciate or meniscal injuries, osteochondritis dissecans, cartilage erosions, and osseous cyst-like lesions).[111,187,188] Primary bone contusion generally develops as a consequence of subchondral bone trauma, and osseous fluid signal typically begins in the subchondral region and radiates into the trabecular bone of the medullary cavity.

Cartilage Lesions

Cartilage erosions are readily visible in the stifle thanks to the innately thicker cartilage than in distal limb joints. Focal cartilage erosions are more easily recognized than generalized thinning of degenerative cartilage. Invariably cartilage erosions are accompanied by an increase in subchondral fluid and STIR signal intensity.[87]

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THERMOGRAPHY

TRACY A. TURNER

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Thermography is the pictorial representation of the surface temperature of an object.[9,21] It is a noninvasive technique that measures emitted heat. A medical thermogram represents the surface temperatures of skin making thermography useful for the detection of inflammation. This ability to noninvasively assess inflammatory change makes thermography an ideal imaging tool to aid in the diagnosis of certain lameness conditions in the horse.[1,9,10,12–14,20,21,24]

Thermography was first used in veterinary medicine 45 years ago. Originally, its use was limited for the most part to university hospitals or large referral practices. But over the last 20 years, thermography has been used practically in equine medicine. This is a physiological imaging modality that requires a learning curve for interpretation, but it offers the operator new insight into their patients.

THERMOGRAPHIC INSTRUMENTATION

Thermographic instrumentation in the past has been divided into contacting and noncontacting devices.[15] However, in the 1990s, a new technology using focal plane array detectors made all older thermographic devices obsolete with regard to equine veterinary use. Focal plane array detectors are uncooled technology. This technology employs special lenses to focus infrared radiation on a series of detectors. These instruments are self-contained and highly portable.

One of the most important factors to consider before purchasing a thermographic camera is the spectral range.[15] For medical use the range of 8–14 μm is ideal because this is the peak emissivity of skin. From a practical standpoint, there is also less environmental artifact at this range. The author prefers real-time thermography versus still thermography because real-time thermography eliminates any problems with motion, makes thermographic assessment more dynamic in that the operator can immediately observe change, and allows for faster imaging. Sensitivity refers to the amount of temperature difference that can be detected; uncooled units can differentiate 0.1 °C, which is sensitive enough for medical uses. The final factor is portability and durability. Uncooled cameras utilizing the focal array technology are very portable and durable because of no moving parts.

PRINCIPLES OF USE

The circulatory pattern and the relative blood flow dictate the thermal pattern, which is the basis for thermographic interpretation.[6,8,21] The normal thermal pattern of any area can be predicted on the basis of its vascularity and surface contour. Skin overlying muscle is also subject to temperature increase during muscle activity. Based on these findings, some generalizations can be made regarding the thermal patterns of a horse: The midline will generally be warmer,[9,21] and this includes the back, the chest, between the rear legs, and along the ventral midline (Figure 3.251). Heat over the legs tends to follow the routes of the major vessels, the cephalic vein in forelimb, and the saphenous vein in the hindlimb.

On the dorsal view of the distal limb, the metacarpus (metatarsus), fetlock, and pastern appear relatively cool because the image recorded is away from the major blood supply. Thermographically, the warmest area in the distal limb is around the rich arteriovenous plexus of the coronary and laminar corium located proximally on the hoof wall. Normally, there is increased warmth between the third metacarpus and flexor tendons, following the route of the median palmar vein in the forelimb and the metatarsal vein in the hindlimb. Over the foot, the warmest area corresponds to the coronary band. From the palmar (plantar) aspect, the tendons are relatively cool, and the warmest area is consistently

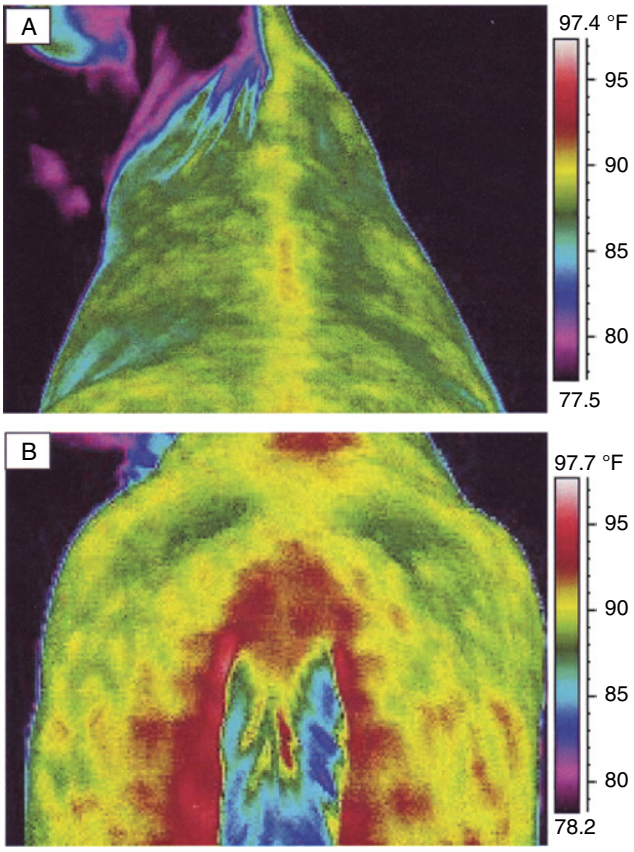


Figure 3.251. Normal back (A) and croup (B) thermograms of a horse. Note the back is warm down the center with isothermic bands around it. The croup forms a "T" of uniform temperature between the tuber coxae and the midline.

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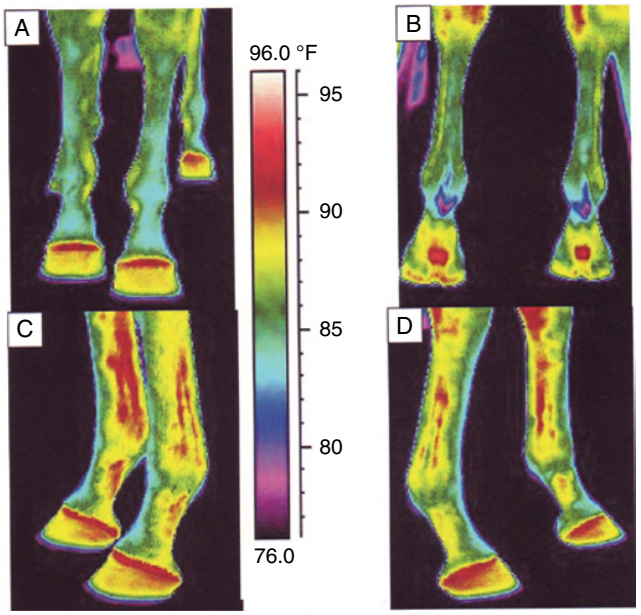


Figure 3.252. Normal distal limb of a horse. The warmest areas follow the vasculature. Abnormalities are heat where it is not expected or changes in the normal thermal pattern. Essentially, if the image is not normal and cannot be explained as an artifact, then it represents abnormal thermography. (A) Dorsal view. (B) Palmar view. (C) Left side. (D) Right side.

between the bulbs of the heel along the midline (Figure 3.252).

Injured or diseased tissues will invariably have an altered circulation.[21] One of the cardinal signs of inflammation is heat, which is due to increased circulation. Thermographically, the "hot spot" associated with the localized inflammation will generally be seen in the skin directly overlying the injury.[1,3–5,9–14,20,21,23–25] However, diseased tissues may in fact have a reduced blood supply either due to swelling, thrombosis of vessels, infarction of tissues, or change in sympathetic tone.[14,21] With such lesions the area of decreased heat is usually surrounded by increased thermal emissions, probably due to shunting of blood.

To produce reliable thermographic images, certain factors need to be controlled: motion, extraneous radiant energy, ambient temperature, and artifacts.[15] Motion can be controlled by immobilizing the horse in stocks or using a qualified handler. The use of real-time thermography eliminates the need for complete immobilization. Chemical restraining agents to keep the horse from moving should be avoided because these drugs affect the peripheral circulation and cardiovascular systems that could cause false thermal patterns to be produced; however, the author has not encountered this. To reduce the effects of extraneous radiant energy, thermography should be performed under cover shielded from the sun.[21] Preferably, thermography should be done in darkness or low-level lighting. Ideally, ambient temperature should be in the range of 20 °C (68 °F), but any temperature as long as the horse is not sweating is acceptable. Heat loss from sweating does not occur below 30 °C (86 °F), as radiation and convection are responsible for heat loss below that temperature. Very cold environmental temperatures may cause vasoconstriction of the lower legs and interfere with imaging. In these cases, low-level exercise to stimulate vasodilatation is necessary. The thermographic area ideally should have a steady, uniform airflow so that erroneous cooling does not occur. Practically, the horse should be kept from drafts. Likewise, the horse should be allowed 10–20 minutes to acclimate to the environment or room where thermography is performed. Artifacts are extraneous sources on the skin that can cause irregular images. Among these are debris, scar tissue, hair length, liniments, leg wraps, and equipment.[21] To avoid artifacts, make sure all subjects are groomed and free of leg wraps and equipment for 2 hours whenever possible. Hair insulates the leg and blocks the emission of infrared radiation. But, as long as the hair is short and of uniform length, the thermal image produced is accurate. The skin should always be evaluated for changes in hair length that may cause false "hot spots" in the thermogram.

Multiple thermographic images of a suspect area should be made.[25,26] The area in question should be evaluated from at least two directions approximately 90° apart, to determine if a "hot spot" is consistently present. The horse's extremities should be examined from four directions (circumferentially).[21] Significant areas of inflammation will appear over the same spot on each replicate thermogram.

There are at least four ways in which thermography can be utilized in equine veterinary practice. The first is as a diagnostic tool. In these cases, thermography is a physiologic imaging method where a 1.0 °C difference between two anatomically symmetrical regions indicates a region of inflammation.[15] A decrease in temperature is just as important as an increase in temperature. The image will identify an area of interest to pursue with an anatomic imaging method such as ultrasonography and/or radiography.

The second method is to enhance the physical examination. In this case, thermography is used to identify changes in heat and therefore locate "areas of suspicion".[15] Thermographic cameras are easily 10 times more sensitive than the hand in determining temperature differences. This method simply helps identify asymmetry, and then the practitioner must utilize the information to determine the actual cause and significance of the temperature difference.

The third method of using thermography is in a wellness program. In this method, horses in training are followed on a routine basis, once weekly.[22] In the author's experience, thermographic changes often occur 2 weeks prior to clinical changes. In these cases, thermography can be used to identify subclinical problems, and then training alterations can be made so that injury may be avoided altogether.

The fourth method is in the regulation of equine events.[15] Presently, thermography is used by the *Federation Equestre Internationale* (FEI) and the *United States Department of Agriculture* (USDA) to enforce regulations. The instrument is used as a screening tool to determine potential misuse. In these cases, the final determination is made by a group of examining veterinarians.

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SPECIFIC APPLICATIONS FOR LAMENESS DIAGNOSTICS

The Examination

If thermography is to be used, all bandages and leg wraps should be removed 2 hours before the examination. The horse should be thoroughly brushed to remove any dirt and debris. Thermography is best performed after the clinical examination has been performed (including watching the horse move, palpation, and manipulations) but before nerve or joint diagnostic analgesia. The horse is brought into a covered area away from direct sunlight and any breezes. A 10-minute acclimatization period is allowed, and then a systematic examination of the horse is performed using the thermographic camera.[15]

The camera is positioned 90° to the area of interest, and the sequential images are taken. The limbs are examined from all four directions, the neck from both sides (ventrally), the torso from both sides (dorsally and ventrally), the shoulders from the front and both sides, the hips from both sides and the back, and the head from both sides (dorsally and ventrally). Each of the images is then assessed in relation to the findings of the clinical examination of the horse.

The Foot

Thermography of the hoof has been useful for the diagnosis and evaluation of several conditions of the foot.[9,13,21] Laminitis, palmar foot pain, subsolar or submural abscesses, corns, and any other inflammatory condition of the hoof are specific conditions where thermography provides valuable information concerning the disease. Obviously, thermography is not needed to definitively diagnose these problems. Thermography does however provide additional information that is helpful in localizing the problem, assessing the degree of inflammation associated with it, and deciding the best course of treatment. Thermographic evaluation is particularly helpful with early or occult conditions of the foot, where the physical and/or radiographic examination findings are inconclusive.

Normally, the coronary band is the warmest area of the leg, and because of this, inflammation of this area can be difficult to detect.[9] The hooves should be thermographically compared in several ways. Comparisons should be made between all four hooves, front to front and front to rear. A difference of more than 1 °C between hooves is significant. In cases where all 4 feet are involved, comparisons of the hoof temperature with the temperature of the area between the bulbs of the heel should be made.

Laminitis is characterized by inflammation of the laminar structures of the hoof. Laminitis can be divided into four phases—developmental, acute, chronic, and post-chronic—and each of the phases has characteristic thermal information. The developmental stage has been studied thermographically the least. During the developmental stage there are no clinical signs, but the thermal image changes. There are progressive periods of vasoconstriction followed by periods of profound vasodilatation; the heat seen gets hotter and broader following each vasoconstrictive period. During the acute phase a change in the thermal pattern of the hoof wall is useful in recognizing laminitis. Generally, the coronary band is 1–2 °C warmer than the remainder of the hoof. An inflammatory problem is indicated when the hoof begins to approach the temperature of the coronary band. Thermography can also be very beneficial in the progressive evaluation of the contralateral limb where support limb laminitis is potential sequelae. Thermography makes it possible to detect inflammation in the contralateral foot well before lameness is evident. Preventative therapy can therefore be instituted sooner in the course of disease and hopefully before the laminitis is irreversible. The chronic phase is characterized by progressive displacement of the third phalanx. The thermal image is the opposite of the acute phase. As the coffin bone displaces, the vasculature is pulled further away from the surface causing the thermal image to appear progressively cooler. That is, the coronary band is more difficult to distinguish from the remainder of the hoof wall. Finally, the post-chronic phase is seen when the laminae have healed, but vascular changes (damage) persists secondary to coffin bone displacement. Thermal imaging can be useful to identify the vascular damage that has occurred. These will be areas of intense cold, which will identify the areas of vascular damage.

Thermography is also an excellent method of evaluating the patient with palmar foot pain syndrome.[20] The author has never identified a consistent thermal pattern associated with these cases but has been able to characterize reduced blood flow to the caudal hoof and to identify thermal stresses associated with hoof imbalance and superficial inflammatory disease. Thermography is one of the few methods that can readily determine the relative blood flow to the palmar foot area. This is accomplished by evaluating the foot thermographically both before and after exercise. The normal horse will sustain a 0.5 °C increase in temperature of the foot after exercise, but about 40% of horses with palmar foot pain syndrome will not sustain this increase in the caudal foot due to the low blood flow. This is in sharp contrast to other focal inflammatory conditions of the hoof such as bruises, or fractures, which are characterized by focal areas of increased temperature that correspond to the site of injury. Exercise in these cases intensifies the "hot spot."

Hoof abscesses before they drain are often seen as relatively cold areas. This is due to the pressure caused by the abscess that decreases the circulation to the abscessed area. The hoof capsule is a closed environment, and the swelling that occurs due to the abscess is seen thermographically as an abnormal cold area. Once the abscess opens and the pressure is relieved, the cold area changes to a hot area.

Joint Diseases

Joint inflammation produces characteristic thermal patterns. The best view to study most joints is from the dorsal aspect.[13,23] Typically, the normal joint is cool compared with the surrounding structures. An exception to this rule is the hock, which has a vertical "hot spot" along the medial aspect that corresponds to the saphenous vein (Figure 3.253). As a joint becomes inflamed, the thermal pattern changes to an oval area of increased

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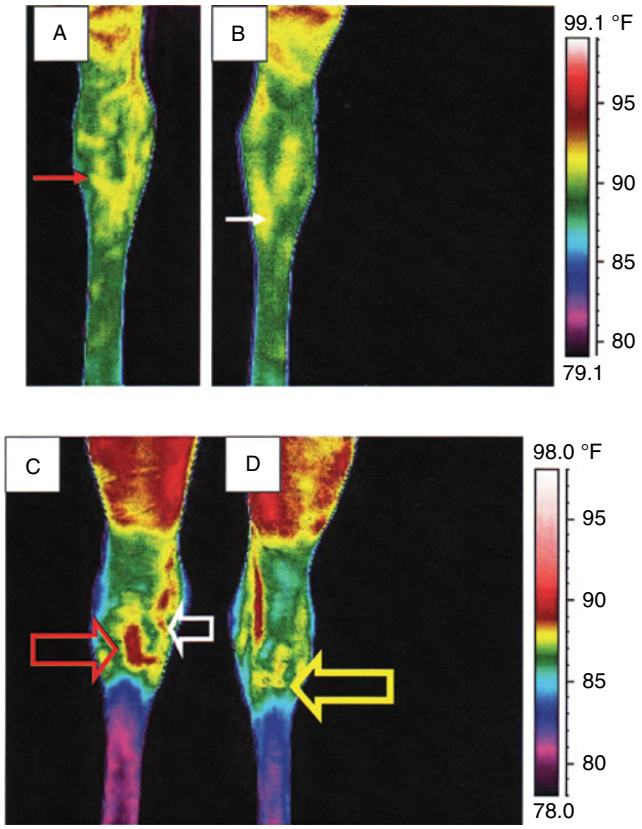


Figure 3.253. Thermal views of the dorsum of the hock of two different horses. (A) Right hock of horse 1. (B) Left hock of horse 1. (C) Right hock of horse 2. (D) Left hock of horse 2. (B) is the most normal thermogram; the white arrow indicates the saphenous vein and the warmest area. Image (A) shows a horizontal line (red arrow) indicating early lower tarsal joint inflammation. Images (C) and (D) show variations of distal hock inflammation. Image (C) shows the horizontal line and area over the central hock, which are hotter (hollow red arrow) than the area of the saphenous (hollow white arrow). Image (D) shows that the distal hock is a horizontal line (hollow yellow arrow).

temperature that is centered over the joint and widest horizontally medial to lateral. The exception to this rule is the joints of the distal limb, where the thermal pattern associated with inflammation of this joint is a circular pattern. The areas of joint capsule attachment tend to be "hotter," but the center of the joint is relatively "cooler". This may be due to joint swelling or pressure and subsequent loss of microcirculation (Figure 3.254). No specific correlation can be made between heat and joint damage. The temperature of the joint appears to be related to many factors: the chronicity of the problem (the more chronic the problem, the less heat), the degree of synovial involvement, the actual amount of cartilage damage, and the presence or absence of osteochondral fragments. These factors have a complicated interaction, and all affect the inflammatory response of the joint temperature. The degree to which each affects this response has yet to be determined.

Thermal patterns of joints have been shown to change 2 weeks before the onset of clinical signs of lameness.[22,24] In this manner, thermography can be used to assist training and help prevent serious injuries. By locating inflammation before clinical signs are evident, training programs can be changed to reduce stress on the inflamed area, thereby preventing serious injury.

Long Bone Injuries

Thermography is of less value in the diagnosis of most long bone problems. Since thermography evaluates skin temperature, a bone needs to be in relatively close contact with the skin to affect its temperature.[3] Consequently, bones that are heavily covered with muscle cannot be as accurately assessed by thermography. Thermography is best utilized to evaluate dorsal metacarpal disease or stress fractures of the radius or tibia.

Dorsal metacarpal disease, the so-called bucked shin complex, is categorized into three grades.[7] Grade 1 is characterized by eliciting pain upon palpation of the cannon bone, but radiographic evidence of bone pathology cannot be identified. Grade 2 is characterized by pain over the cannon bone, but there is radiographic evidence of subperiosteal callus. Finally, grade 3 is characterized by cannon bone pain and radiographic evidence of a stress or fatigue fracture. Grades 2 and 3 may be indistinguishable, and radiographic confirmation of a stress fracture may not be possible for 2–3 weeks. Thermal variations between the latter two may help differentiate grade 3 lesions earlier than radiographs. Grade 1 and 2 diseases are characterized by "hot spots" located midshaft over the dorsal cannon bone. The "hot spot" is generally 1–2 °C warmer than the surrounding tissues. In contrast, the grade 3 disease has "hot spots" that are not centrally located and are usually seen on the lateral and medial views in addition to the dorsal view. These areas are characteristically 2–3 °C warmer than the surrounding tissues. Since thermographic changes typically precede radiographic changes by 2 weeks,[13] with accurate thermography, a tentative diagnosis can be made earlier and appropriate treatment measures taken sooner.

Tendon Injuries

Thermal patterns of the normal flexor tendons are bilaterally symmetrical and consist of elliptical isothermic zones.[11,12] The lowest temperature is centered over the palmar aspect of the tendons, and the peripheral areas near the carpus and fetlock are approximately 1 °C warmer.

Acute tendinitis invariably causes a "hot spot" over the site of the tendon lesion[12] (Figure 3.255). The "hot spot" of a tendon lesion can usually be demonstrated up to 2 weeks before physical evidence of swelling and pain around the tendon. Therefore, tendon lesions of potentially clinical significance can be identified, and adjustments in the training protocol can be made to prevent further damage to the tendon.

As the tendon heals, the thermal pattern becomes more uniform but remains abnormally elevated, when compared with normal tendon.[12] As the lesion heals and scar tissue is deposited, the skin over the injured area may actually show a decrease in temperature, whereas the remaining neovascularized tendon continues to have increased thermal emissions. During the assessment of

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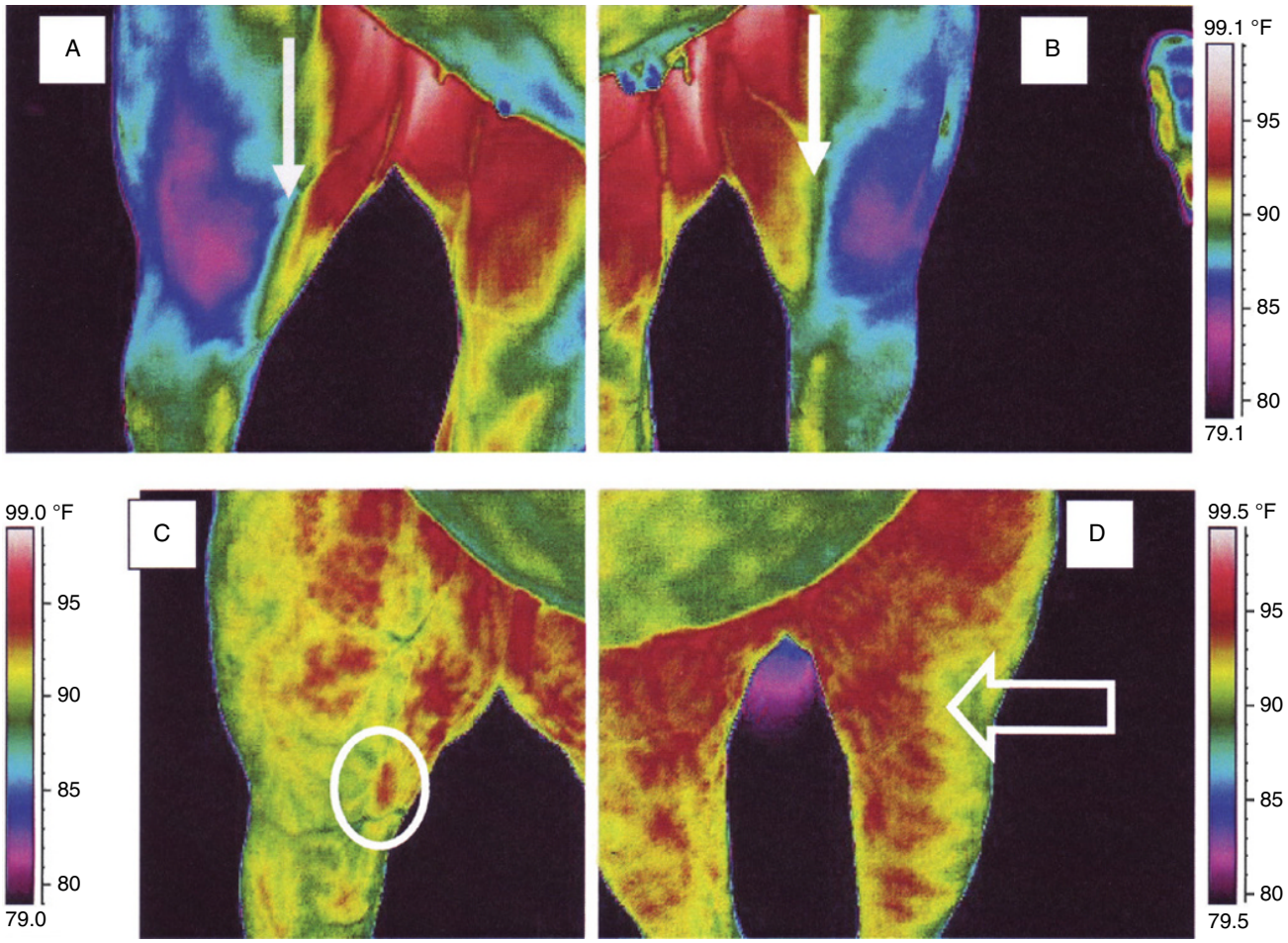


Figure 3.254. Normal right (A) and left (B) cranial stifle thermograms. The thermal pattern on the inside of the stifle is warm, and there is a line of demarcation in the area of the medial patellar ligament (arrow), and the front of the stifle is relatively cool. (C) and (D) represent abnormal thermograms of two different horses. Horse (C) shows a focal hot spot (circle) over the distal medial stifle; radiographs revealed a cyst-like lesion in the medial tibial plateau. Horse (D) shows a pattern that has heat from the medial side covering half the cranial aspect (hollow arrow). This horse had very noticeable effusion but no radiographic changes.

healing, the thermal changes do not correlate well to the structural reorganization of the tendon matrix as assessed by ultrasonography.[2,4] The reason is that as the tendon undergoes neovascularization, the thermal pattern diffuses, so there is no longer a "hot spot". But, if one compares healing tendon to a normal tendon, there is overall increased thermal emissions from the damaged tendon. Mechanical stress proximal to the injury can aggravate the existing tendon damage. Again, thermography can detect these areas of proximal stress before they cause a clinical problem, and therefore specific imaging can be used to decide if a therapeutic desmotomy should be performed.

Ligament Injuries

Thermographically, ligament injuries will appear very similar to tendon injuries. "Hot spots" can be expected to be centered over the injured area (Figure 3.255). An exception to this is in some high suspensory injuries of the metacarpi; the dorsal thermal image of the injured leg shows a focal "hot spot" located proximally on the cannon bone. This is interesting considering the inflammation and pain would be expected to be on the palmar aspect of the limb. Clinically, thermography is most useful when trying to correlate if there is heat associated with a sensitive ligament. This is particularly true of the suspensory ligament where the clinical significance of palpable sensitivity within the body of the ligament can be difficult to determine. Thermography can be used to determine if there is inflammation associated with the sensitivity. Similarly, "splints" or metacarpal callus can cause suspensory desmitis, and thermography can detect if there is inflammation associated with the suspensory ligament adjacent to the "splint." These indications would apply to any ligament.

Muscle Injuries

Thermography may have its greatest clinical application in the assessment of individual muscle injuries that are difficult to diagnose.[14,19] Even though serum muscle enzyme elevation may nonspecifically indicate muscle damage, the specific muscle or muscles damaged may be difficult to identify. Thermography offers two types of information important in the evaluation of muscle

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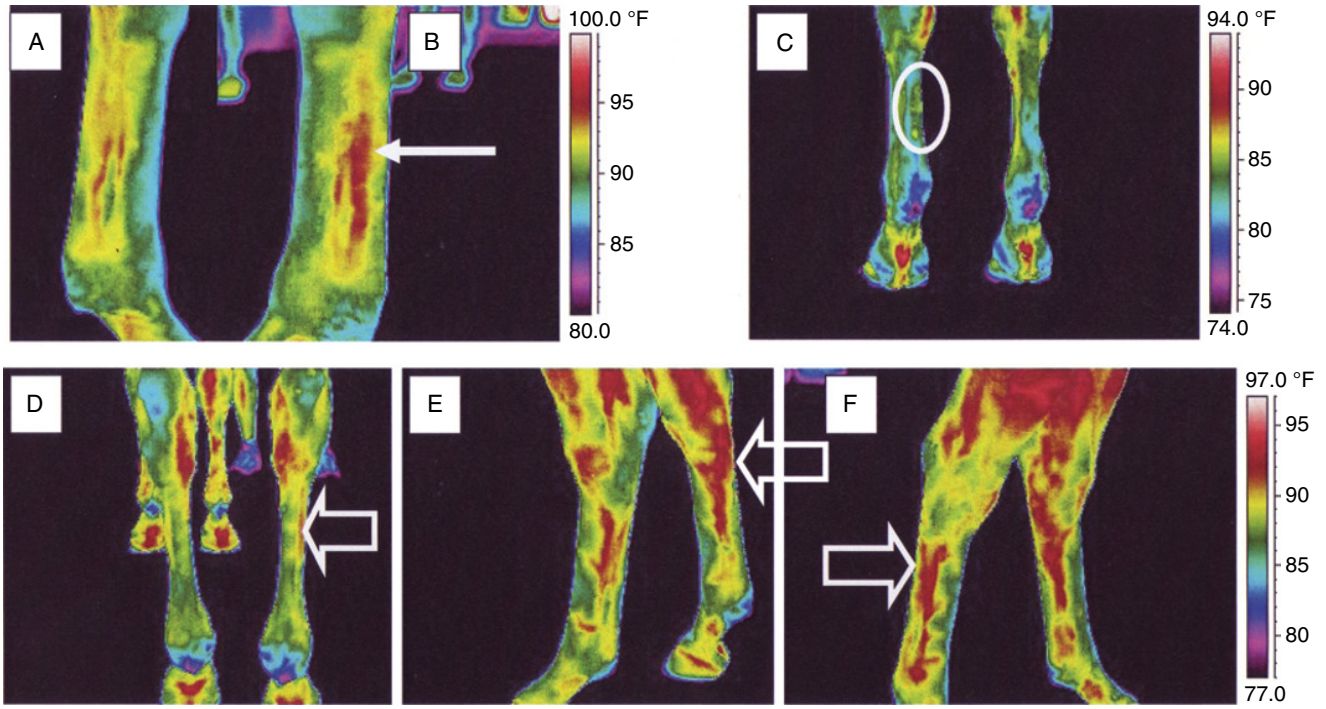


Figure 3.255. Abnormal thermograms. (A) Normal medial aspect of the left fore of a horse. (B) Abnormal medial aspect of the right fore of the same horse. The hot area (arrow) centered over the suspensory ligament was shown via ultrasonography to be due to suspensory desmitis. (C) Superficial digital flexor tendon lesion (circle) was confirmed sonographically. (D–F) Three views of the same horse showing a high suspensory ligament lesion confirmed ultrasonographically (hollow arrows).

injury: First it can locate an area of inflammation associated with a muscle or muscle group, and second it can illustrate atrophy well before it becomes apparent clinically.

Muscle inflammation will be most commonly seen thermographically as a "hot spot" in the skin directly overlying the affected muscle.[14,19] On a rare occasion, swelling and edema in the affected muscle will be severe enough to inhibit blood flow through the muscle. In this case the injured muscle will be seen thermographically as a "cold spot." Thermographic evaluation of muscle must be made from the right and left sides. These comparison images should be nearly identical. Consistent variations from side to side would indicate muscle damage located at either the "hot" or "cold spot."

The most common cause of muscle inflammation is muscle strain.[19] A classification of first-, second-, or third-degree strain injuries, described in human athletes, has been applied to horses.[14] Muscle strains have not been commonly documented in the forelimb. The author has most commonly identified pectoralis and shoulder extensor myopathies. Thermographic description of muscle strains of the back and hindlimb muscles have been best described.[14,19] These strains have been termed croup and caudal thigh myopathies. Croup myopathies are actually strains of the longissimus, the origin of the gluteus medius (level of the sacroiliac), the body of the gluteus medius, the insertion of the gluteals on the greater trochanter, and the third trochanter of the femur. Caudal thigh myopathies consist of injuries to the biceps femoris, semitendinosus, or semimembranosus muscles. Injuries to the biceps femoris and semimembranosus most commonly are midbody muscle strains, but semitendinosus injuries usually occur at the musculotendinous junction.

Back Problems

Diagnosis of injuries to the vertebral column can be aided through thermography.[13] Many of these injuries are undiagnosed, or the diagnosis is delayed because radiography of the equine spine is difficult and/or may require general anesthesia. Thermography offers a distinct advantage as it is best performed on the standing animal and in suspect cases may be used as a general screening test to determine if referral for radiography is warranted. Injuries to the vertebral column are characterized by either "hot spots," "cold spots," or "root signatures."

Thermography has been used to identify six different back injuries: overriding dorsal spinous processes (kissing spines), dorsal spinous ligament injuries, muscle pain, withers injuries, sacroiliac problems, and saddle fit problems.[16] The author uses two different thermal images to assess the back: one a thoracolumbar view and the second a croup view.[16] The thoracolumbar view shows the withers and the sacrum and is especially good for looking at the mid back region. The croup view is best for evaluating the sacroiliac region. The thermal pattern is the most important aspect of assessing the thermogram of the back. It must be remembered that thermography establishes the location of a possible problem but does not characterize the lesion. However, there are certain thermal patterns that have been seen consistently with particular back problems.

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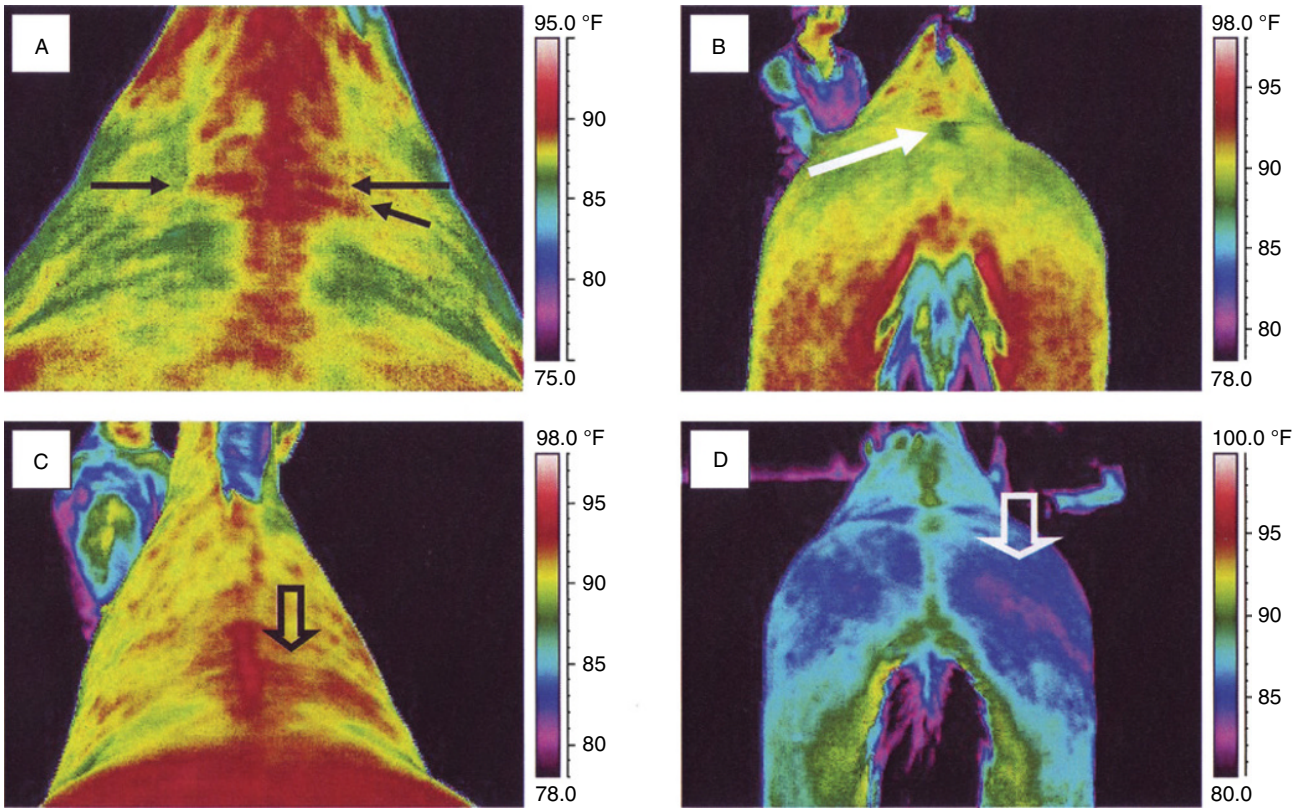


Figure 3.256. (A) Thermogram typical of overriding spinous processes in the horse's back. Note the horizontal hot spots (black arrows); overriding spinous process was confirmed radiographically. (B) Thermogram depicting a cold area over the tuber sacrale (white arrow). This is seen whenever the horse does not bend its back normally and has been associated with thoracolumbar and sacroiliac pain. This horse had evidence of sacroiliac desmitis on ultrasonography. (C) A back with heat over the right epaxial muscles (hollow black arrow), which correlated with palpable pain on the same area. (D) Nonspecific thermal pattern over the croup that shows thermal asymmetry with the right side being colder (white hollow arrow). When this pattern is seen, the lame leg is the cold leg and is thought to be associated with decreased use.

The normal back thermal pattern is warmest down the midline, cooling slightly in the lumbar region, warm from tuber coxae to tuber coxae and over the tuber sacrale, and the same warmth down the middle of the croup (Figure 3.251).[16] The warm area has symmetric isothermic bands on either side. Any variation of this pattern is either due to an artifact (thin hair, rubbed area) or pathologic process. If the lesion occurs along the midline of the thoracolumbar area, radiographs of the thoracolumbar spine are indicated. If the lesion occurs off the midline or in the croup region, we usually image the area with ultrasonography.

Overriding spinous processes or kissing spine has been associated with any one of three different thermal patterns.[18] Pattern 1 is a hot streak perpendicular to the thoracic spine (Figure 3.256), pattern 2 is a cold streak perpendicular to the thoracic spine, and pattern 3 (the most common) is a combination hot spot–cold streak pattern over the back. Our practice has evaluated over 150 cases over a 5-year period of time and has determined that the sensitivity of thermography to diagnose overriding spinous process is 99%; however, the specificity was only 70%. This resulted in a positive predictive value of 91% compared with the positive predictive value of palpable pain in the thoracolumbar area for kissing spine of 67%.

Dorsal spinous ligament injuries have not presented with that type of pathognomonic thermal pattern.[16] Thermographic changes associated with this injury have been increased heat, decreased heat, or simply an abnormal back thermogram. The lesion in our hands has been detected using ultrasonography after knowing there is an abnormal thermogram.

Muscle injuries of the thoracolumbar region also do not have characteristic thermograms.[16] Typically, however, the thermal patterns will show either hot spots or cold spots off the midline (Figure 3.256). It is in these regions we have concentrated sonography to determine if muscle lesions can be seen. Withers injuries have all shown hot spots in the area of the withers but nothing more characteristic than that. The croup muscles will show some specific changes.[14]

Sacroiliac region thermography has shown several different patterns.[16] The most common pattern is a cold area centered over the region of the tuber sacrale (Figure 3.256). This finding of cold is hypothesized to be due to lack of normal movement in the sacroiliac region. This lack of movement can be either due to primary pathology or secondary to other causes of the horse not moving normally through the pelvic region. This has been consistent with our clinical findings as well. Specifically, we have found that only about half the horses exhibiting this thermal pattern actually show

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pain in the sacroiliac area. In addition, sonographic evaluation reveals pathology in only about half the cases, and this pathology usually looks chronic in nature. We have identified both thinning and thickening of the cross-sectional diameter of the dorsal sacroiliac. In cases with a cold area and palpable pain, we will sonographically examine the sacroiliac region. On the other hand, if there is no pain or the horse moves normally through the pelvis, we look for other causes of loss of back mobility. Pathology is seen much more commonly if the area over the tuber sacrale is hot or if there is a hot spot centered over one tuber sacrale or the other. The pathology varies from hypoechoic areas within the dorsal sacral ligaments to generalized edema in the region. This thermographic pattern is almost always associated with either pain or marked stiffness in the sacroiliac region.

Saddle fit thermography is very interesting and requires multiple examinations.[17] In evaluating the dynamic interaction between the saddle and the horse's back, thermography will show not only the heat generated in contact areas on the saddle but also the physiologic effects of the saddle on the horse's back. Our protocol is to perform a baseline thermographic examination of the horse's back and again after the horse has been worked under saddle. The horse should be saddled with a simple cotton pad with the girth tightened as it would be for riding. The horse is then lunged for at least 20 minutes. The horse should be exercised at its normal gaits (walk, trot, and canter) and equally in both directions. Both the bottom of the saddle and the horse's back are evaluated. The most important criterion for the saddle is thermal symmetry. In regard to the horse, due to the heat generated by the saddle, the horse's midline is now colder than the other structures under the saddle. In addition, the examiner is looking for focal hot spots, particularly along the spine, or hot or cold spots over the musculature. These abnormalities indicate problems caused by the saddle. The assessment is then repeated after a similar exercise session with the rider mounted. This evaluation allows consideration of the effect of the rider on the horse's back.

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CHAPTER

Lameness of the Distal Limb

NAVICULAR REGION/PALMAR FOOT

Randy B. Eggleston and Gary M. Baxter

NAVICULAR SYNDROME/DISEASE

Navicular disease or syndrome remains one of the most controversial and common causes of intermittent forelimb lameness in horses between 4 and 15 years of age.[35,115,121] It is estimated that the syndrome is responsible for one-third of all chronic forelimb lameness in horses.[23,115] Quarter horses, Thoroughbreds, and Warmbloods, particularly geldings, appear to be at greatest risk, whereas the syndrome is rarely diagnosed in ponies or Arabian horses.[35,115] A recent study that evaluated a large population of horses with foot lameness in the United Kingdom also indicated that there was increased risk of foot pain associated with breed, occupation, age, and body weight–height ratio.[83] The disease has been shown to have a hereditary predisposition possibly due to limb conformation of the horse or to the specific shape of the navicular bone.[12,30,31] The shape of the proximal border of the navicular bone has been determined to be inherited in Dutch Warmblood horses, and horses with an undulating or concave proximal border are more predisposed to the disease.[30,31,96] Other factors such as faulty conformation, hoof imbalances, improper or irregular shoeing, and exercise on hard surfaces are also believed to predispose and aggravate the condition.[88,115,121] Although the hindlimbs can be affected, it is rare, and navicular disease is primarily considered a problem of the forelimbs.

There is some debate as to the proper nomenclature to use when diagnosing a horse that blocks to palmar digital (PD) anesthesia. The early diagnosis of navicular disease ("disease" implies a specific sign or symptom and a specific location) was based on abnormalities present on the navicular bone seen radiographically. However, a significant number of affected horses had normal radiographic findings, which led to the suspicion of soft tissue injuries, giving rise to the term navicular syndrome.[2,100,101] Many horses with bilateral foot pain have concurrent soft tissue and navicular bone abnormalities, also suggesting that navicular syndrome may be more appropriate in the majority of horses. The term "palmar foot syndrome" has also been recommended based on the variety of pathological entities that can be identified in lame horses that improve following PD anesthesia (Box 4.1).[35,46,96] This diagnosis seems most appropriate for those horses that do not have definitive navicular bone involvement or disease. Making a diagnosis of navicular syndrome or palmar foot syndrome is a bit arbitrary, but it avoids labeling horses as having navicular disease when the lameness could be due to many other sources in the foot. As our imaging capabilities have improved, there has been a growing appreciation for the variety of soft tissue abnormalities that are associated with palmar foot lameness, with or without concurrent navicular bone involvement.[39,100,101]

Navicular disease/syndrome has been defined as a chronic forelimb lameness associated with pain arising from the navicular bone and closely related structures including the collateral suspensory ligaments (CSLs) of the navicular bone, distal sesamoidean impar ligament (DSIL), navicular bursa, and the deep digital flexor tendon (DDFT).[35,36,42,100,101] Horses with primary DDF tendinitis within the foot are usually not considered to have navicular disease/syndrome.[35] The disease is characterized by degenerative changes in the structure, composition, and mechanical function of the cartilage, subchondral bone, and surrounding soft tissues of the

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Box 4.1 Abnormalities that may exist in horses classified as having navicular disease, navicular syndrome, palmar foot syndrome, or palmar heel pain

1. Navicular disease: radiographic, CT, or MRI abnormalities within the navicular bone
2. Desmitis/trauma of the podotrochlear apparatus
   a. Collateral suspensory ligaments (CSL) of the navicular bone
   b. Desmitis of the distal sesamoidean impar ligament (DSIL)
   c. Desmitis of the distal digital annular ligament
3. Tendonitis of the DDFT: usually at three locations
   a. The insertion
   b. Palmar to the navicular bone
   c. Proximal to the navicular bone
4. Desmitis of the collateral ligaments (CLs) of the DIP joint
5. Navicular bursitis
6. Synovitis/capsulitis/OA of the DIP joint
7. Primary hoof imbalances (improper trimming or shoeing)
8. Hoof capsule and/or heel distortions

navicular apparatus.[46,49,96] However, documenting many of these abnormalities in horses that do not have radiographic abnormalities requires advanced imaging such as computed tomography (CT) or magnetic resonance imaging (MRI).[101] In addition, hoof imbalances, hoof capsule distortion, contracted or sheared heels, etc. may be the sole cause of lameness without abnormalities within the deeper structures of the foot.[4,25,115]

The bottom line is that not all horses with lameness conditions associated with the palmar aspect of the foot should be labeled as having navicular disease or syndrome. Some clinicians feel that navicular disease/syndrome should be reserved for horses with chronic bilateral forelimb lameness that fit a very specific set of diagnostic criteria.[115] Most horses with true navicular disease have a bilateral forelimb lameness that switches to the opposite limb after a low PD block, pain on hoof testers across the central or cranial aspect of the frog, and some evidence of radiographic abnormalities within the navicular bone. However, hoof tester sensitivity may be variable, and based on recent MRI studies, the lack of radiographic abnormalities does not rule out navicular bone pathology.[2,35,100]

Etiology

The two proposed causes of navicular disease are vascular compromise and biomechanical abnormalities leading to tissue degeneration.[6,22,23,43,71,82,88,96,97,115,119,136] With the vascular theory, thrombosis of the navicular arteries within the navicular bone, partial or complete occlusion of the digital arteries at the level of the pastern and fetlock, and a reduction in the distal arterial blood supply due to atherosclerosis was thought to result in ischemia of the navicular bone.[22,23,97] The vascular theory has not withstood scientific scrutiny because of failure either to reproduce the disease by altering blood flow or to identify histological tissue changes compatible with the theory.[96,97,119] In addition, increased bone remodeling/modeling and vascularization have been demonstrated in horses with navicular disease.[22,82,136] The increased vascularization was shown to be a combination of active arterial hyperemia and passive venous congestion. Obstruction of the venous outflow was thought to result in venous congestion, increased bone marrow pressure, and pain.[86,88,117]

There is increasing evidence that abnormal nonphysiological biomechanical forces leading to tissue degeneration is the most likely cause of navicular disease.[6,16,45,70,88,97] Forces exerted on, or experienced by, the navicular bone and the podotrochlear apparatus are central to these biomechanical studies. Abnormal forces on the navicular bone could arise from either excessive physiological loads applied to a foot with normal conformation or normal loads applied to a foot with abnormal conformation.[61,119] Poor hoof conformation and balance, particularly the long-toe, low-heel hoof conformation accompanied by the broken-back hoof-pastern axis, have historically been considered major risk factors for the development of navicular disease. This concept has withstood the test of time, supporting the theory that excessive and repetitive forces applied to the distal third of the navicular bone by the DDFT are a major contributor to the disease. Further supporting this is the finding that the force exerted on the navicular bone is negatively correlated to both the angle between the third phalanx (P3) and the ground and the ratio between heel and toe height.[32,47,51,61] Extreme cases of collapsed or underrun heels can result in a negative palmar/plantar angle where the palmar/plantar margin is lower than the dorsal margin of the distal phalanx (Figure 4.1). This type of conformation greatly increases the contact stress on the navicular bone by the DDFT. In contrast, heel elevation is used to treat horses with navicular disease and is thought to decrease the tension on the DDFT, which reduces the forces applied to the navicular bone.[51,96,132]

The location of the navicular bone between P3, the second phalanx (P2), and the DDFT suggests that it functions primarily as a buffer to spread forces among the anatomical structures.[96] It ensures that the DDFT maintains a constant angle of insertion, and the distal aspect of the bone must withstand great forces during the propulsive phase of the stride.[13,17,28] A study in isolated limbs using pressure-sensitive film documented that the contact load on the navicular bone and associated joints was highest during dorsiflexion (extension) of the limb, which corresponds to the end of the stance phase or the beginning of the propulsion phase.[17] However, in another study, both force and stress on the navicular bone in horses with navicular disease were approximately double those of normal horses during the early stance phase of the stride.[133] This was associated with greater tension on the DDFT, which the authors attributed to contraction of the deep digital flexor muscle in the early stance phase. Contraction of the deep digital flexor muscle was thought to help avoid heel-first landing in an attempt to unload the heels, but this appears to simultaneously increase the forces on the navicular bone.[96,133]

It has been estimated that the peak forces on the navicular bone approximate 0.67× body weight during the walk and 0.77× body weight at a slow trot.[88,115] However, the forces applied to the navicular region are influenced not only by body weight but also by limb and

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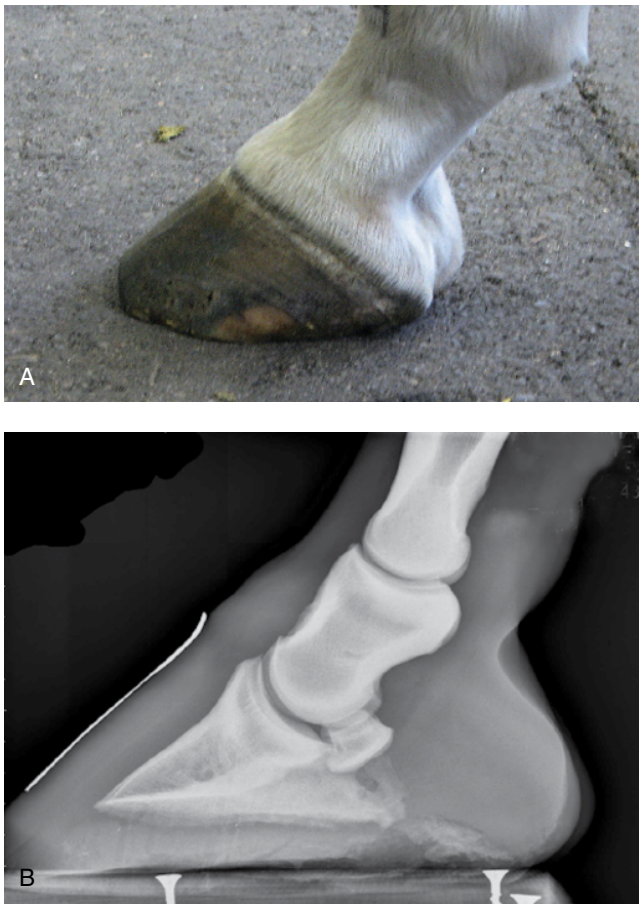


Figure 4.1. (A) Front foot and (B) lateral radiograph of a horse with a reverse or negative angle of P3 that is thought to predispose to problems in the palmar aspect of the foot.



Figure 4.2. Broken-back hoof-pastern axis that is often seen in horses with navicular disease/syndrome.

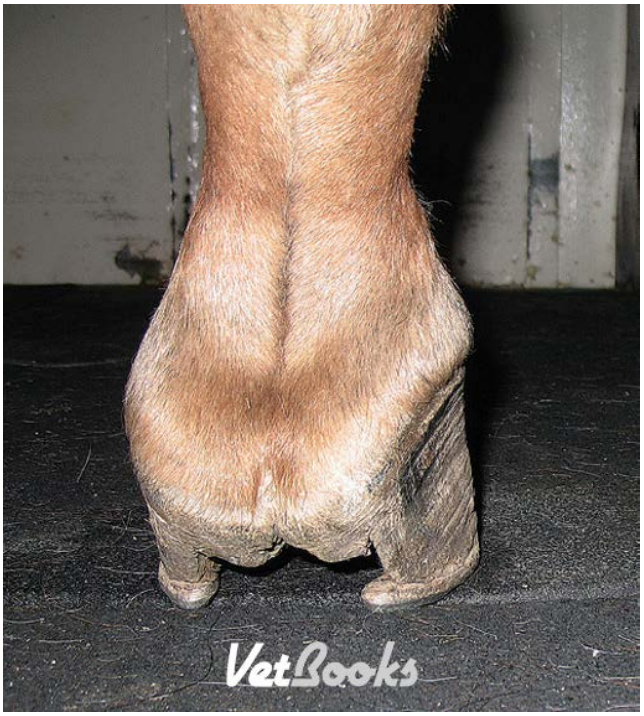


Figure 4.3. Palmar view of a foot with medial to lateral hoof imbalance. The medial hoof wall is longer and more upright than the lateral aspect, and the coronary band is displaced proximally on the medial side.

foot conformation and the type of work performed by the horse. Factors such as excessive body weight, small feet, broken pastern angles, long toes, low heels, hoof imbalances, and work on hard surfaces are likely to increase the forces per unit area of the navicular bone and podotrochlear apparatus (Figures 4.1–4.3).[4,48] Minor conformational abnormalities and other predisposing factors do not cause excessive loading of the navicular bone in most horses, resulting in normal modeling/remodeling of the navicular bone over time without the development of lameness.

In horses with severe or multiple predisposing factors, nonphysiological forces exerted on the navicular bone region begin the pathologic process that can affect the palmar/plantar fibrocartilage, dorsal articular cartilage, underlying bone, navicular bursa, podotrochlear apparatus, and DDFT.[96] Many of the pathologic changes within the navicular bone resemble those seen grossly and microscopically with osteoarthritis (OA) at other locations such as the distal tarsal and proximal interphalangeal (PIP) joints.[96,115,119] These microscopic changes include focal degeneration, cartilage erosion, subchondral bone sclerosis associated with thickening of the trabeculae, focal areas of lysis, edema, congestion, and fibrosis in the marrow spaces (Figure 4.4).[35,70,86,88,96,136]

Recent work has also indicated that the navicular bones in lame horses that lack radiographic evidence of disease can have a variety of histologic abnormalities. In this study, the presence of increased signal intensity in the spongiosa of the navicular bone with MRI was associated with the histologic findings of fat atrophy, proliferation of capillaries within the altered marrow fat, perivascular or interstitial edema, enlarged intertrabecular bone spaces, fibroplasia, and thinned trabeculae showing loss of bone.[49,69]

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Figure 4.4. Cross section of the navicular bone at necropsy demonstrating cyst-like lesions within the body of the navicular bone.



Figure 4.5. Postmortem view of the navicular bone illustrating degeneration of the flexor cortex. This horse also had surface fibrillation of the DDFT.

Additional abnormalities that have been found on the flexor surface of the navicular bone in lame horses compared to age-matched controls included thinning, crevicing or loss of the fibrocartilage layer, chondrone formation, and subchondral bone necrosis (Figure 4.5).[9] In this study there was no relationship between age and the severity of the histological abnormalities in the navicular bone, but the lesions were more common in lame horses than controls.[9] Studies from both lame and non-lame horses have identified early degenerative changes on the sagittal ridge, and it is thought that degenerative changes within the navicular bone initiate at the sagittal ridge. Such early changes included thinning of fibrocartilage and subchondral bone along the flexor surface and fibrillation and disruption of the DDFT.[35,46,69]

Scanning electron microscopy studies of the distal sesamoid bone and DDFT have identified fraying of collagen fibers in the DDFT and fissuring of the fibrocartilage on the flexor surface of the navicular bone.[33,134] Damage to the fibrocartilage together with DDFT fibrillation may predispose to adhesion formation between the tendon and the bone. Other studies have identified superficial or deep sagittal or parasagittal crevices or splits of the DDFT, abrasions or fibrillation on the dorsal surface, focal fibrocartilaginous metaplasia, and focal fibroplasia (Figure 4.5).[10,96] These abnormalities were not related to age, but lesions on the dorsal aspect of the DDFT were significantly more common in lame horses than control horses.[11] In addition, true core lesions appeared to be uncommon, and there was little evidence of acute inflammatory changes within the DDFT.[10] Superficial lesions on the dorsal surface of the DDFT may occur without detectable lameness, but deep sagittal splits are usually related to lameness.[96] Abnormalities within the podotrochlear apparatus (CSL, DSIL) have also been described in horses with navicular disease, but their clinical significance is difficult to assess based on the inability to definitively distinguish between individual structures and their contribution to lameness when multiple lesions are identified.[9,84,96] Fibrocartilaginous metaplasia, collagen degeneration, and fibroplasia were the most common microscopic findings.[9,10]

The pain and lameness in horses with navicular disease/syndrome presumably comes from within the bone and/or the surrounding supporting soft tissue structures. In most cases, it probably originates from more than one site because combination injuries within the foot appear to be most likely. With significant bone degeneration, the origin of pain is probably similar to that of horses with OA. Venous drainage of marrow spaces below lesions of degenerative joint disease is thought to be sluggish, and pain is associated with dilated vessels in the subchondral spongiosa.[88] Horses with navicular syndrome have been shown to have impaired venous drainage[67,86] and have bone marrow pressure exceeding 50 mm Hg, which is significantly higher than that of control horses.[86,117] Therefore, increased intraosseous pressure associated with venous distension and hypertension may be the cause of bone pain in affected horses.

Pain from injuries to the supporting ligaments and the DDFT most likely contribute to the lameness in most cases. Significant DDFT lesions (sagittal splits and core lesions) may be very painful, similar to tendinitis at other locations. Enthesophyte formation on the proximal or distal borders of the navicular bone may represent previous tearing of the CSL attachments, but it is often difficult to determine if these abnormalities actively contribute to the pain. In addition, sensory nerve endings have been identified in the CSL, DSIL, and DDFT at its attachment to the distal phalanx.[14–16] The location where the DSIL and the dorsal aspect of the DDFT insert onto the distal phalanx has been referred to as the "intersection" and is thought to have a rich sensory innervation, particularly of substance P and neuropeptide-producing fibers, and abundant arteriovenous complexes (Figure 4.5).[14,15,119] This anatomic region may act as an initiation site for many of the more extensive tissue changes that subsequently develop in many horses with navicular disease.[119]

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Clinical Signs

Horses with navicular disease/syndrome usually have a history of progressive, chronic, unilateral, or bilateral forelimb lameness, which may have an insidious (most common) or acute onset.[35,84,115] The history may include a gradual loss of performance, stiffness, shortening of the stride, loss of action, unwillingness to turn, and increased lameness when worked on hard surfaces.[35,46] Chronic bilateral forelimb lameness is considered the norm, and navicular syndrome has been described as heel pain that blocks to a PD nerve block on both forelimbs with or without radiographic abnormalities.[100,101] However, unilateral lameness can also occur, especially with lesions that involve the flexor surface of the navicular bone and/or the DDFT. Most horses with an acute onset of unilateral lameness that blocks to the foot are not considered to have navicular disease/syndrome, and lesions in the DDFT or other problems in the foot should be suspected.

Navicular disease/syndrome is considered to be a degenerative process due to wear and tear similar to OA; therefore, middle-aged to older horses are most commonly affected. Clinical signs usually become apparent in most horses between 7 and 10 years of age, although younger horses can be affected.[35,46,100,115] Horses with developmental/congenital abnormalities such as bipartite navicular bones can often become lame as early as 2–3 years of age (Figure 4.6). Geldings are more commonly affected than mares or stallions and Quarter horses, Warmbloods, and Thoroughbreds seem more predisposed to the disease than other breeds.[35,100] The classic signalment and history for many horses with navicular disease/syndrome is a middle-aged Quarter horse gelding with a history of chronic bilateral forelimb lameness.

Although navicular disease/syndrome usually affects both front feet, the lameness may initially appear unilateral.[35,46,115] Greater than 95% incidence of asymmetrical lameness has been reported.[135] Most horses are more lame in one forelimb, both at a straight trot and when circled on a hard surface, but often demonstrate lameness on the opposite forelimb when circled with that limb on the inside. Bilateral forelimb lameness observed when circled both directions was present in 76% of the horses with less than 6 months' duration of lameness and in 52% in horses with greater than 6 months' duration of lameness in a recent study.[100] The remainder of the horses had a unilateral lameness until a PD nerve block was performed, at which time the lameness switched to the opposite forelimb.[84,100,101] However, the true severity of a unilateral lameness at a straight trot may be misinterpreted because of the concurrent lameness in the opposite forelimb. In addition, the horse may tend to point one forelimb or alternate pointing each forelimb.[115,135] Asymmetry in the extensor muscles with atrophy of the muscles associated with the lame limb can often be observed in horses with chronic lameness.[115,116,135]

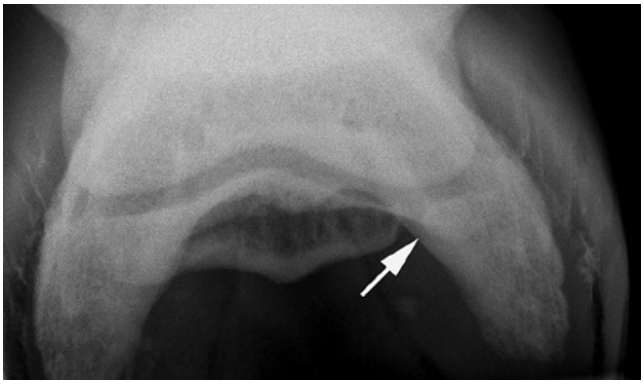


Figure 4.6. Bipartite navicular bone (arrow) as seen on a skyline radiograph in a young horse with lameness isolated to the foot.

In a recent study that compared specific sources of foot lameness verified on MRI (navicular bone lesions alone, lesions of the podotrochlear apparatus, lesions of the DDFT alone, and lesions involving navicular bone and soft tissue), there were no significant associations among groups with respect to onset of lameness, foot conformation, posture, presence of distal interphalangeal (DIP) joint effusion, hoof tester examination, flexion testing, or blocking pattern.[84] Most (73.3%) horses were lamer when lunged on a firm surface than a soft surface, and 32.7% of horses were lamer on a circle than in a straight line. Horses with podotrochlear apparatus injuries were more likely to be unilaterally lame, and horses with injury to the DDFT were more likely to exhibit lameness at the walk when turning tight turns.[84]

Various abnormalities of the hoof can be present in horses with navicular disease/syndrome. It can often be difficult to determine whether the hoof abnormalities are contributing to the disease or have developed secondary to the lameness and disuse of the foot. For example, both low, collapsed heels typical of Thoroughbreds and narrow, upright feet typical of Quarter horses can occur in horses with navicular disease/syndrome. Secondary hoof abnormalities are most likely to develop in horses with a chronic duration of lameness. Common hoof problems seen in horses with navicular disease/syndrome include low, underrun heels, contracted or collapsed heels, medial to lateral imbalances, and long toes (Figures 4.1–4.3).[4,35,47,121,135] Dorsal to palmar imbalances such as the broken-back hoof-pastern axis are also commonly seen and are considered to be a predisposing factor for development of a multitude of problems in the foot. In one study, a broken-back hoof-pastern axis occurred in 71% of the horses with navicular disease/syndrome.[135] One forefoot is often smaller, narrower, and more upright (longer heels) than the other, which is presumably from disuse atrophy. In most cases the limb with the more upright hoof conformation is the limb with the greatest lameness.[4,25,35,47,48,135] Angular limb deformities, either valgus or varus, associated with the carpus and fetlock region have also been observed in a small percentage of cases.

At exercise, most horses with navicular disease/syndrome exhibit a mild to moderate lameness (2–3 of 5) that is worse on hard surfaces.[35,84] Only occasionally is severe lameness encountered. Some clinicians feel that the severity of lameness usually increases with the duration of the lameness.[115] However, in a recent study, the mean lameness grade of horses with navicular syndrome was grade 3 for horses with less than 6 months' duration of lameness and grade 2 for horses with more than 6 months' duration of lameness.[100] The severity of

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lameness may be more related to the specific cause(s) of pain rather than the duration of the lameness.[84]

While walking or trotting, many horses tend to land toe first and may occasionally stumble.[22,96,119] At a trot, horses with bilateral lameness tend to have a stiff shuffling gait and often carry their heads and necks rigidly. This stilted gait is usually worsened when circled and has been described as the horse "trotting on eggshells." Owners often misinterpret this characteristic gait as an unwillingness to advance the limbs associated with shoulder pain. When circled in either direction on a hard surface, the lameness is usually exaggerated in the limb that is on the inside of the circle. The lameness and stilted gait is often exaggerated as the size of the circle is reduced. The horse may hold its head and neck to the outside of the circle in an effort to reduce the amount of weight carried on the inside limb. One study reported that 20% of horses that presented with no overt signs of lameness by owners displayed a bilaterally symmetrical short-stepping gait when evaluated by a veterinarian. Asymmetric forelimb lameness was easily observed when these horses were circled. This finding highlights the importance of recognizing this type of gait in horses with bilateral forelimb lameness and the necessity to evaluate these horses on different surfaces and at the lunge.[84]

Hoof tester examination is considered essential for the clinical diagnosis of navicular disease/syndrome by many clinicians. However, the reliability of this test is somewhat controversial because a negative response to hoof testers over the frog region is not uncommon in horses with navicular syndrome/disease.[35,84,100,122] In one study, only approximately 50% of the horses with lameness localized to the navicular region responded positively to hoof tester pressure over the central third of the frog.[124] In contrast, a nonfatiguable painful withdrawal to intermittent hoof tester pressure over the central and occasionally the cranial third of the frog is considered a fairly consistent feature of navicular disease/syndrome by others.[115] In addition, it is important to apply direct compressive pressure to the navicular region rather than simply applying lateral (shearing pressure) across the frog when using the hoof testers. Horses with very thick soles and hard frogs usually do not respond to hoof tester pressure. Hoof tester pain may also be present over the toe secondary to bruising from landing toe first but is usually of minor clinical significance.

Distension of the DIP joint is a nonspecific finding that may be found in normal horses as well as horses with navicular disease.[35] DIP joint distension was identified in only 4.2% of horses with various injuries to the foot that were confirmed on MRI[84] and is often detected more frequently by MRI than by clinical examination.[44,76] However, the presence of DIP effusion may be an important finding when developing a treatment plan for an individual horse. In addition, asymmetrical DIP joint effusion is usually clinically relevant and often suggests a secondary problem within the joint. Many horses with navicular disease/syndrome may react positively to a phalangeal flexion test, which often exacerbates the lameness.[115,135] However, a positive phalangeal flexion test is not specific for the navicular region and is usually not that beneficial in localizing the lameness to the foot. A positive phalangeal flexion may suggest a primary or secondary problem within the DIP joint in some horses with foot pain.

Use of a wedge block, although nonspecific, can be useful to evaluate horses with foot pain. The wedge block allows perhaps more specific manipulation of the distal limb than can be performed with flexion tests (Figure 2.111). Elevating the toe, similar to a naturally occurring negative palmar solar angle, is thought to increase the strain on the DDFT and podotrochlear apparatus and increase the pressure on the navicular bone and navicular bursa. Little work has been done to support the use of the wedge block, but in one study, toe elevation tests resulted in a sensitivity of only 55% and a specificity of only 42% for the presence of navicular pain.[122] In contrast, raising the heels is thought to decrease the strain on the DDFT and podotrochlear apparatus and decrease the pressure on the navicular bone and navicular bursa. However, it will result in increased heel pressure. One study found that heel elevation had a sensitivity of 76% and a specificity of 26% for the presence of navicular pain.[122] The observation of worsening lameness following heel elevation has also been observed in many horses with MRI-confirmed injury to the DDFT.[110]

Wedge block manipulation of the distal limb typically consists of placing the foot on the block squarely in the toe-up position. The opposite limb is usually held up in a relaxed neutral position and held for 60 seconds. The horse is then trotted off and any change in the lameness is observed and documented. The same test is then performed in the heel-up position. Because we know that many horses with "navicular disease" commonly also have injuries to the supporting soft tissues, the test can also be performed in a medial and lateral up position. With the foot placed in the lateral wedge position, the lateral aspect of the foot is placed in compression while the medial aspect of the foot is placed under tension. The authors have found horses with confirmed DIP joint disease and injuries to the collateral ligaments (CLs) of the DIP joint to show a positive response to this test.

Diagnosis

Local Anesthesia

The diagnosis of navicular disease/syndrome begins with localizing the site of lameness to the foot or more specifically to the palmar aspect of the foot using diagnostic anesthesia. Historically, a PD nerve block was thought to only desensitize the palmar aspect of the foot, but it is now known that it is relatively nonspecific and alleviates pain in the navicular bone, podotrochlear apparatus, navicular bursa, distal aspect of the DDFT, distal phalanx, middle phalanx, DIP joint, dorsal aspect of the hoof, and possibly the digital tendon sheath and PIP joint.[96,102] Therefore, a multitude of clinical problems in the foot can be desensitized with a PD block; this has been confirmed in numerous studies.[38,39,41,84,100,101]

Using a small volume of anesthetic (1.0–1.5 mL) and performing the PD block as low as possible in the heel region (axial and distal to the proximal limits of the collateral cartilages) is recommended to improve the specificity of the block to the structures within the palmar aspect of the foot.[46,96,108,109,111] Several studies have

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documented that the diffusion and distribution patterns of anesthetics are affected by volume, injection site, and time.[79,80] Some advocate that if lameness is not abolished within 10 minutes following a nerve block, the block should be deemed negative and to continue blocking.[111] It is important to be mindful of technique, volume of anesthetic used, and timing between nerve blocks. However, using good technique does not guarantee that multiple structures have not been desensitized and that the block has extended beyond what is to be expected.

The majority of horses affected with navicular disease/syndrome (80%–100%) improve substantially following a PD block and the lameness in the opposite forelimb either worsens or becomes apparent if a unilateral lameness was initially found.[99–101,115] Kinematic gait analysis has found that the mean maximal extension of the fetlock during the stance phase of the stride and the maximum flexion of the carpal joint during swing phase of the stride were significantly increased after PD blocks in horses with navicular disease.[68]

Intrasynovial anesthesia of the DIP joint and the navicular bursa can also be performed to potentially further localize the site of pain. Historically, blocking the DIP joint was thought to help distinguish between problems associated with the joint and the navicular region. However, several studies have demonstrated a lack of specificity of intrasynovial anesthesia of the DIP joint due to diffusion of anesthetic and the location of sensory nerves in close approximation to the synovial outpouchings of the DIP joint.[15,16,34,55,87,107,108] Anesthesia of the DIP joint is known to improve the lameness in a large percentage of horses diagnosed with navicular syndrome.[34,35,46,99,111,115] However, the amount of improvement in lameness following DIP joint anesthesia is often less than that following a PD block in most horses with navicular disease/syndrome. Recommended methods to improve the specificity of DIP joint anesthesia are to use no more than 5–6 mL of anesthetic and to assess the response to the block within 5–10 minutes.[96,99,107,111]

Anesthesia of the navicular bursa is probably the most specific nerve block that can be used to help localize the site of pain in horses with navicular disease/syndrome. However, it is not routinely used as the initial diagnostic block because of the need for radiographic or ultrasonographic confirmation of needle placement. A positive response to intrasynovial anesthesia into the navicular bursa may indicate problems of the navicular bursa, navicular bone, and/or navicular supporting ligaments, sole, and/or toe, or distal aspect of the DDFT.[107–109,111] Even though diffusion of local anesthetic into the navicular bursa occurs following DIP joint injection, the converse does not occur, and analgesia of the navicular bursa does not usually result in analgesia of the DIP joint.[108,111] Pain from the DIP joint can likely be excluded as a cause of lameness if analgesia of the navicular bursa improves the lameness within 10 minutes.[108,111] In addition, a positive response to intra-articular analgesia of the DIP joint together with a negative response to navicular bursa analgesia incriminates pain within the DIP joint as the cause of lameness.[111]

See Chapter 3 for further information about local anesthesia.

Radiography

Despite its limitations, radiography remains the initial diagnostic tool to assess the navicular bone in most horses with navicular disease/syndrome. However, degenerative changes within the bone can be missed because a 40% change in bone density is required before it can be identified with radiographs.[96] Therefore, the lack of abnormalities in the navicular bone does not eliminate the bone as the site of the pain and does not necessarily indicate a soft tissue problem in the foot. Several studies evaluating horses showing signs of navicular syndrome but with normal or mild radiographic abnormalities of the navicular bone have documented that the majority of these horses' navicular bones were abnormal on MRI or histological examination.[49,69,100,101] The navicular bone was considered the primary abnormality in only 33% of horses with a recent onset of lameness and in only 16% of horses with a more chronic duration of lameness.[100,101]

A complete radiographic evaluation of the navicular bone requires a minimum of lateromedial, 60° dorsoproximal to palmarodistal oblique, and palmaroproximal to palmarodistal oblique (skyline) high-quality views.[35,37,50,115] Additional radiographs such as the 60° dorsoproximal to palmarodistal oblique view of the distal phalanx and the weight-bearing dorsopalmar view are often included to completely evaluate all bony structures in the foot. Adding a shallow angle (35°) palmaroproximal to palmarodistal oblique projection has been recommended to improve the accuracy of identifying flexor cortex lysis.[64] This is supported by the fact that the majority of flexor cortex lesions occur in the mid to distal aspect of the flexor surface.[9,49] Careful attention to foot preparation (packing the frog), limb position, and centering and directing the X-ray beam according to the hoof capsule conformation is important to avoid artifacts and inaccurate image interpretation.[37,50] For example, slight obliquity to a lateral medial projection will result in a poorly defined sagittal ridge, misinterpretation of the true flexor cortex thickness and uniformity of opacity of the flexor cortex, poor demarcation between the endosteal surface of the flexor cortex navicular bone and the trabecular bone, poor representation of the proximal to distal shape of the flexor aspect, and poor definition of the articular margins of the navicular bone.[37]

There are a number of radiographic abnormalities to the navicular bone that have been described; some are considered incidental and may or may not be pathologic, and some correlate strongly with lameness. Those seen more commonly in lame horses include the following:

1. Enthesophytes at the proximomedial and proximolateral aspect of the bone. Small enthesophytes can be seen in sound horses, but large enthesophytes are usually associated with lameness.
2. Proximal or distal extension of the flexor border of the bone. This change in shape is indicative of stress at the insertion of the CSLs and origin of the DSIL.
3. Fragments at the distal border of the bone. These fragments are usually located at the distal medial and lateral angles of the bone and can be associated with lameness. Their presence may be due to ectopic

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mineralization in the DSIL, fracture directly off the distal border of the navicular or fracture of enthesophyte formation at the origin of the DSIL.
4. Numerous (>7) large and variably shaped distal border radiolucent zones.
5. Discrete radiolucent areas in the spongiosa with or without detectable communication with the flexor cortex.
6. New bone at the sagittal ridge.
7. Increased thickness of the flexor cortex.
8. Sclerosis of the spongiosa.
9. A bipartite bone.[37]

Additional abnormalities that are considered important include flexor cortex defects or erosions, loss of corticomedullary distinction, and mineralization of the supporting ligaments of the navicular bone or the DDFT.[37,65,122,130]

The radiographic abnormalities that appear to be the most reliable indicators of navicular disease/syndrome are cyst-like lesions within the medullary cavity (Figures 4.7 and 4.8), flexor cortex lesions, and sclerosis of the spongiosa with loss of demarcation of the flexor cortex and medulla (Figures 4.9 and 4.10).[35,37,130] Flexor cortex defects were seen in less than 1% of normal horses; they represent lysis of subchondral bone and are linked to fibrocartilage degeneration and damage to the DDFT (Figure 4.11).[65,130] Sclerosis was present in up to 80% of horses with navicular syndrome but in less than 16% of normal horses.[65]

Less reliable radiographic indicators include enthesophytes at the proximal or distal border of the bone, elongation of the flexor border of the bone, distal border fragments, and enlarged synovial invaginations. All of these abnormalities of the navicular bone can be present in non-lame horses and by themselves may not constitute radiographic evidence of navicular disease.[130] However, distal border fragments appear to be recognized more frequently with digital radiography and are thought to be associated with navicular disease (Figure 4.12).[7,8,37] These fragments may represent a fracture, enthesophyte, or dystrophic mineralization within the DSIL.[7] See Chapter 4 for further information on radiography of the navicular bone.

Ultrasonography

Ultrasound is an economical and readily available diagnostic technique that can be used to help diagnose potential soft tissue injuries within the foot in horses with navicular disease. However, the keratinized hoof wall, frog, and sole all limit contact with the ultrasound probe and prevent good images from being obtained.[19,96] With experience, ultrasound can be used to assess the flexor surface of the navicular bone, navicular bursa, podotrochlear apparatus, and distal part of the DDFT; evaluation of the dorsal pouch of the DIP joint and CL of the DIP joint phalanx should also be performed.[19,56] The most common abnormalities identified using ultrasound



Figure 4.8. Multiple smaller cystic lesions along the distal border of the navicular bone as demonstrated on an oblique radiograph.

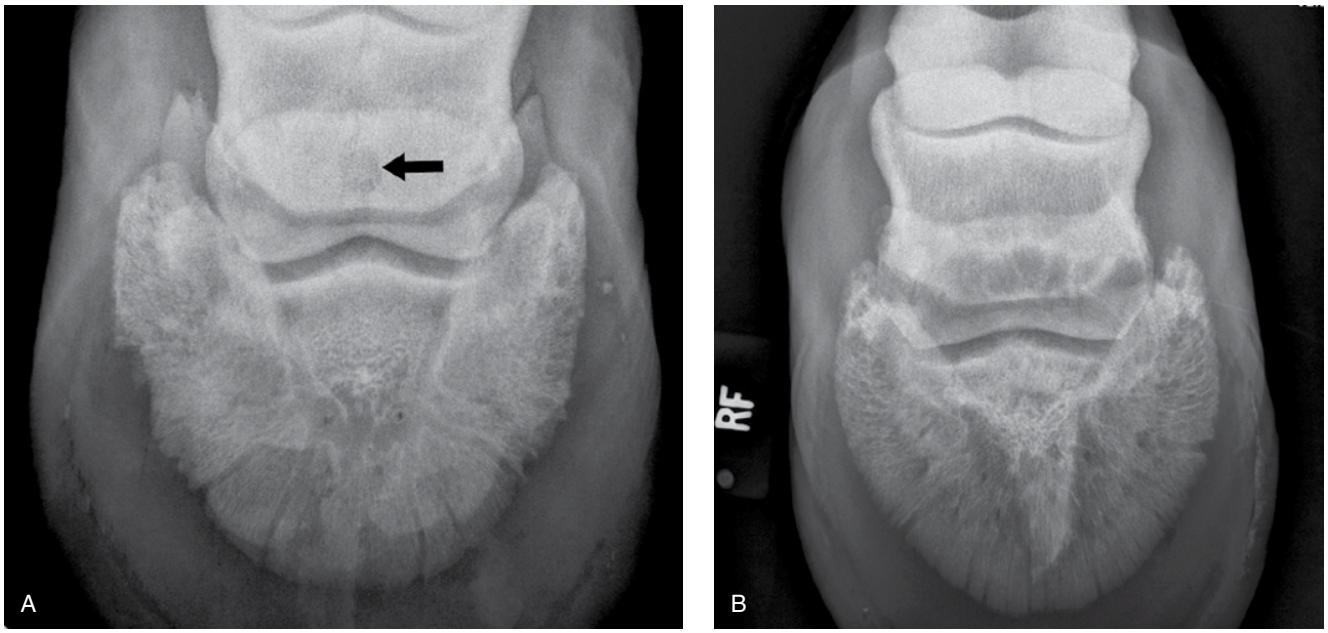


Figure 4.7. Single large cystic lesion (A; arrow) and multiple cystic lesions (B) within the navicular bone demonstrated on oblique radiographs of two different horses with signs of navicular syndrome.

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in horses with lameness isolated to the foot but with no radiographic abnormalities included changes to the DDFT (73%), effusion and synovial proliferation to the DIP joint (69%), and navicular bursitis (56%).[94] Lesions of the DDFT were most commonly found in the lateral lobe of the tendon and located just above the navicular bone in the majority of cases. These findings were similar to results of previous imaging and histopathologic studies.[10,11,20,39,42,44] Similar to radiography, the accuracy of ultrasound to document pathology within the foot has been criticized based on results of more advanced imaging such as MRI. Therefore, negative findings with ultrasound do not rule out the presence of abnormalities in the navicular region and currently is not used routinely clinically.



Figure 4.9. Multiple abnormalities within the navicular bone as seen on an oblique radiograph. Abnormalities present include remodeling along the proximal border, multiple cystic lesions along the distal border, and enthesophytes on the wings of the navicular bone.

Scintigraphy

Scintigraphy (nuclear imaging) was initially thought to be able to identify early pathologic changes within the navicular bone related to its ability to identify early alterations in bone metabolism rather than relying on anatomic changes. However, increased radiopharmaceutical uptake (IRU) has been documented to occur in only 36% of the limbs of horses with navicular disease/syndrome and in a very small percentage of limbs with soft tissue injuries within the foot.[38,40,67] Scintigraphic assessment of the foot can be helpful to identify the potential source of pain causing lameness, but false-positive results can occur, especially in horses with low-heel conformation.[38,40] Additionally, a negative scintigram of the foot does not preclude significant injuries, and scintigraphy does not provide a definitive diagnosis.[38,40,96] Results of a recent study that evaluated the diagnostic accuracy of scintigraphy in lame and poorly performing sport



Figure 4.11. Lateral radiograph of the foot in the horse in Figure 4.5. A lytic defect is present in the flexor surface of the navicular bone (arrow).

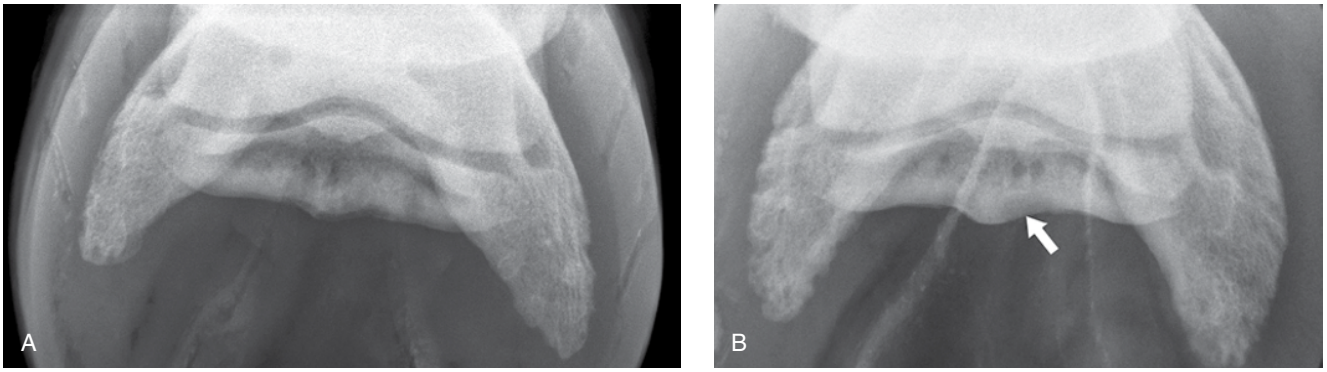


Figure 4.10. Skyline radiographs of the same horse as in Figure 4.9 (A) demonstrating sclerosis of the medullary cavity of the navicular bone and erosions along the flexor surface. (B) A single large erosive lesion on the flexor surface of the navicular bone (arrow) in a horse with severe unilateral lameness.

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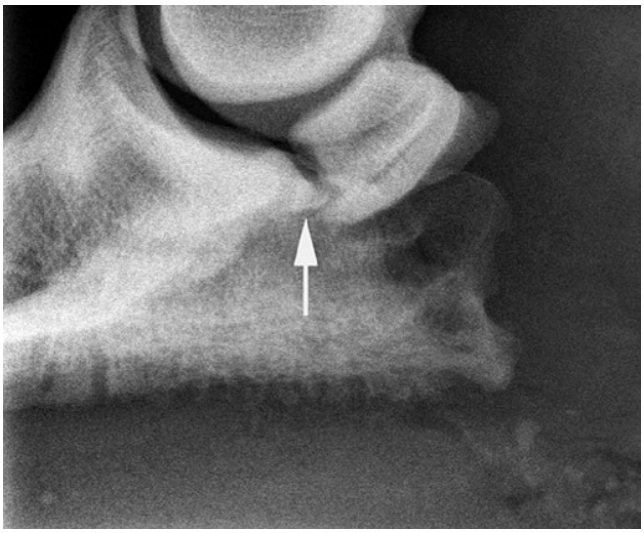


Figure 4.12. An avulsion fracture from the distal border of the navicular bone (arrow) can be seen on this lateral radiograph.

horses found an overall poor agreement between IRU and a positive diagnosis for lameness and poor performance.[93] For these reasons, and the economics of performing multiple advanced imaging techniques on the same horse, MRI is usually performed instead of scintigraphy in most horses with foot-related lameness.

Computed Tomography (CT)

CT is thought to be the best modality to detect and assess pathology within the cortex and trabeculae of the navicular bone.[91,96,118] Many osseous changes seen on CT are not radiographically evident, such as shape changes within the bone, distal border fragments, and intramedullary changes.[96,118] Soft tissue abnormalities within the foot may be detected with CT, but in general, CT is best at detecting abnormalities within bone and MRI is preferred to assess soft tissue lesions.[131] However, contrast-enhanced CT has been shown to improve the imaging of soft tissue structures within the foot and may be an alternative to using MRI.[89,91,125] Studies have suggested that the frequency of soft tissue injury identification with CT and MRI may be similar,[19,125] and using contrast-enhanced CT has been shown to compare favorably to MRI for identification of DDFT lesions within the foot.[90,92] Additional advantages to using CT include decreased anesthesia time since CT scans are very fast. The use of CT could facilitate an earlier and more accurate diagnosis of navicular bone pathology in horses but may not be able to detect the many concurrent soft tissue abnormalities that are often present in horses with foot pain.[96] However, new advances in CT technology such as the equine positron emission tomography (PET) CT may further enhance the ability of CT to document soft tissue pathology within the foot and elsewhere.[89,91] See Chapter 3 for further information on musculoskeletal CT imaging.

Magnetic Resonance Imaging (MRI)

MRI has become increasingly available in recent years and is currently the preferred diagnostic technique to assess most horses with navicular disease/syndrome.[20,39,41,43,44,78,100,101] The different sequences permit accurate evaluation of soft tissues, cartilage, and bone within the digit in near anatomic detail. The MRI technique can be performed in both the standing (low-field) and recumbent (high-field) patient, but better-quality images are thought to be obtained with high-field units. The ability to image and appreciate pathologic changes to articular cartilage and subtle soft tissue and bone abnormalities is thought to be superior with high-field vs. low-field MRI.[3]

Numerous pathologic entities have been identified with MRI in horses with navicular disease/syndrome, and many horses appear to have multiple abnormalities present within the same foot.[38,39,44,78,101] The most common abnormalities found on MRI involve the navicular bone, CSL, DSIL, DDFT, navicular bursa, CL of the DIP joint, and the DIP joint.[38,39,42,44,78,100,101] Several studies have documented that the DDFT and podotrochlear apparatus can be abnormal in horses with foot pain (Figure 4.13).[39,43,44,78,100,101] Injury to the DDFT, with a reported prevalence of 30%–64% in horses with foot lameness, is thought to be the most common soft tissue injury causing lameness in the horse's foot.[106] However, the frequency of occurrence of other soft tissue lesions such as those of the CSLs and the CL of the DIP joint differs among studies.[39,43,44,100,101] This may represent differences in the population of horses of the different studies or possibly differences in the interpretation of the MRI. From a clinical viewpoint, one difficulty with interpreting the results of MRI is determining what the primary abnormality may be when multiple lesions are found. However, this may be less important if we now assume that multiple abnormalities within the foot most likely contribute to the overall pain in horses with navicular disease/syndrome. See Chapter 3 for further information on MRI.

Treatment

Multiple factors are involved when deciding on a treatment protocol for horses with navicular disease/syndrome. In most cases the treatment must be tailor-made for each individual horse based on the severity of lameness, intended use of the horse, wishes of the owner, results of diagnostics (or lack of diagnostics such as MRI), hoof conformation, previous treatments, and most likely diagnosis.

Realistically, horses with advanced radiographic abnormalities in the navicular bone will be problematic regardless of the treatments employed. Historically, horses with minimal radiographic abnormalities of the navicular bone were thought to respond to therapy better than those with radiographic abnormalities.[115] However, this generalization is debatable because horses with DDFT injuries with or without navicular bone abnormalities tend to do poorly regardless of treatment.[41,96,106] In addition, developing a treatment protocol for horses without radiographic abnormalities and without performing an MRI can be challenging because a definitive diagnosis is difficult to make in these horses. The ability to develop a more precise treatment protocol is considered one of the benefits of performing an MRI in horses with foot pain. Using MRI to create an

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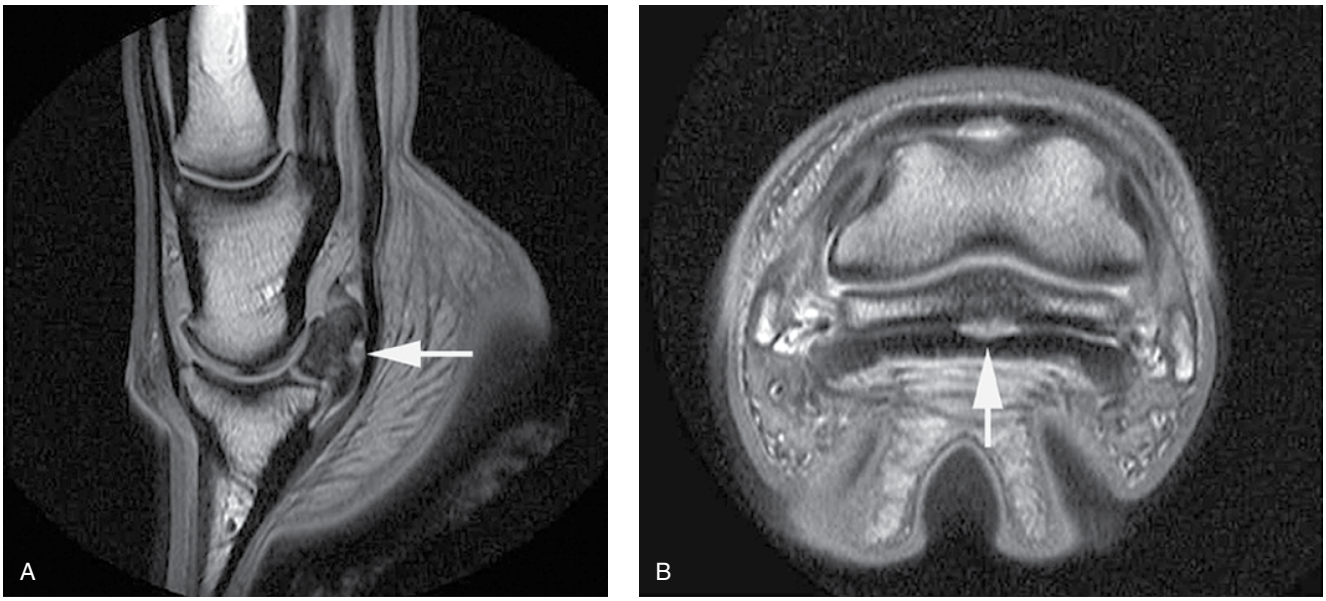


Figure 4.13. A lateral STIR MR image (A) and cross-sectional MR image (B) of the horse in Figure 4.11 demonstrating abnormal signal within the navicular bone (A; arrow) and surface damage to the DDFT (B; arrow).

individually tailored treatment regimen is thought to promote a more positive outcome in many equine cases.[3]

A variety of treatment options are available for horses with navicular disease/syndrome with the goal of managing the disease. One of the most important treatments is corrective trimming and shoeing.[96,115,120] Other nonsurgical treatments include rest and controlled exercise, nonsteroidal anti-inflammatory drugs (NSAIDs), bisphosphonates such as tiludronate and clodronate, intrasynovial medications, and medications aimed at preventing secondary OA of the DIP joint. Surgical treatments include palmar/plantar digital neurectomy, inferior check ligament desmotomy, and endoscopy of the navicular bursa. A brief overview of these treatments is discussed below. See Chapters 8 and 11 for more information.

Rest and Controlled Exercise

In the past, prolonged rest has not been recommended for most horses with navicular disease because the lameness often returns shortly after the horse resumes exercise.[73] However, significant soft tissue injuries within the foot such as DDFT or CSL lesions may warrant a more extended rest and rehabilitation period, similar to that recommended for horses with tendinitis elsewhere in the limb. However, many of the lesions within the DDFT appear to be degenerative and not inflammatory and do not generally respond to rest alone. Nevertheless, a short period of rest is usually recommended for most horses with navicular disease/syndrome to help reduce the soft tissue inflammation and allow the horse to acclimate to corrective trimming and shoeing.[115] This is substantiated by MRI findings that concurrent soft tissue and bone pathology most likely contribute to the pain in many horses with palmar foot pain.[39–41,43,44,78] Although the rest period may vary for individual cases, the author usually recommends no work until at least the first shoeing change in most horses. A decision is usually made regarding further exercise and continued treatment at the first shoeing change based on the response to treatment.

Corrective Trimming and Shoeing

Corrective trimming and shoeing are the basis for managing the majority of horses with navicular syndrome/disease.[4,25,96,120] Many horses respond favorably to shoeing alone without requiring further medical or surgical therapy. Improvement in clinical signs within 3 months of initiation of treatment has been reported with 86% of the horses remaining free of lameness for 1 year.[120,121] The goals of trimming and shoeing are to (1) restore normal foot balance; (2) correct foot problems such as shearing of the quarters and heels, underrun heels, and heel contraction; (3) reduce biomechanical forces on the navicular region; (4) ease break-over at the toe; (5) support the heels and prevent heel descent during loading; and (6) protect the injured areas of the foot.[25,115]

Multiple types of shoes have been used for horses with heel pain, and one single type of shoe cannot be used in every horse. For instance, a horse with the low-heel, long-toe conformation may benefit from an elevated heel shoe, whereas a horse with a narrow upright foot may actually benefit from heel removal and placement of a flat natural balance or egg-bar shoe. When the heels are long, they tend to grow forward, decreasing heel support and contributing to dorsopalmar hoof imbalance (Figure 4.14).[25] In general, foot preparation or trimming is more important than the type of shoe used in most horses with navicular disease/syndrome. A well-made shoe placed on an improperly trimmed foot serves little purpose. The heels should be trimmed to the widest aspect of the frog and the toes shortened as much as possible to shift weight-bearing to the back of the foot.[4,25] See Chapters 8 and 11 for more information on foot trimming and shoeing.

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Much information to aid trimming and shoeing of horses with navicular disease/syndrome can often be obtained from lateral and weight-bearing dorsopalmar radiographs (Figure 11.31). Medial to lateral and dorsopalmar hoof balance, toe length in relation to the distal phalanx, heel height, sole depth, and solar angle of the distal phalanx are all very important pieces of information that should be used during the shoeing process (Figure 11.2).

Several different types of steel or aluminum shoes are thought to be effective in treating horses with heel pain. These include the Tennessee navicular shoe, rolled or rockered toed shoes, egg-bar shoes, natural balance shoes, equine digital support system (EDSS), onion shoes, and full-bar support shoes (Figures 4.15 and 4.16).[4,25,73,96,120] Rolling, rockering, or squaring the toe of



Figure 4.14. Lateral view of a foot with excessive heel growth resulting in the weight-bearing surface of the heels being shifted further forward within the foot (arrow).



Figure 4.15. Using egg-bar shoes is a common shoeing technique to treat horses with navicular disease/syndrome.



Figure 4.16. Heel elevation may be helpful in treating select horses with navicular disease/syndrome (A) and not in others (B). The horse in (A) was shod with wedge pads and dental impression material and improved after shoeing. The horse in (B) was shod with elevated heel shoes despite having a very upright hoof conformation on the LF, which likely contributed to the dorsal hoof wall concavity (arrow) and continued lameness.

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any of these shoes enhances break-over, thus reducing the stress on the DDFT. The goals of any of these shoes should be to ease break-over, support the heels, and reduce the forces acting on the navicular bone by the DDFT.

Heel elevation has been shown to decrease tension in the DDFT, reduce pressure applied to the navicular bone, reduce the load to the forelimbs, and reduce the stresses on the hoof capsule (Figure 4.16).[96,98,102,132] The heels are supported for a greater proportion of the stride, and the wedge tends to increase the weight transferred through the heels. A recent study that quantified the pressure distribution across the palmar surface of the navicular bone documented that the maximum pressures exerted by the DDFT corresponded to the areas of known navicular pathology.[128] However, raising the heels by 5° and 10° only redistributed the pressures on the navicular bone, suggesting that raising the heels may not be that beneficial in horses with navicular syndrome.[128] Some clinicians feel that elevated heel shoes offer mixed results and should only be used temporarily especially in horses with DDF tendinopathy.[106] However, heel elevation alone or when combined with phenylbutazone has been shown quantitatively (using a force plate) to improve lameness in clinical cases of horses with navicular syndrome.[102] The disadvantages of raising the heels with the use of pads in horses with underrun heels may cause further collapse of the poorly supported heels, and heel growth appears to be reduced with wedge pads.[25]

Egg-bar shoes and natural balance shoes have not been shown to reduce the forces on the navicular bone as effectively as heel elevation.[96] However, egg-bar shoes increase the length of the toe–heel axis causing the center of pressure to shift caudally.[98] Egg-bar shoes are usually recommended in cases where the hoof capsule is unstable due to shearing or in horses with severely underrun or collapsed heels. The egg-bar shoe increases the surface area of contact and the stability of the hoof. The shoe should be applied so it is clearly visible at the quarters and extends far enough palmarly to cover the heels.[115,120,121] Natural balance shoes, by their design, reduce break-over and if combined with heel elevation also reduce the forces acting on the navicular bone. See Chapters 8 and 11 for more information on corrective trimming and shoeing.

A minimum 2- to 3-week adaptation period is often necessary to achieve pain relief with any type of shoeing.[96,102] In most cases improvement in clinical signs after corrective trimming and shoeing should be evident within 6 weeks but may depend on the severity of the initial lameness and the specific structures affected. Severe hoof imbalances may require several shoeing intervals to achieve a normal hoof conformation, and, in some cases, the underrun heels may persist indefinitely. Additionally, not all horses respond in the same manner to a particular shoeing type, and we as veterinarians should remain flexible in using a different type of shoe if a clinical response is not obtained.

Nonsteroidal Anti-inflammatory Drugs

The use of NSAIDs is a common adjunctive treatment for horses with navicular disease/syndrome. Flunixin meglumine, phenylbutazone, and firocoxib are used most commonly, and all should be effective in reducing inflammation and lameness. Horses with navicular disease that were treated with 1.1 mg/kg of flunixin meglumine or 4.4 mg/kg phenylbutazone once daily for 4 days showed significant reduction in lameness scores for 24 hours after the last dose compared with baseline and horses treated with saline.[52] The clinical responses between flunixin meglumine and phenylbutazone were similar and suggest that once-daily dosing of NSAIDs should be effective to control pain in the majority of these horses.

NSAIDs are often used for pain relief if the horse is severely lame or if the horse needs to continue working through the initial treatment period. Phenylbutazone is usually used most frequently because it is less expensive than flunixin meglumine. It is usually given at a dose of 4.4 mg/kg PO once daily for 7–10 days as the initial treatment. This appears to effectively break the pain cycle and allows for adjustment to corrective trimming and shoeing.[96] NSAIDs may be used intermittently thereafter if they are permitted for the athletic activity of the horse.

Intrasynovial Medication

Injecting medications into the DIP joint or navicular bursa is often used as an adjunctive treatment for horses with navicular disease/syndrome.[3,25,27,75,102,127] Injection of medications into the digital flexor tendon sheath (DFTS) may also benefit some horses with more proximal lesions of the DDFT that are documented with MRI.[106] Deciding when to treat horses with navicular disease/syndrome intrasynovially and with what medication(s) to use is strictly empirical based on many clinical and diagnostic findings. The types of medications used are similar to those used to treat OA/synovitis/capsulitis in joints at other locations, such as corticosteroids alone, corticosteroids combined with hyaluronan (HA), polysulfated glycosaminoglycans (PSGAGs), or other biologic therapies.

Horses with navicular disease are thought to benefit from intra-articular treatment of the DIP joint by reducing the inflammatory response both within the joint and within the navicular region.[18,85] Studies have demonstrated that methylprednisolone acetate (MPA) or triamcinolone (TA) can diffuse between the DIP joint and navicular bursa after intra-articular or intrabursal injection.[18,85] Other studies have documented the benefit of intrabursal injections in providing substantial but temporary relief of pain in many horses with navicular disease.[27,75,127] The benefit of DIP joint injections is less well documented, but anecdotally it is considered effective in many horses.[4,25] One study did not find a significant reduction in lameness after treating the DIP joint with TA compared to corrective shoeing alone although the overall severity of lameness was further reduced following treatment.[102] However, many clinicians use intrasynovial treatment of the DIP joint as part of the initial treatment regimen, together with corrective trimming and shoeing and phenylbutazone for most horses with navicular disease/syndrome.

In many horses, especially if advanced imaging cannot be performed, there is a stepwise progression of

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treatment options based on the clinical response. The decision to use intrasynovial treatment is often made at the first or second shoeing interval and is usually based on the clinical improvement in lameness. In most cases, treatment of the DIP joint is performed before treatment of the bursa because it is easier to perform and has fewer potential complications. However, horses with radiographic abnormalities confined to the flexor surface of the navicular bone would most likely respond better to treatment of the navicular bursa than the DIP joint. Intrasynovial medication strategies including whether to use anti-inflammatories vs. biologics may be greatly improved if a complete diagnostic picture of the foot abnormalities can be determined with the help of MRI.[3]

Both TA (5–10 mg) and MPA (20–60 mg) may be used for intrasynovial treatment, but MPA is usually reserved for use in the navicular bursa rather than the DIP joint. In most cases the corticosteroid is combined with HA, and amikacin is included when injecting the navicular bursa.[25,27,75] An immediate improvement in lameness is often seen in the majority of cases after intrabursal treatment.[27,75,127] In the most recent study, 75% of treated horses returned to their intended use for a mean of 9.6 months.[75] Horses treated within 6 months of becoming lame were more likely to return to performance. Intrabursal injections appear to reduce the clinical signs of navicular disease in most horses for several months, but repeated injections may be necessary and potentially predispose to rupture of the DDFT in some horses. In general, horses with multiple abnormalities within the foot and those with chronic adhesions from the navicular bone to the DDFT do not respond well to intrabursal treatment.[75]

Polysulfated Glycosaminoglycans

PSGAGs have been used in horses with navicular disease/syndrome based on the assumption that the etiology may be similar to that of OA. One double-blinded clinical trial documented a benefit of 500 mg of PSGAG IM at 4-day intervals for eight treatments compared with saline controls.[24] In addition, PSGAG given weekly or every other week IM was thought to benefit western performance horses with chronic navicular problems.[25] PSGAG may be injected intrasynovially into the DIP joint or navicular bursa, similar to corticosteroids, but no studies have documented the benefit of this in horses with navicular disease. Therefore, intrasynovial PSGAG treatment is not commonly used in horses with navicular disease.

Bisphosphonates: Tiludronate or Clodronate

Bisphosphonates, such as tiludronate and clodronate, are drugs that have been suggested as a treatment for horses with navicular disease because of their ability to promote osteoclast-mediated bone resorption. Areas of increased bone resorption and formation are often typical of lesions within a diseased navicular bone and areas of OA, and clinicians believe that reducing bone turnover in these conditions may have beneficial clinical effects. A multicenter, double-blind, placebo-controlled clinical trial using clodronate in horses with navicular syndrome found that 75% of treated horses improved at least one grade of lameness at day 56 compared with 3% in the placebo group.[53] At day 180, 65% of horses were still considered to have an improvement in their lameness.[53] An additional study has also suggested that a single dose of clodronate reduced lameness in horses with navicular syndrome without producing detectable effects on bone turnover markers.[77] Clodronate is an FDA-approved bisphosphonate licensed for treatment of navicular syndrome in horses (Osphos®, Dechra Veterinary Products, Overland Park, KS). Current recommendations for Osphos administration are 1.8 mg/kg by IM injection with a maximum dose of 900 mg per horse (e.g. 900-mg dose standard for a 500-kg animal); recommendations to divide the dose between three sites are also given (Osphos). Tiludronate is currently not licensed for use in the United States but is labeled for use in other countries for horses with navicular disease, bone spavin, and fetlock suspensory ligament enthesopathies. The drug is currently recommended at a dose of 1 mg/kg IV over 30 minutes as a single treatment instead of 10 daily treatments.

Tiludronate has been used either systemically (1 or 0.1 mg/kg IV) or locally (0.1–0.2 mg/kg) using intravenous regional limb perfusion (IVRLP).[29,103,129] In one clinical trial of 73 horses, tiludronate (0.1 mg/kg) given IV once daily for 10 days resulted in clinical improvement of lameness, and horses returned to a normal level of activity 2–6 months' posttreatment.[29] Another study reported that systemic tiludronate (1 mg/kg IV) appeared to be beneficial in horses with navicular syndrome but no horses became sound.[129] Using tiludronate locally with IVRLP (0.1 and 0.2 mg/kg) was reported to provide no further improvement in lameness over therapeutic shoeing and injection of the DIP joint.[103] Further studies are necessary to determine the true efficacy of these drugs in treating horses with navicular disease/syndrome. See Chapter 8 for more information on bisphosphonates.

Surgical Treatments

Palmar Digital Neurectomy

PD neurectomy remains the most commonly performed surgical technique to manage horses with navicular disease/syndrome. It is usually performed as a last resort after other treatment options have failed. It should be done in conjunction with corrective hoof trimming and shoeing to reduce abnormal forces on the foot, thus slowing the progression of the degenerative changes associated with the navicular syndrome.[4,115] A PD nerve block should always be performed before the surgery to document how much clinical improvement can be expected following the neurectomy.

Horses with suspected or confirmed abnormalities of the DDFT at the level of the navicular bone are usually not good candidates for a neurectomy because of an increased risk of rupture of the DDFT. However, an increased risk of DDFT rupture post neurectomy has not been documented, but a recent study recommended that horses with core or linear lesions of the DDFT should not undergo a PD neurectomy as these horses experience residual lameness or recurrent lameness aftersurgery.[58] The decision of whether to perform a

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neurectomy is often based on the severity of lameness, age and intended use of the horse, known abnormalities based on imaging results, responses to previous treatments, and the wishes of the owner.

Various methods of PD neurectomy have been described, and all have been developed to ensure complete desensitization, prevent painful neuroma formation, and prevent axonal regrowth, which may result in reinnervation.[26,58,63,74] Choosing which surgical technique to perform is often a personal preference of the veterinarian as several studies have failed to determine the ideal PD neurectomy technique. The pull-through or stripping technique is recommended by the author, and the clinical results appear to be very good.[26,74] The advantages of the pull-through approach are that no special equipment is needed, it can be easily performed in the standing or recumbent horse, and the end of the transected nerve is not located within the incision. Regardless of the technique used for a PD neurectomy, a clean and atraumatic surgery and diligent postoperative care will minimize complications.[26,63,74] Limited activity for 30–60 days and careful bandaging of the limbs for at least 3–4 weeks are thought to reduce the inflammatory response created by the surgery and reduce scar tissue formation around the nerve stumps.

The prognosis of horses following a PD neurectomy appears to be very good initially but becomes less favorable as time progresses. In a study of 57 horses, 74% of the horses were sound at 1 year, but this decreased to 63% after the second year.[63] In a study of horses that underwent the pull-through neurectomy technique, 88% were free of lameness at 1 year.[74] Survival analysis that was used to assess the time to recurrence of lameness indicated that the mean survival with no lameness after surgery was estimated at 4.14 ± 0.33 years (median, 5 years).[74] A more recent study reported that 92% of horses responded positively to surgery and were able to return to previous athletic use for a medial of 20 months.[58] The most significant complications after a neurectomy include rupture of the DDFT, regrowth of the nerves resulting in recurrence of the lameness, and severe infection within the foot that goes unnoticed.

Inferior Check Ligament Desmotomy

Inferior check ligament desmotomy has been used in selected cases of horses with navicular syndrome in which dorsal palmar hoof imbalance (either broken-forward or broken-backward axis) appears to be a major contributing factor. In one study, four horses with navicular syndrome that were treated with an inferior check ligament desmotomy all returned to full use, and the surgery made it possible to align the hoof-pastern axis after surgery.[124] This surgery seems to be most useful in horses with an upright heel conformation and a broken-forward hoof-pastern axis, as is sometimes seen in Quarter horses with mismatched front feet. More recently the procedure has been used in horses with chronic DDF tendinopathy to supposedly reduce the tension on the DDFT, comparable with the superior check ligament desmotomy procedure that is performed in horses with SDF tendinitis.[62,106] The desmotomy is thought to reduce the strain on the DDFT, lengthen the musculotendinous unit of the DDFT, and minimize the compressive forces on the navicular bone.[62,106] The goal is to promote tendon healing while simultaneously reducing pain and lameness. Further clinical cases will need to be assessed to determine the effectiveness of this procedure.

Endoscopy/Bursoscopy

Endoscopy of the navicular bursa has become the preferred surgical treatment of horses with infections or wounds that enter the navicular bursa. Endoscopy of the bursa may also be performed as both a diagnostic and treatment tool in a select group of horses with navicular disease using a modified surgical approach.[113,114] Horses with known or suspected abnormalities on the flexor surface of the navicular bone (flexor cortex erosions) and those with DDFT lesions (dorsal abrasions) at the level of the navicular bone are the ideal candidates for navicular endoscopy (Figure 4.17). In a study of 114 horses that had navicular endoscopy, 92 horses had injuries within the bursa, and 98% of the injuries involved the DDFT.[113] Sixty-one percent of horses returned to work sound with only 42% returning to their previous level of performance. As would be suspected, horses with extensive DDFT lesions and those with combination injuries of the DDFT and navicular bone identified with MRI had worse outcomes. Although endoscopy of the navicular bursa should be considered as a treatment option for selected horses, the overall outcome of horses may not be that different than horses managed conservatively with rest and rehabilitation.[21,106] Careful case selection may improve the advantage of surgery over conservative management, but in general the popularity of using bursoscopy to treat horses with DDF tendinopathy is less than it was a few years ago.[106] Advanced imaging with CT or MRI is usually necessary prior to endoscopy to optimize the potential benefit of surgery.

Prognosis

The prognosis of horses with navicular disease/syndrome is difficult to predict because of the numerous bone and soft tissue abnormalities that can occur concurrently. Radiographs alone are not adequate to completely assess the pathology within the foot and therefore cannot be used alone to determine the prognosis unless the abnormalities are advanced. It is also known that the absence of radiographic abnormalities of the navicular bone does not indicate the lack of bone pathology.[101] The combination of bone and soft tissue lesions is usually present in most horses, which can greatly alter the prognosis.[39,44,101] In addition, most abnormalities are degenerative in nature and worsen with time and continued use of the horse. In general horses with combination injuries to the DDFT and navicular bone tend to have worse outcomes with many remaining persistently lame.[44,106]

In most instances, the numerous treatment protocols are probably more appropriately termed management strategies because the disease/syndrome is unlikely to resolve completely. In general, clinical resolution may occur in 40%–50% of the horses with navicular disease/syndrome, but the optimal treatment and prognosis will

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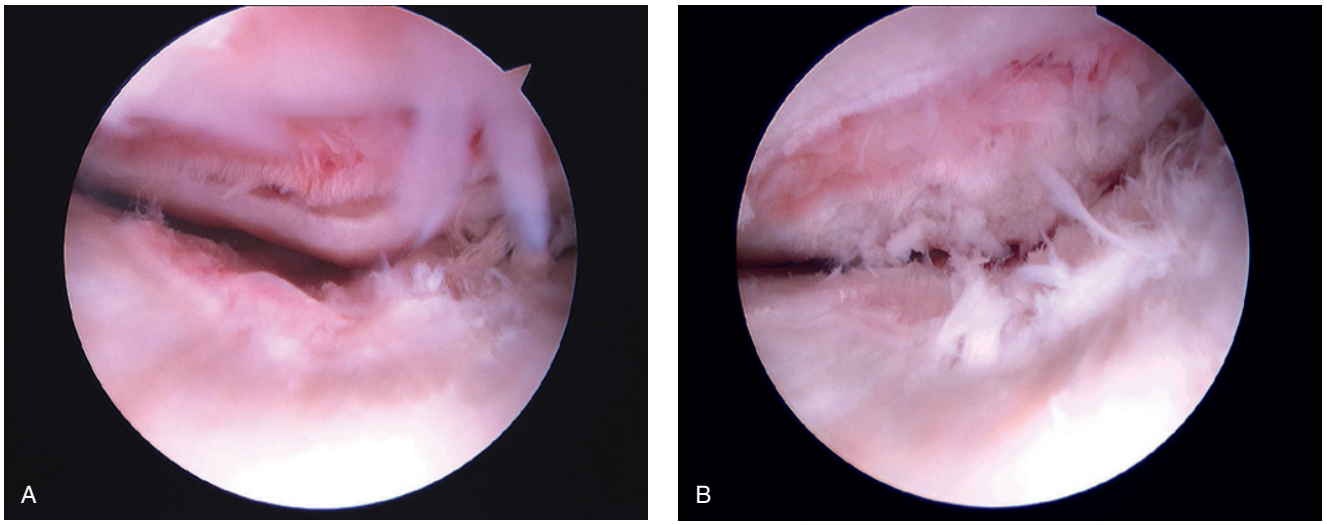


Figure 4.17. Endoscopic view of the flexor cortex lesion on the navicular bone (A) and the fibrillation of the DDFT (A and B) that was present in the horse in Figures 4.11 and 4.13. The navicular bone is at the top of the image and the DDFT is on the bottom.

differ depending on the specific pathologies that may be present.[3,39,41,44,96] For instance, horses with major radiographic abnormalities most likely will have a worse prognosis than horses with no radiographic changes and minor soft tissue pathology. Horses with navicular bone pathology demonstrated on radiographs or MRI together with concurrent DDFT lesions will also tend to have a poor prognosis. In general, horses with primary soft tissue injuries are thought to have a guarded prognosis for return to full athletic function with injuries of the DDFT being the most problematic.[21,106] Horses with significant lesions in the navicular bone per se or combination bone and soft tissue injuries usually have a poor prognosis for performance.[39,41,44,96,106] Due to the diversity of underlying problems associated with the disease, the key to controlling navicular disease/syndrome is most likely prevention.[4,96] Good foot care, proper trimming and shoeing, maintaining correct dorsopalmar hoof balance, and preventing overextension of the palmar aspect of the foot cannot be overemphasized due to the biomechanical nature of the disease.

FRACTURES OF THE NAVICULAR (DISTAL SESAMOID) BONE

Fractures of the navicular bone are an uncommon cause of lameness in horses.[54,72,116] They have been reported in many breeds and in horses with varied use.[5,72] Complete fracture can occur after acute trauma or secondary to severe bone demineralization due to navicular disease or osteomyelitis from sepsis. [5,72,95] Avulsion fractures usually occur along the distal border of the navicular bone and are thought to be associated with navicular disease,[7,8,37] although this is not universally accepted.[137] The forefeet appear to be at a greater risk for fracture than the hindfeet.[5,59,72] In one report, 22 of 25 fractures of the navicular bone were in the forelimbs,[59] and in another, 15 of 17 were in the forelimbs.[72]

Fractures of the navicular bone have been classified as avulsion fractures/fragments, simple complete fractures (transverse or oblique), comminuted complete fractures,

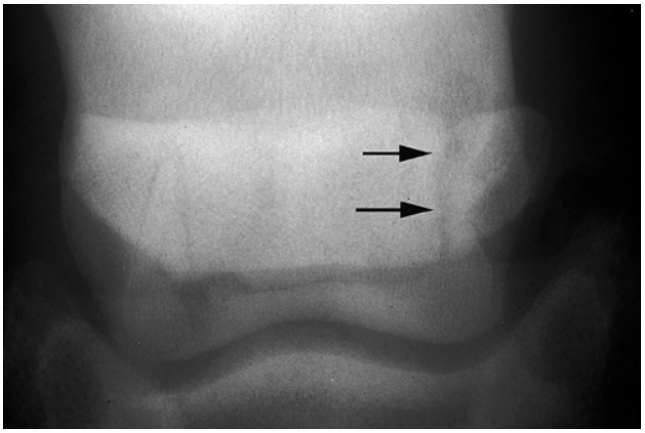


Figure 4.18. A wing fracture of the navicular bone (arrows) as seen on the oblique radiograph of the foot.

and congenital bipartite navicular bones.[37] The latter is not considered a true fracture but can be confused with a chronic fracture based on its radiographic appearance. Avulsion fractures/fragments usually involve the distal border of the navicular bone in the forefeet and are frequently associated with other radiographic signs of navicular syndrome.[8,37] These fracture fragments vary in shape, are usually small (0.2–1.2 cm in size), and occur more commonly in lame horses than clinically normal horses (Figure 4.12).[7,8,37] Simple complete fractures may be vertical, slightly oblique, or transverse.[37,66] The vertical and slightly oblique fractures usually occur medial or lateral but fairly close to the central eminence (sagittal ridge) of the navicular bone. Generally, these fractures are not displaced, but they are usually slightly separated so an obvious fracture line exists on the radiograph (Figure 4.18). Comminuted complete fractures are even more uncommon than simple complete fractures (Figure 4.19).[5] In one report only 3 of 18 horses with complete navicular bone fractures were comminuted.[5] Rupture of the distal sesamoidean (impar) ligament or DDFT may accompany these fractures.[60,116]

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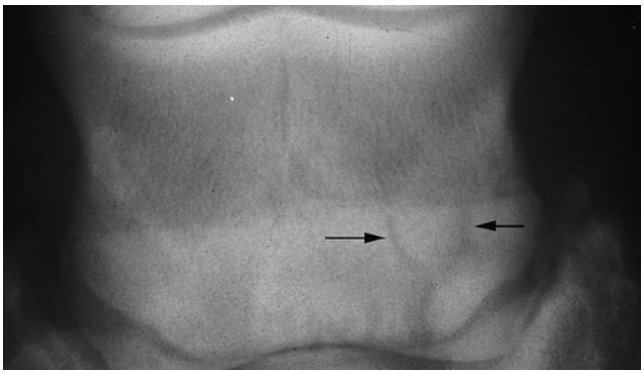


Figure 4.19. A comminuted Y-shaped fracture of the navicular bone (arrows).

Etiology

Acute trauma (concussion) to the foot is the most likely cause of most simple and comminuted complete navicular bone fractures. However, severe navicular bone osteolysis associated with navicular disease or sepsis may predispose to pathologic fractures. Avulsion fractures are most likely due to pathologic changes occurring within the navicular bone associated with navicular disease but may also be trauma induced.

Clinical Signs

The severity of lameness and clinical signs in horses with avulsion fractures are similar to those in horses with navicular disease. However, there may be a history of sudden worsening of the lameness in a single limb that responded to rest. Horses with complete navicular bone fractures typically have a history of an acute severe lameness in a single limb that improves with time. One retrospective study found that most horses were severely lame at presentation and that the less lame horses were evaluated at a mean time of 90 days after the onset of lameness (range, 30–150 days).[72] In another study, the mean duration of lameness before presentation was 4.3 months.[5]

Most horses have a painful response to hoof testers across the frog region and have effusion within the DIP joint. An increased digital pulse may be present in the acute stage, similar to horses with P3 fractures, but this is rare in more chronic cases. Horses with significant hindlimb lameness (grade 2–3 out of 5) and clinical signs referable to the navicular region should be suspected of having a navicular bone fracture. A PD nerve block should improve the lameness in most cases although anesthesia at a more proximal level (abaxial sesamoid nerve block) may be required. In some horses,[72] the reason for this is uncertain since the PD block is thought to desensitize most if not all of the foot including the DIP joint. However, the fracture may cause articular pain or pain within the DDFT that is not completely eliminated by a PD nerve block. Intra-articular anesthesia of the DIP joint usually eliminates the lameness associated with navicular bone fractures.[116]

Diagnosis

Radiographic examination of the foot is required to confirm the diagnosis. Careful packing of the frog is necessary to avoid confusing the lines from the lateral sulci of the frog that cross the navicular region with a fracture. If the line extends beyond (above or below) the navicular bone, it is usually not a fracture. When in doubt, it is best to retake the radiograph at a slightly different angle. Complete simple fractures are typically located in the sagittal plane medial or lateral to the midline (parasagittal).[37,95] Most complete fractures are best identified on the skyline or 60° oblique views of the navicular bone and should be present on multiple views (Figures 4.18 and 4.19). The fracture line should begin and end at the edges of the navicular bone and is usually easily visible, especially in chronic fractures. Navicular bone fractures need to be differentiated from congenital bipartite or tripartite separation.[37,72] Bipartite navicular bones are usually symmetric with smooth edges and have a wide radiolucent region (Figure 4.6).[4,37] They are often bilateral so the opposite navicular region can be imaged to confirm the diagnosis.

Avulsion fractures can be difficult to identify on radiographs and close scrutiny is often required. They can often be best seen on the 60° oblique view of the navicular bone but may also be present at the distal aspect of the navicular bone on a lateromedial view (Figure 4.12) or within the medullary cavity of the navicular bone on the skyline view.[8,37] There is some question as to whether these osseous fragments represent true avulsion fractures of the navicular bone or are ectopic mineralization of the DSIL or a fracture of an enthesophyte at the origin of the DISL.[37]

Treatment

There is no known specific treatment for avulsion fractures of the navicular bone. Horses are treated similar to those with navicular syndrome (see navicular disease/syndrome above) but may benefit from heel elevation (provided the heels are not already too long) to relieve tension on the DSIL and DDFT. Horses with complete navicular bone fractures are usually treated nonsurgically by confinement alone, confinement and corrective shoeing (usually with heel elevation), or external coaptation aimed at reducing hoof expansion.[5,54,72]

In one study, horses were treated with 12° of heel elevation by using 4 3° wedge pads and a flat shoe.[123] The large amount of heel elevation was thought to be an important aspect of treatment, but it is likely that more moderate levels of heel elevation (3°–6°) may also be beneficial. Regardless of the amount of heel elevation used, the horse should be reshod monthly and gradually returned to normal foot angles over a 4- to 6-month period. A minimum of 4–6 months of stall rest has been recommended because these fractures are very slow to heal.[4,72,116] One study found that a minimum of 6 months of stall rest was necessary before there was resolution of clinical signs.[72]

Surgical repair of simple complete navicular bone fractures using a single cortical bone screw placed in lag fashion has been reported.[81] Intraoperative imaging (fluoroscopy, radiography, or CT) and a specially developed aiming device is necessary to implant the screw precisely along the transverse axis of the navicular bone.[54] This technique was reported to be successful in 4 of 5 horses, and all fractures healed without excessive

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callus.[81] Computer-assisted surgery systems may provide further advantages in screw placement in the future.[54]

Palmar or plantar digital neurectomy can also be performed to relieve pain in cases that have not responded to conservative treatment.[5,72] The navicular bone is very slow to heal, and these fractures are invariably associated with damage to the impar ligament, DDFT, and DIP joint.[5,60] Chronic lameness may result from poor fracture healing and adhesions that develop between the DDFT and the navicular bone.[72] Follow-up radiography on 17 horses with complete navicular bone fractures revealed increases in the width of the fracture gap for up to 4 months after injury.[72] A noncalcified fibrous union can still be evident years after the fracture occurred, although some may heal completely (Figure 4.20).[4,116] Failure of an osseous union may be due to a combination of the soft tissue attachments and loading forces continually distracting the fracture fragments. Also the navicular bone is located between two synovial spaces, and synovial fluid may also inhibit healing. If a neurectomy is performed, prolonged rest afterward should be considered to permit the fracture to heal and prevent further damage to the surrounding soft tissue structures.[116]

Prognosis

The prognosis is considered guarded to poor for horses with complete navicular bone fractures to return to athletic performance. In general, horses with fractures in the hindlimb are considered to have a better chance to return to performance than those affected in the forelimbs. In one report 3 of 6 horses treated by stall rest alone or stall rest and external coaptation, and 2 of 4 horses treated with stall rest and corrective shoeing returned to their intended use.[72] Also, only 1 of 5 horses that underwent neurectomy returned to its intended use.[72] In another report 3 of 7 horses treated with rest alone returned to performance and 4 remained lame. Of the 6 horses that were treated with a neurectomy, 2 were sound for light riding, 2 were sound for only 1 year, and 2 remained lame.[5] In the study that used a 12° elevated heel shoe together with confinement, 4 of 4 horses became serviceably sound for riding, and 2 of 4 returned to competition.[123] Although the horses were sound for use, complete radiographic healing did not occur in any case in this study.[123] Important clinical aspects of navicular bone fractures that can be summarized from these clinical studies include the following:

1. Heel elevation should be an important aspect of treatment.
2. Four to 6 months of confinement may be necessary.
3. Complete fracture healing is unlikely to occur.
4. A PD neurectomy will minimize the lameness but does not guarantee a sound horse.

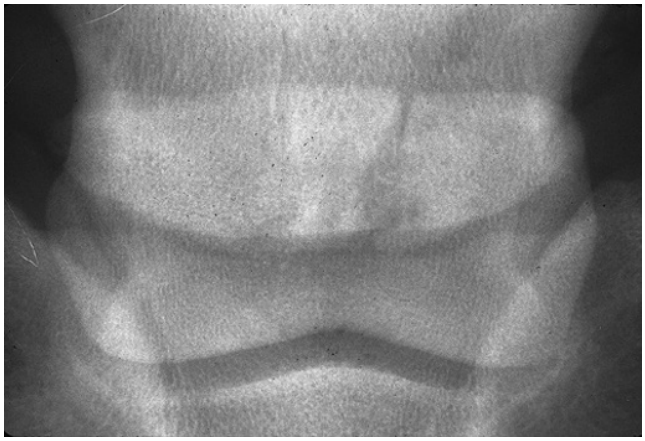


Figure 4.20. This oblique radiograph of the navicular bone was taken 23 months after the fracture occurred.

SOFT TISSUE INJURIES IN THE FOOT (DDFT AND PODOTROCHLEAR APPARATUS)

Multiple bone and soft tissue structures within the foot are desensitized with a PD nerve block.[111] Historically, radiography has been used to help differentiate between bone and soft tissue injuries, but the lack of radiographic abnormalities within the navicular bone does not indicate the lack of pathology.[2,84,101] In addition, radiographic scores of the navicular bone do not accurately predict navicular bone pathology.[84] Soft tissue injuries of the foot have always been suspected in horses with foot pain without radiographic abnormalities, and advances in ultrasound, CT, and MRI techniques have enabled improved recognition of these potential problems.[19,20,38,39,41–45,56,84,90,91,125,131] These imaging modalities have helped determine that it is common for soft tissue and bony abnormalities to coexist in many lame horses with foot problems, and multiple abnormalities are often present. For instance, several MRI studies have documented the association between DDFT lesions and navicular bone pathology, although primary tendinitis of the DDFT and other primary soft tissue injuries do occur.[39,41,42,44,84,105,112]

Soft tissue structures within the foot that can be desensitized with a PD block include the heel bulbs, digital cushion, navicular bursa, collateral cartilages of the distal phalanx, podotrochlear apparatus (DSIL, and CSLs of the navicular bone), DDFT (may only partially improve with a PD block), and CLs of the DIP joint.[35,42,44,84,111] Soft tissue abnormalities of the podotrochlear apparatus and the DDFT are most closely associated with navicular syndrome in horses and are therefore discussed below. Injuries to the CLs of the DIP joint are covered in the section on the DIP joint later in this chapter.

Etiology

The DDFT is the most commonly affected soft tissue structure in the foot and injuries may occur alone, in conjunction with navicular bone pathology, or as one component of a complex of multiple soft tissue injuries in the foot.[38,39,41–44,84,104–106] The DDFT is bilobed within the foot, and lesions can occur in either lobe anywhere along its length, from the level of the PIP joint distally to its attachment on P3.[104] Retrospective studies have indicated a prevalence of 30%–64% in horses with foot lameness with no or equivocal radiographic abnormalities of the foot.[106] Four primary lesion types in the distal portion of the DDFT have been identified: core lesions,

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dorsal border lesions, sagittal plane and oblique splits, and insertional lesions.[105,106] Abnormalities have been identified at the DDFT insertion to the distal phalanx (least common), the level of the navicular bone or CSLs (most common), more proximally in the pastern, or a combination of sites.[38,39,41–44,78] The most common location is at the level of the navicular bone and CSLs and can be true core lesions, sagittal splits, or dorsal abrasions (Figure 4.13).[10,11,39,44] DDFT lesions can occur in a single or multiple locations and can extend variable distances up or down the tendon (Figure 4.21). Abnormalities of the DDFT at the level of the proximal phalanx are more typical of true core lesions within the tendon.[39]

Abnormalities of the podotrochlear apparatus are often present in association with abnormalities of the navicular bone, especially involving the proximal or distal borders and the medulla.[39,44] Acute-onset or repetitive trauma is considered the most likely cause of most soft tissue injuries within the foot.[35,36] Concurrent abnormalities of the podotrochlear apparatus and navicular bone suggest that similar biomechanical forces and repetitive trauma to the palmar aspect of the foot likely contribute to both types of injuries.[39] However, primary injuries to the podotrochlear apparatus (CSL + DSIL) do occur.[84]

Horses that jump or have a low-heel hoof conformation may be at risk for injuries to the DDFT.[36,105] Western performance horses, especially reining and cutting horses, are also considered at risk because of the work that they are required to perform. Chronic repetitive trauma to the DDFT is often the most likely cause although single-event traumatic "tearing" of the DDFT causing a true tendinitis may also occur. Based on MRI studies, this type of lesion is most likely to occur more proximal in the foot at the level of the proximal phalanx.[39] Exceptions to this may be true ruptures of the DDFT, but the location of where these ruptures occur has not been documented. In many cases, however, it would seem likely that previous chronic abnormalities of the DDFT would predispose to complete failure. In addition, chronic DDFT injuries are not always isolated to a single site (Figure 4.21), and there is evidence to suggest that DDFT lesions may be a degenerative process due to vascular compromise rather than an inflammatory process from trauma.[11]

Clinical Signs

In general, horses that have primary soft tissue injuries in the foot are more likely to have a history on an acute onset of lameness and be unilaterally lame compared with horses with navicular disease. Horses with multiple foot problems and those with concurrent navicular pathology and soft tissue injuries are less likely to conform to this generalization. In addition, horses with flexor cortex erosive lesions of the navicular bone are often unilaterally lame. The majority of horses will improve with a PD nerve block, but the lameness will not be completely abolished in many horses with lesions of the DDFT.[36,42] More specific clinical information has been obtained from horses with injuries to the DDFT and CL of the DIP (see section of DIP joint) than from horses with injuries to the podotrochlear apparatus. The clinical signs of horses with abnormalities of the podotrochlear apparatus may resemble those with navicular disease because these injuries often occur concurrently with navicular bone pathology.[39] A large clinical study indicated that horses with primary DDFT lesions were more likely to exhibit pain on turning than horses with other types of lesions within the foot and that horses with navicular pathology combined with injuries to the podotrochlear apparatus (±DDFT lesion) were more likely to be unilaterally lame.[84]

The clinical signs may vary, depending on whether the DDFT lesion is primary or associated with navicular

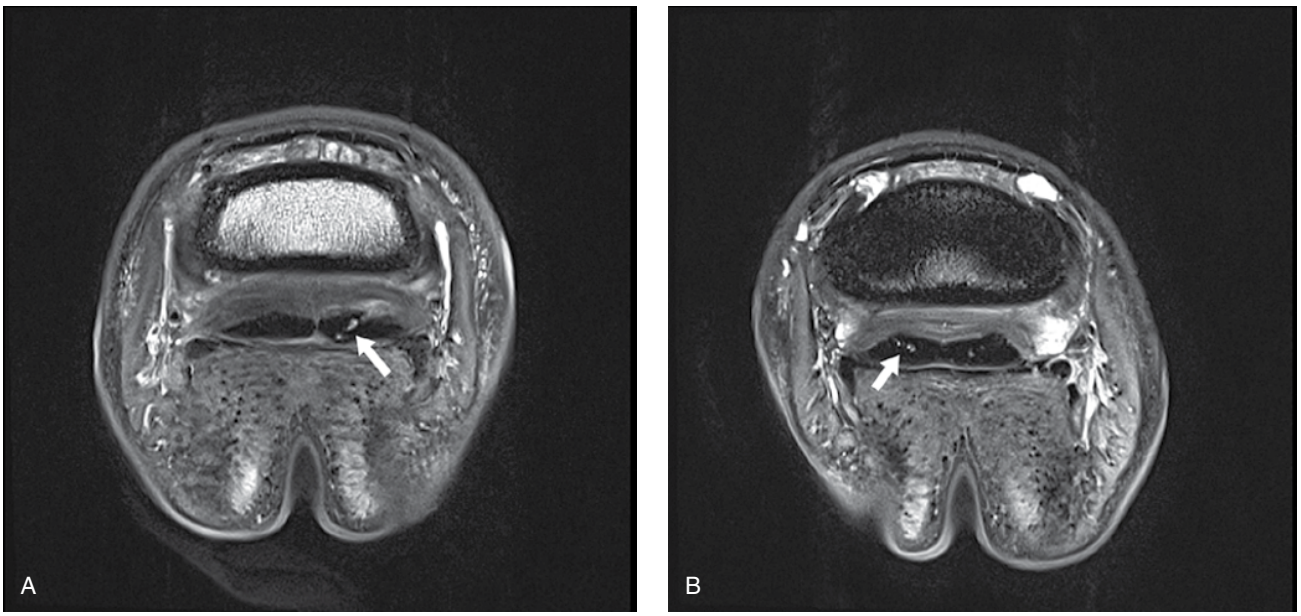


Figure 4.21. MRI images demonstrating a single large DDFT lesion (A; arrow) and multiple smaller DDFT lesions (B; arrow) at different locations along the DDFT in the same horse.

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pathology. However, there is often a history of an acute onset of moderate to severe lameness that may improve with rest and worsen with exercise.[35,36] There may be a history of activity that caused excessive hyperextension of the foot such as working in soft ground and/or jumping. The lameness may be unilateral and may worsen in a circle or when exercised on soft ground. Occasionally, pain may be elicited with deep palpation of the DDFT between the collateral cartilages of the heels. Hoof tester pain is variable but may be present if a DDFT lesion and navicular pathology are present concurrently. Phalangeal flexion may cause a positive response but is often variable. Increasing tension on the DDFT with the navicular wedge test may accentuate the lameness.[35,36]

The lameness is not reliably abolished with a PD nerve block in horses with DDFT lesions.[36,42] Horses often improve but in one study only 24% of those with DDF tendinitis responded completely to a PD nerve block.[42] The lameness should respond to basisesamoid block and many improve after IA anesthesia of the DIP joint. Anesthesia of the DIP joint was more effective in alleviating the lameness in horses with DDFT lesions than was the PD block in one study.[42] This finding differs from the clinical experience of the author. However, the response to perineural anesthesia may depend on the location of the lesion(s) and whether concurrent problems exist in the foot.

Diagnosis

A definitive diagnosis of a soft tissue injury of the foot is best determined with MRI in either the recumbent or standing patient.[38,39,41–44,78,105,106] Tendon damage within the DDFT is often seen as focal signal increase on both the T1- and T2-weighted sequences and swelling of the affected lobe in the acute stages of the disease. There is good correlation between MRI appearance of DDFT lesions and their pathological classification into core lesions, sagittal splits, insertional lesions, and dorsal surface erosions (Figures 4.21 and 4.22).[104,106] See Chapter 4 for more information on MRI evaluation of DDFT lesions.

Horses with primary DDF tendinitis often have minimal to no radiographic abnormalities. This is not the case with concurrent DDFT injuries. Ectopic mineralization may be seen in some horses with DDFT lesions but may not be correlated with tendinitis of the DDFT (Figure 4.23).[36] Enthesophytes involving the podotrochlear apparatus attachments to the navicular bone and other radiographic abnormalities of the navicular bone may suggest damage to these structures but do not provide a definitive diagnosis. Likewise, erosive lesions of the flexor surface of the navicular bone are most likely associated with dorsal abrasions of the DDFT, but navicular bursal endoscopy or MRI is usually needed for a definitive diagnosis (Figure 4.24). Not all of these erosive lesions can be identified with radiography and may require standing low-field or high-field MRI for documentation.[112] Histological examination of a few of these horses documented fibril formation and fibrocartilaginous metaplasia of the adjacent DDFT.[112]

Ultrasound can aid the diagnosis of some injuries to the DDFT and podotrochlear apparatus but is most useful to help document injuries to the CL of the DIP



Figure 4.22. A core lesion within lobe of the DDFT above the level of the navicular bone (arrow) can be seen in this PD axial MR image. *Source*: Courtesy of Dr. Natasha Werpy.

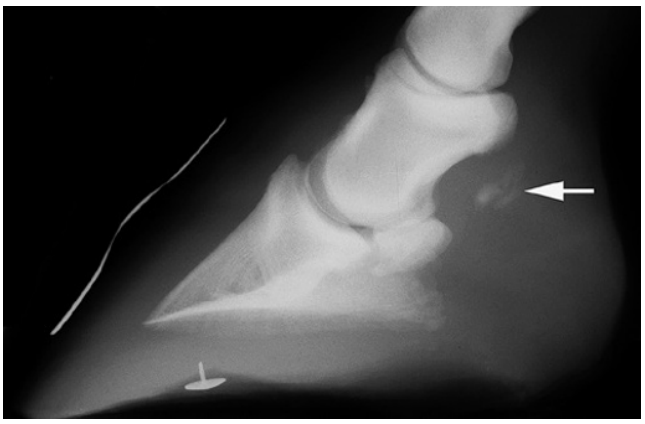


Figure 4.23. Lateral radiograph of a horse with chronic navicular disease that demonstrates calcification within the DDFT proximal to the navicular bone (arrow).

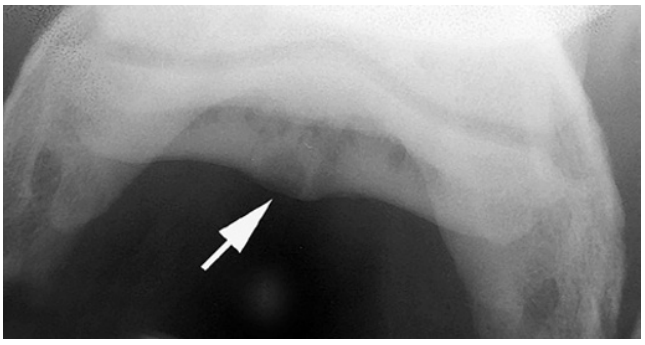


Figure 4.24. Secondary dorsal border lesions of the DDFT should be suspected in horses with erosive lesions on the flexor surface of the navicular bone (arrow).

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joint.[36,45,56] Ultrasound can be performed either between the heel bulbs or through the frog after it has been softened by soaking. However, documenting the lesion may be difficult depending on its location and the skill of the operator. Certain aspects of both the DDFT and podotrochlear apparatus are inaccessible with ultrasound, suggesting that negative findings do not rule out the presence of a lesion.[56] Because of these limitations, ultrasound is currently being used to aid in the treatment of specific lesions within the DDFT that have been identified with other imaging modalities. Similar to MRI, contrast-enhanced CT can also be used to document abnormalities in the DDFT within the hoof capsule but can only be performed under general anesthesia.[89–92,131] See Chapter 3 for further information on the use of diagnostic imaging modalities within the foot.

Treatment

The most important aspects of treatment of soft tissue injuries of the foot are rest, rehabilitation, and corrective foot care.[57,104–106] Foot imbalances that may have contributed to their occurrence such as low heels, long toes, mediolateral imbalance, reverse angle of the distal phalanx, etc. should be corrected if possible. Horses with injuries to the podotrochlear apparatus are usually treated similarly to horses with navicular disease. Systemic and intrasynovial (DIP joint or navicular bursa) anti-inflammatory therapies are usually performed especially in horses with combination bone and soft tissue injuries.

Types of shoes that may benefit horses with DDFT injuries include egg-bar shoes, reverse shoes, and onion shoes, all fitted with a rolled toe.[106] The goal of shoeing is to prevent heel descent during loading in soft ground and promote early break-over at the toe.[106] Raised heel shoes may offer mixed results as they may paradoxically exacerbate lameness and may induce contracture of the DDFT during tendon healing.[106] Clinically, however, many horses with DDFT lesions initially appear more comfortable when placed in elevated heel shoes.

Rest and rehabilitation are usually performed over a minimum of 6 months and may be necessary for even longer depending on the lesion severity. The duration of rehabilitation is usually dependent on many variables including DDFT lesion type, location and size, concurrent hoof abnormalities, use of the horse, and wishes of the owner. In general, horses treated with an extended period of rest and rehabilitation are thought be able to return to use for a longer duration than horses without rest.[57,106] Even though rest and rehabilitation are important aspects of treatment, the prognosis with this therapy alone has not been considered favorable.[36,105,106] Adjunct treatment approaches for these injuries are discussed below.

Additional treatment options for horses with DDFT lesions often depend on the location of the lesion, but the overall prognosis remains poor. Dorsal border lesions at the level of the navicular bone may benefit from endoscopy of the navicular bursa and debridement of any torn tendon fibers.[113,114] Intralesional treatment of the DDFT injury and/or treatment of the navicular bursa with biologics or anti-inflammatories can be performed at the time of surgery or subsequent to surgery. Although navicular bursoscopy with tendon debridement may be indicated in select cases, its potential benefit over conservative treatment has been questioned.[106] Other surgical options in horses with DDFT injuries include desmotomy of the inferior check ligament to reduce the strain on the DDFT during healing and PD neurectomy if other therapies have failed to improve the lameness.[106]

Intralesional treatment of DDFT lesions with biologics at other locations may be performed with CT, MRI, or ultrasound guidance but may require the horse to be anesthetized.[3,90,91] A technique to inject the insertion of the DDFT at the level of P3 using radiographic guidance has also been described.[1] Intrabursal or intrasynovial injection of medications (biologics or anti-inflammatories) without navicular bursoscopy can also be performed. Corticosteroids alone or combined with HA may be used to reduce the inflammation and pain, or biological compounds such as stem cells or platelet-rich plasma may be used to hopefully promote tendon healing. Regional IV perfusion of stem cells has also been used in select horses with DDFT injuries, but the efficacy of this treatment is unknown. See Chapter 8 for more detailed information on these treatment modalities.

Prognosis

In general, horses with primary or secondary DDFT injuries within the foot have a poor prognosis for return to performance.[21,44,57,106] The injury is often a career-ending with less than one-third of affected horses being able to return to performance.[106] DDFT injuries are often negatively associated with prognosis for a variety of reasons and can be difficult and expensive to treat. In one study of primary DDFT injuries, only 25% of horses returned to their previous level of activity within 18 months. However, the prognosis was dependent on lesion type with dorsal border lesions having a better prognosis than core or parasagittal split lesions. Thirty-five percent of horses with dorsal border lesions returned to their original level of activity.[21,106] Additionally, core lesion size was reported to affect prognosis with lesions with a cross-sectional area of >20% of the affected lobe or a total length of >35 mm corresponding to horses that remained lame after treatment.[106,126] Therefore, the overall prognosis of DDFT injuries is often dependent on lesion size, type, location, and severity and the presence of concurrent abnormalities within the navicular bone or other soft tissues (primary vs. secondary). Several studies have documented that horses with multiple injuries within the bone and soft tissues of the foot have difficulty returning to performance.[21,36,44,57,106]

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COFFIN JOINT AND DISTAL PHALANX

Gary M. Baxter

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OSTEOARTHRITIS (OA) OF THE DISTAL INTERPHALANGEAL (DIP) JOINT

Osteoarthritis (OA) of the distal interphalangeal (DIP) joint, or "low ringbone," is a common cause of forelimb lameness in horses. It can be a primary cause of lameness, but more commonly, it occurs concurrently with other lameness conditions of the foot. Synovitis/capsulitis of the DIP joint is considered to be a less severe form of OA that may occur in some horses in the early stages of the disease process. Although advanced low ringbone can be associated with dorsal exostosis of the extensor process of P3, contributing to an enlargement at the coronary band and pyramidal distortion of the hoof, this occurs uncommonly.[5] Distortion of the hoof leading to pyramidal disease or buttress foot is most likely associated with large extensor process fractures of the distal phalanx.[15]

Etiology

Primary OA of the DIP joint can be due to acute or repetitive trauma to the joint comparable to any articulation in the horse. Horses with a broken pastern axis (forward or backward) and other types of hoof imbalances appear particularly prone to repetitive trauma to the DIP joint and development of OA. Acute or repetitive trauma may cause tearing of the joint capsule (capsulitis) and/or direct damage to the articular cartilage and subchondral bone. Excessive strain of the attachments of the long or common digital extensor tendon to the extensor process may also occur and contribute to periostitis and enthesophyte formation along the dorsal aspect of the joint.[5] Primary OA of the DIP joint not associated with trauma, conformational defects, or developmental abnormalities of the joint has also been reported.[50] This condition was characterized as a chronic condition with a slow onset of cartilage degeneration and reactive changes within the DIP joint of a single front limb. The diagnosis of this condition was challenging with radiographs alone, and low-field standing MRI was necessary to make the diagnosis.[50]

Secondary OA can occur from other lameness conditions that involve the DIP joint, either directly or indirectly. These include navicular disease, complete navicular bone fractures, articular fractures of the distal phalanx, subchondral cystic lesions (SCLs) of the distal phalanx, osteochondral fragmentation within the joint, and desmitis of the collateral ligaments (CLs) of the DIP joint.[5,18,19,21,47] These abnormalities are thought to directly or indirectly cause pathology to the DIP joint, which leads to the development of OA over time. Prevention of DIP joint OA is often an important aspect of treatment for many of these conditions. See the discussion of each of these conditions in this chapter for more information.

Clinical Signs

Effusion of the DIP joint is usually present in most horses with OA or synovitis/capsulitis of the DIP joint (Figure 4.25). However, effusion can be present in normal horses, so this finding is not always indicative of problems within the DIP joint.[17] However, sound horses usually have less DIP joint effusion that lame horses and the effusion is often symmetrical from right to left. Most times significant effusion of the DIP joint can be seen as a slight bulging just above the coronary band. The fluid can usually be balloted from medial to lateral along the dorsal midline of the joint. With chronic or advanced disease, the joint capsule may become thickened, resulting in a firm swelling just above the dorsal aspect of the coronary band. Digital pressure over the swelling may elicit a painful response.[5] The joint may be painful to flexion and rotation, but this is uncommon unless the OA is advanced or secondary to another problem in the joint. Lameness is variable and often depends on the severity of the disease, whether it is primary or secondary, and whether one or both limbs are affected.[17] The lameness is often worse on hard ground, when circled, and after distal limb or phalangeal flexion.

Lameness associated with the DIP joint is often improved and sometimes alleviated completely with a PD nerve block.[16,55] However, anesthesia at the base of the sesamoid bones or a pastern ring block may be required for complete resolution of the lameness. Intra-articular (IA) anesthesia of the DIP joint is not specific for problems within the joint, but using a small volume of anesthetic (6 mL or less) and observing for a change in lameness very soon after the injection (within 10 minutes)



Figure 4.25. Effusion within the DIP joint can be seen and palpated as swelling just above the coronary band (arrow).

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can improve the specificity of the block for the joint.[17,55] Most horses with DIP joint pain improve rapidly and substantially after IA anesthesia. If the lameness only partially improves but persists after 10 minutes, the DIP joint is not likely the primary site of the pain. In addition, a positive response to an IA block combined with a negative response to navicular bursa anesthesia often incriminates the joint as the primary problem area.[55] See Chapter 2 for more details related to perineural and intrasynovial anesthesia.

Diagnosis

A definitive diagnosis of OA of the DIP joint can often be obtained with radiography of the foot. However, horses with synovitis/capsulitis or early OA of the DIP joint may have no radiographic abnormalities. A complete radiographic study of the foot should be performed to rule out other potential problems because DIP joint OA may be secondary to other conditions and anesthesia of the DIP joint is not always specific for the joint.

Oblique views of the DIP joint can aid in detecting periarticular new bone formation of the distal aspect of the middle phalanx (Figure 4.26).[17,39] Close inspection of the extensor process, palmar/plantar aspect of distal P2, and dorsoproximal aspect of the navicular bone for osteophyte and enthesophyte formation is important. Joint space congruity and the shape of the proximal surface of the distal phalanx should be assessed carefully. Joint space narrowing is difficult to assess because limb positioning is known to affect joint space width.[10] In general, the radiographic abnormalities surrounding the DIP joint should not be overinterpreted because there is much variation in the shape of the extensor process among horses and enthesophytes may not be associated with lameness (Figure 4.27).[17,47] In contrast, the presence of any radiographic abnormalities of the DIP joint was associated with a poor response to treatment in one study.[16]

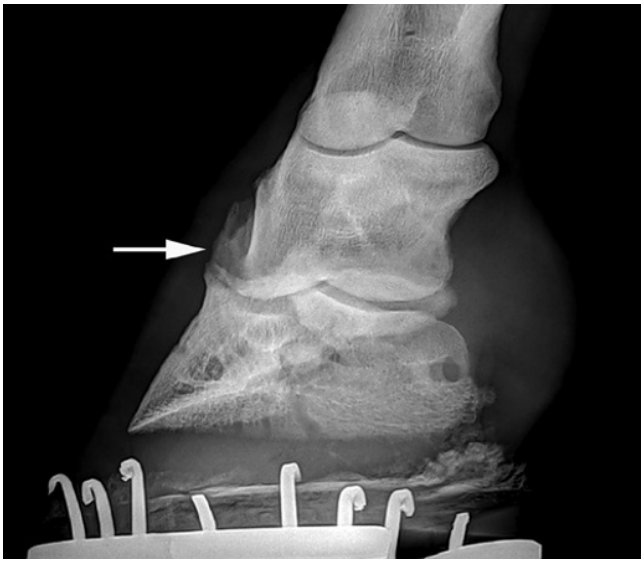


Figure 4.26. Oblique radiographs of the DIP joint are important to document abnormalities. The bony proliferation (arrow) and narrowing of the DIP joint seen on this oblique radiograph were not apparent on other views of the joint.



Figure 4.27. The calcification of the extensor tendon seen on this lateral radiograph was not clinically significant in this horse following removal of an extensor process fracture.

Additional diagnostics that may be used to confirm the diagnosis of a problem within the DIP joint, particularly if the radiographs are nondiagnostic, include ultrasound, MRI, and diagnostic arthroscopy. Ultrasound can be helpful to document problems within the CLs of the DIP joint proximal to the hoof wall and thickness of the dorsal joint capsule.[12,19] MRI is the most comprehensive advanced imaging modality that can detect articular cartilage, subchondral bone, and soft tissue abnormalities of the DIP joint if present.[18,20,21,26] Arthroscopy may be used to document articular cartilage or subchondral bone damage, but much of the joint surface of the DIP joint is not visible. Since many horses with DIP OA will have other abnormalities within the foot, MRI is usually the next imaging modality recommended after radiography.

Treatment

Horses with primary OA or synovitis/capsulitis of the DIP joint are usually treated with a combination of IA medication and corrective shoeing. Predisposing factors such as mediolateral and dorsopalmar hoof imbalances should be corrected to reduce repetitive trauma to the joint. Shortening the toe and moving the break-over further palmarly often helps these horses, and using a rim pad may alleviate concussion to the joint.[5] Direct medication of the DIP joint is usually more effective than systemic medications to reduce the inflammatory response within the joint. The type(s) of medication chosen may depend on the severity and duration of the lameness and the severity of the radiographic abnormalities. Corticosteroids alone or corticosteroids combined with hyaluronan are used most frequently. Chondroprotective agents such as autogenous serum or PSGAGs may also be used if desired.

Typically, the author recommends a combination of hyaluronan and triamcinolone (TA). However, one study reported that horses treated with three weekly injections of the DIP joint with PSGAG had a 67% successful outcome, compared with 46% of those receiving MPA alone.[35] In addition, a significantly better result was obtained in

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dressage horses than in jumping horses (eventing and show jumping).[35] This study and the clinical observations of the author support the rationale for treating horses with DIP joint pain with IA medication although horses that block completely with DIP joint anesthesia usually respond the best to this treatment. Repeat IA injections may be required, depending on the severity of the abnormalities within the joint and the response to treatment.

Treatment of horses with secondary OA of the DIP joint usually focuses on the underlying contributing problem. Treatment of the primary condition is usually beneficial to prevent worsening of the problems within the DIP joint. For instance, extensor process fractures of the distal phalanx should be removed in most cases, and other articular distal phalanx fractures should be stabilized with corrective shoeing or internal fixation if necessary. SCLs should be debrided if possible, and horses with known trauma to the articular cartilage, subchondral bone, or CLs of the DIP joint should be treated appropriately until healing has occurred. Horses with navicular disease should be shod appropriately to minimize progression of the DIP joint OA. Many of these horses may also benefit from IA medication, depending on the underlying problem. For instance, horses with navicular disease usually benefit from IA treatment of the DIP joint by reducing the inflammatory response, both within the joint and within the navicular region.[45] A recent study documented that a clinically effective concentration of MPA or TA diffused between the DIP joint and navicular bursa after IA or intrabursal injection.[45]

Prognosis

Horses with synovitis/capsulitis of the DIP joint usually have a very good prognosis to return to performance if the predisposing hoof imbalances can be corrected and maintained. Recurrence is possible but is often related to relapses in the hoof imbalances. However, one study indicated a less optimistic prognosis with only 30% of horses responding to treatment.[16] The prognosis of horses with primary or secondary OA of the DIP joint is usually related to the severity of the radiographic or MRI abnormalities.[17,18] Horses with advanced OA often respond less to any form of treatment, or the lameness recurs more quickly. Horses with mild to moderate OA have a good to guarded prognosis to return to performance. Horses with secondary OA of the DIP joint have a variable prognosis depending on the underlying problem. However, the development of radiographic signs of OA within the DIP joint does not preclude athletic performance. For instance, several racehorses with type II fractures of the distal phalanx returned to racing despite radiographic evidence of OA within the DIP joint.[47] Some feel that the radiographic abnormalities within the DIP are overinterpreted as to their influence on lameness. Correlations between MRI abnormalities and the prognosis of horses with DIP OA have not been reported to the author's knowledge.

FRACTURES OF THE DISTAL PHALANX (P3, COFFIN BONE)

Fractures of the distal phalanx are an uncommon cause of lameness compared to the numerous other conditions that affect the horse's foot.[5,56] An older report identified 65 cases of P3 fractures in 20,638 cases admitted to a hospital.[56] These fractures can occur in any foot but most commonly affect the lateral aspect of the left forelimb and the medial aspect of the right forelimb in racehorses.[5,47] Type I and II "wing" fractures are most common and the majority of these fractures enter the DIP joint.[47,56] In one report of P3 fractures in Thoroughbred and Standardbred racehorses, 71 of 74 fractures were wing fractures (types I and II), and the majority of these fractures were articular.[47] Although all breeds and classes of horses can be affected, there appears to be a higher incidence observed in racing breeds.[5,47,56]

Although fractures of P3 can assume a variety of configurations, these fractures can be classified into seven types (Figure 4.28).[6] Type I fractures are nonarticular oblique palmar/plantar process (wing) fractures (Figure 4.29). Type II fractures are articular oblique

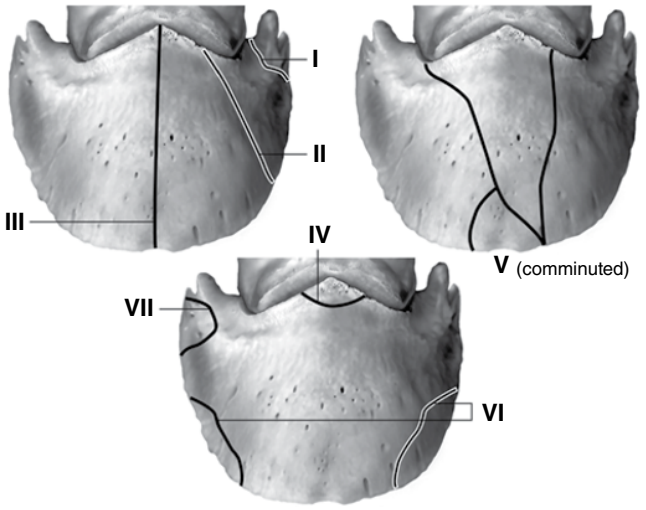


Figure 4.28. Classification of P3 fractures in horses. *Source:* Reprinted with permission from Dr. Alicia Bertone, Equine Fracture Repair, Nixon AL, ed. Reproduced with permission of John Wiley & Sons.



Figure 4.29. Type I fracture of the wing of P3. This fracture is nonarticular, but it may be difficult to document this. This particular fracture is larger than most type I fractures and may be a type II articular fracture.

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palmar or plantar process (wing) fractures (Figure 4.30) and are by far the most common type.[47,56] Type III fractures are sagittal articular fractures that roughly divide the distal phalanx into two separate halves (Figure 4.31). These fractures are uncommon and represent 3%–4% of fractures of the distal phalanx[30,47,56] and tend to occur more commonly in the hindlimb than the forelimb. Type IV fractures are articular fractures involving the extensor process. They occur most frequently in the forelimbs and can be bilateral (Figure 4.32).[11,15,30] Type V fractures are comminuted articular or nonarticular fractures and can be a variety of configurations (Figure 4.33). Type VI fractures are nonarticular solar margin fractures of the distal phalanx (Figure 4.34).[29] Type VII fractures are nonarticular fractures of the palmar or plantar process of the distal phalanx in foals (Figure 10.55). These fractures begin and end at the solar margin and are usually triangular or oblong in shape.[30,32] Initially they were thought to represent osseous bodies, but histologic findings were consistent with a fracture.[32] Frontal fractures of P3 (fracture line runs lateral to medial and splits the bone into dorsal and palmar/plantar halves) occur but are extremely rare.[1]



Figure 4.30. Type II articular "wing" fracture. This is the most common type of P3 fracture.

Etiology

Single-event trauma appears to be the predominant cause of P3 fractures. However, repetitive trauma leading the stress-related bone injury is likely the cause in racehorses.[47] Scintigraphic abnormalities of P3 have been identified in racehorses before radiographic evidence of a fracture was present.[34,47] This suggests that P3 fractures are similar to other "stress"- or "fatigue"-type fractures that occur in racehorses. Acute trauma is often the cause in nonracehorses, especially in those that involve the hindlimb.[3,6,49] Type III sagittal articular fractures usually result from direct trauma to the hoof from kicking a solid object.[56] Type VI solar margin fractures may be related to the shape and location of the solar margin within the hoof and the tremendous forces the distal phalanx undergoes during weight-bearing and work. These fractures may also occur concurrently with laminitis and pedal osteitis due to resorption of the apex of P3.[5] Type VII fractures in foals are thought to occur from compression either on the solar or dorsal cortex of the distal phalanx during weight-bearing or from tension forces generated by the DDFT and may be related to hoof conformation.[22,32] They are exclusively in the front feet, and in one study, 74% of foals radiographed had palmar process fractures.[23] Excessive trimming of the heels, sole, and frog in foals does not appear to affect the occurrence of type VII fractures in foals although these fractures appear to be associated with hoof conformation.[23] Occasionally, P3 may be fractured as a result of penetration of a foreign body through the sole. A large sidebone may also serve to predispose to type I or II fracture in the forelimb due to its lever arm effect on the palmar process (Figure 4.35).[56] An MRI study of horses

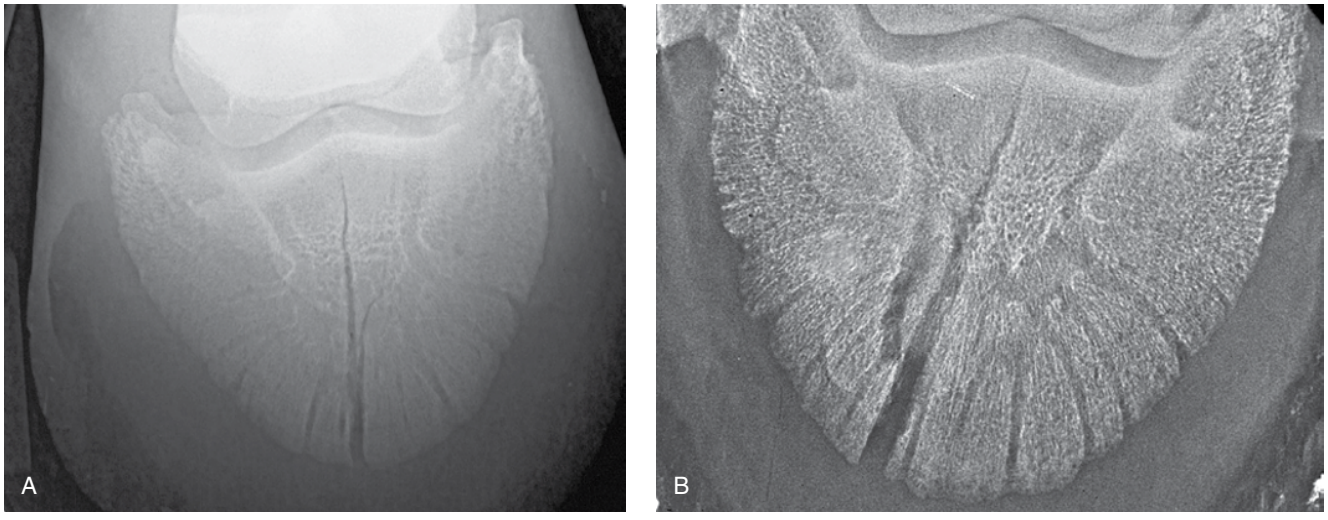


Figure 4.31. Acute (A) vs. more chronic (B) Type III sagittal fractures in two different horses. The fracture essentially splits the bone into two pieces, and the fracture gap usually widens with chronicity (B).

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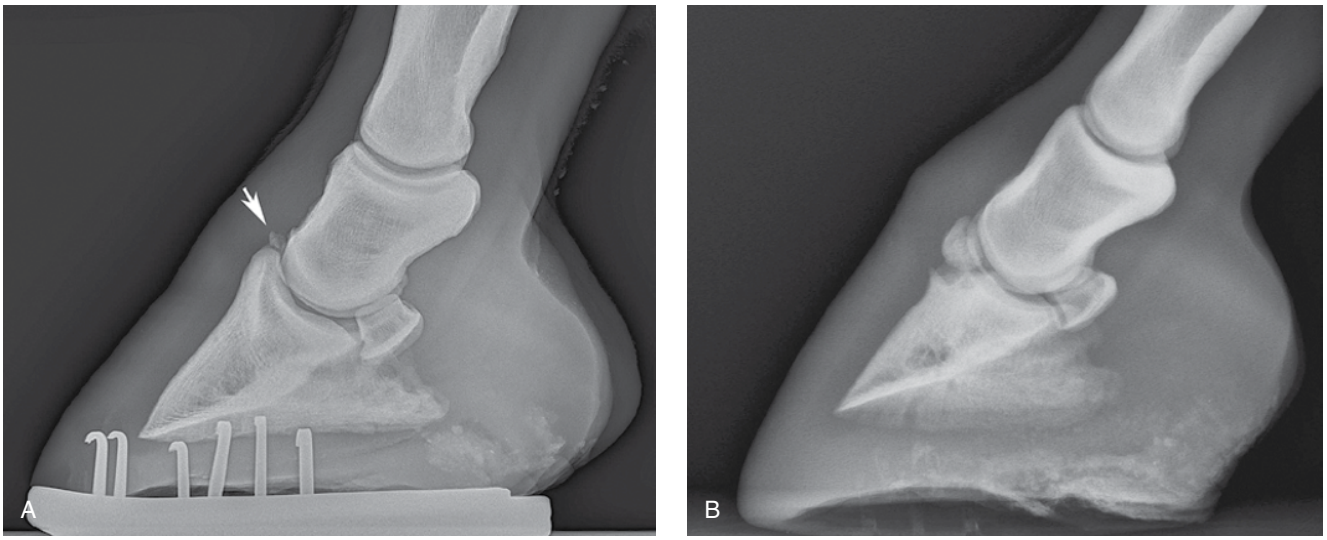


Figure 4.32. Small (A) and large (B) type IV P3 fractures involving the extensor process.

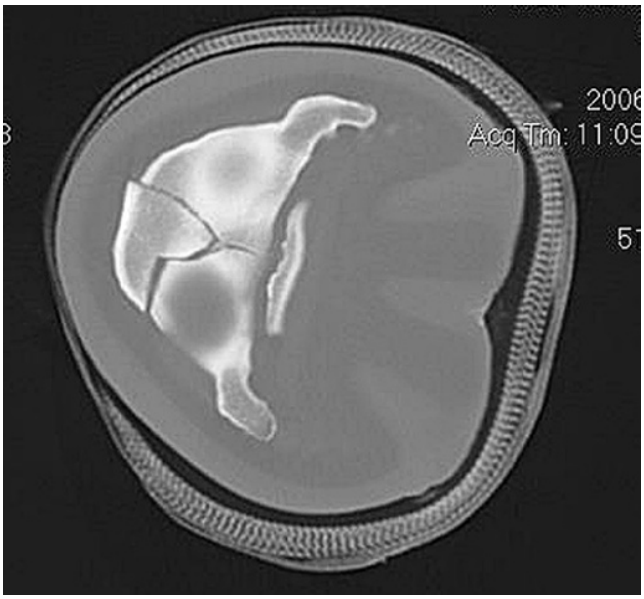


Figure 4.33. CT image of a type V comminuted fracture of P3. Fracture lines were present on both the lateral and dorsoplantar radiographs.



Figure 4.34. Type VI fractures are also referred to as solar margin fractures of P3 (arrow).

with ossified ungual cartilages and a P3 facture found that all fractures occurred at the base of the ossified cartilage suggesting a biomechanical cause or focal stress point from cycling.[57] The fractures involved only one of the lateral processes and were articular.

Type IV extensor process fractures may occur due to excessive tension on the common digital extensor tendon, resulting in an avulsion fracture. Overextension of the DIP joint may cause contact of the extensor process with the middle phalanx, and trauma to the dorsal aspect of the foot has also been proposed as a cause.[30,62] The development of a separate center of ossification or an osteochondrosis lesion of the extensor process may also occur.[30,56,62] Supporting a theory of a developmental lesion is the fact that lesions are often bilateral, occur in relatively young horses, and can be observed radiographically as an incidental finding in horses that are clinically normal (Figure 4.32).[9,15,30]

Clinical Signs

Generally the clinical signs are similar during the acute phases for all types of P3 fractures. A history of an acute onset of a moderate to severe lameness (grade 4–5 out of 5) is common.[5,6] In some cases the lameness will worsen within the first 24 hours after injury, presumably due to increased pressure within the hoof capsule secondary to inflammation and swelling. Exceptions to this are solar margin fractures, type VII fractures in foals,

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Figure 4.35. Type II articular fracture that was associated with a large sidebone of P3.

and developmental type IV fractures of the extensor process. Horses with these types of P3 fractures are usually only mild to moderately lame, and the lameness must be differentiated from the many other potential problems within the foot.[5,6] In all cases, if the fracture is chronic, the signs of lameness are usually diminished.

With acute fractures, an increased digital pulse may be palpable, and heat in the affected foot may be appreciated. With articular fractures DIP joint effusion is often palpable dorsal and proximal to the coronary band. Swelling and edema may also be present above the hoof wall in the pastern region. Hoof tester examination usually reveals pain over the sole region, and focal pressure over the fracture site usually induces a marked painful response. However, a negative hoof tester response does not rule out the presence of a P3 fracture, especially in chronic cases.[5] Perineural anesthesia of the PD digital nerves or IA anesthesia of the DIP joint may aid in localizing the lameness to the foot region. In general, regional anesthesia is usually unnecessary to diagnose type II and III fractures because the clinical signs are sufficient to localize the pain to the foot region. However, regional anesthesia is often necessary in horses with chronic P3 fractures and those that do not cause severe lameness.

Horses with large chronic extensor process fractures may have enlargement of the dorsal aspect of the coronary band and abnormal growth of the dorsal hoof wall. As the hoof grows, it develops a "V" or triangular shape called a buttress foot.[9,15] The abnormal hoof shape eventually will extend from the coronary band to the ground surface. Smaller type IV fractures rarely cause deformity of the dorsal hoof wall, but effusion of the DIP joint is common.

Diagnosis

Radiographic examination (30° dorsopalmar/plantar, 65° dorsoproximal to palmarodistal, lateral, and both obliques) is used to confirm the diagnosis and document the type and location of the fracture (see Chapter 3 for more details on radiographic views). In some cases it may be necessary to take special views of the palmar/plantar processes to identify the fracture. Solar margin fractures are most easily identified on the 60° dorsoproximal to palmarodistal projection using a radiographic technique with approximately one-half the exposure needed to evaluate the navicular bone.[30] Extensor process fractures are usually identified on the lateromedial view.

Most P3 fractures are readily apparent on routine radiographic projections. However, nondisplaced or stress-related fractures in racehorses may not be apparent on the initial radiographic examination because of insufficient time for resorption of the bone along the fracture line or because the cast-like effect of the hoof wall may prevent fracture displacement.[30,34] In these cases, radiographs should be repeated in 1–2 weeks, or nuclear scintigraphy, CT, or MRI can be used to help identify radiographically occult fractures of P3.[5,37,47] In one report, CT found an incomplete oblique fracture of the palmar process that was not evident radiographically 25 days after the injury.[37] CT can also be used in horses with comminuted P3 fractures to help determine the exact fracture configuration (Figure 4.33) and has become the standard to accurately determine fracture configuration.

Treatment

Options for treating horses with P3 fractures include confinement alone, confinement with corrective shoeing or foot casts, lag screw fixation (types II and III), and surgical removal of the fracture/fragment (type IV only). The decision often depends on the age and intended use of the horse, specific characteristics of the fracture, and financial constraints of the owner. In general, the majority of horses with P3 fractures are treated with confinement and corrective shoeing aimed at immobilizing the fracture and preventing expansion of the hoof wall (Figure 4.36). A foot cast or foot rim cast can serve the same purpose as the shoe and is preferred by some veterinarians. A recent study has questioned the need for immobilizing the foot because outcome did not appear to be influenced by whether the foot was immobilized in a series of 223 horses with P3 fractures.[49]

Types of shoes that may be used include a bar shoe with clips (Figure 4.36), a continuous rim-type shoe, or the Klimesh contiguous clip shoe.[1,5] All of these approaches appear to effectively prevent expansion of the hoof wall during weight-bearing, thus stabilizing the fracture. Regional anesthesia of the foot often aids application of the shoe and many horses walk more comfortably shortly after the foot is immobilized.

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In most cases the foot should remain in one of these shoes for 6–8 months, with the shoe reset every 4–6 weeks. Once clinical improvement has occurred, a less restrictive type of shoe (bar shoe only or bar shoe with quarter clips) may be used. Horse should not be worked for approximately 8–10 months, and in some cases, 1 year of rest may be necessary for clinical improvement.[5] Many P3 fractures are very slow to heal and may never develop radiographic bone union, but complete osseous union does not appear to be essential for horses to return to athletic activity.[49] In addition horses do not need to be shod with bar shoes and clips for the remainder of their athletic careers.[44]

Specific treatment of each type of P3 fracture is given below and included in Table 4.1.

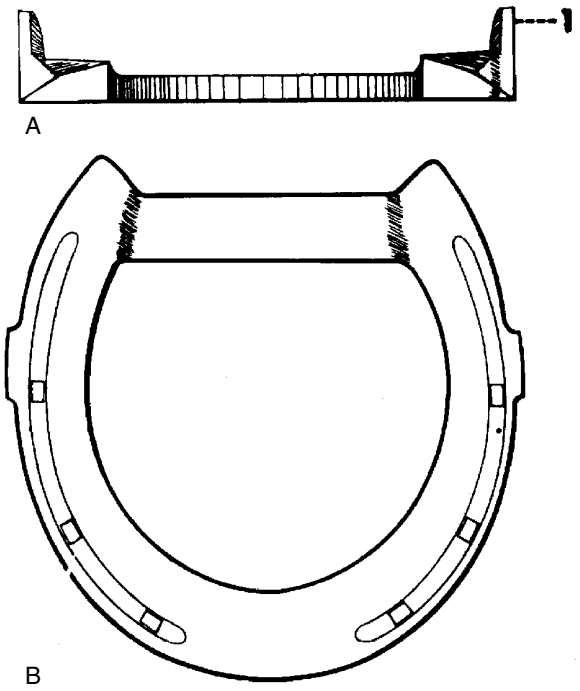


Figure 4.36. Full-bar shoe that can be used to treat horses with distal phalanx fractures. (A) Rear view of shoe showing quarter clips. (B) Ground surface view of the shoe showing full-bar and quarter clips welded to the shoe.

Type I

This nonarticular fracture is best treated with confinement and methods to prevent hoof expansion (shoe or foot cast). However, it may also respond to confinement and rest alone.

Type II

Foals less than 6 months of age should be treated with stall confinement.[60] Treatment that restricts the expansion of the hoof is usually unnecessary and may result in severe hoof contraction. Foals should be confined for 6–8 weeks, and their exercise should be restricted until bony union of the fracture is observed radiographically. Adult horses can be treated with confinement and methods to restrict hoof expansion or surgically by placing a lag screw. However, most type II fractures are treated nonsurgically because screw placement can be very difficult with this fracture type.[47] Surgery is usually only considered in horses with large wing fractures.

Type III

This is an unusual fracture in foals and adult horses and can be treated similarly to a type II fracture. These fractures tend to cause more severe lameness than type II fractures, and foot immobilization is often important to improve weight-bearing on the affected limb to prevent contralateral limb laminitis.

Table 4.1. Types of distal phalanx fractures.

Fracture type	Location	Articular	Recommended treatment	Prognosis
I	Palmar/plantar process	No	Confinement±shoeing (foot cast instead of shoe)	Very good to excellent
II	Oblique fractures of palmar/plantar process ("wing" fractures)	Yes	Confinement±shoeing (foot cast instead of shoe) Lag screw repair of large type II fractures	Fair to good
III	Midsagittal fracture	Yes	Confinement±shoeing (foot cast instead of shoe) Best candidate for lag screw repair	Unpredictable; Usually guarded
IV	Extensor process (variable size)	Yes	Removal in most cases regardless of size: arthroscopy/arthrotomy	Small: excellent Large: good
V	Comminuted	Yes or no	Confinement+shoeing (foot cast instead of shoe) Removal if secondary to infection	Dependent on fracture configuration
VI	Solar margin	No	Confinement+protective shoeing (wide-web shoes or shoes with full or rim pads)	Very good
VII	Palmar/plantar process; begins and ends at solar margin	No	Primarily in foals Confinement alone; no shoeing	Very good to excellent

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Acute type III fractures in adult horses are usually the best candidates for surgical repair using lag screw fixation (Figure 4.37).[3,6,24] Fractures of greater than a few days duration may fill with granulation/fibrous tissue, making it difficult to reduce the fracture. However, if conservative methods are not considered satisfactory, it is feasible to undertake screw fixation several weeks after the injury, even though the opportunity for interfragmentary compression is minimized.[25]

The correct site for screw placement is midway between the articular surface and solar canal through a hole in the side of the hoof wall.[24] Screw placement for type III fractures is usually less difficult than for type II fractures because the bone is essentially divided in half and there is less risk of splitting the fracture when the screw is tightened.[6,24] The major risks with this procedure are infection developing around the implant, the inability to compress the fracture, incorrect screw placement leading to continued lameness, and overriding of the fracture fragments during compression. The primary advantages are less risk of secondary OA developing in the DIP joint and faster healing of the fracture due to surgical compression.

Complete fracture healing can be expected in 6–12 months and the screw may have to be removed if lameness persists or infection around the implant is evident.[3,5,24] A computer-assisted surgery (CAS) technique has been developed to improve accurate screw insertion into sagittal P3 fractures.[2] This technique resulted in greater precision of screw length and placement compared with the conventional technique and may enable placement of two screws for improved compression. Improved accuracy of screw placement can also be accomplished by utilizing CT guidance intraoperatively.[24] Using larger diameter screws (6.5 or 5.5 mm) has been shown to increase axial compression and reduce the fracture gap of P3 fractures when compared with 4.5-mm screws in two separate *in vitro* studies.[31,33] Typically, 4.5-mm screws have been used because of the small space for screw insertion.



Figure 4.37. Lag screw repair of a type III P3 fracture. The screw must be placed through a hole within the hoof wall.

Type IV

Surgical removal of the fracture/fragment is usually the preferred treatment for type IV P3 fractures.[9,11,15,38] Acute extensor process fractures are rare in horses and are the best candidates for lag screw fixation, but this is rarely performed (Figure 4.38).[5,36] Conservative treatment with prolonged stall rest is often unsuccessful because the extensor process fracture does not heal and horses remain lame.[15,46] However, most large type IV fractures are chronic, and lag screw repair of these fractures is generally not recommended. Surgical removal of the extensor process fractures/fragments with arthroscopy or a dorsal arthrotomy is usually the treatment of choice (Figure 4.27). Arthroscopy using a dorsal approach is the preferred technique for removal of small extensor process fractures.[9,11] Large extensor process fractures (greater than 1 cm) have also been successfully removed with arthroscopy by using a motorized burr to help remove the fragment.[9,15,38] These fractures usually involve a large part of the extensor process (>25% of the joint surface) and can be challenging to remove (Figure 4.39).[9] The convalescence time after removing large fragments is generally longer than after removing smaller fragments.

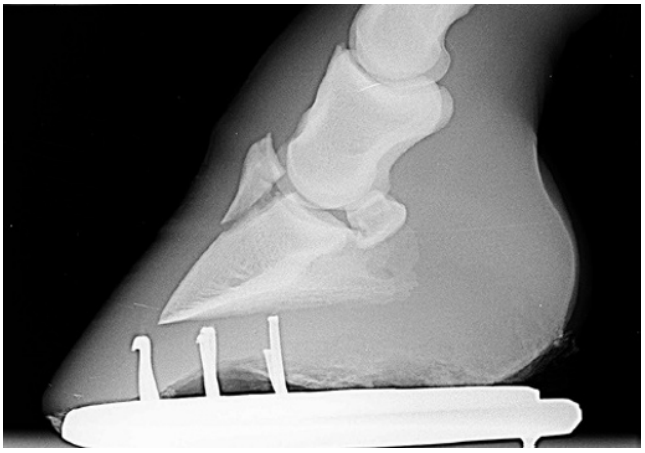


Figure 4.38. A large type IV P3 fracture that may warrant lag screw repair.

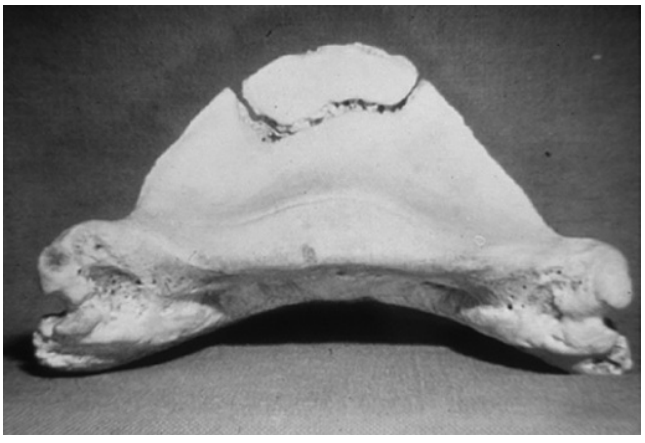


Figure 4.39. Dried bone specimen of a distal phalanx with a fracture of the extensor process. Note the width of the extensor process fragment.

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Type V

This fracture may be articular or nonarticular but regardless is best treated with confinement and methods to prevent hoof expansion (shoe or foot cast; see above for details). Confinement alone is usually not recommended for these fractures unless the fracture only involves the apex of P3.

Type VI

Treatment of solar margin fractures depends on whether the condition is primary or secondary to a chronic foot disorder (e.g. laminitis, osteitis, or infection). However, primary causes of solar margin fractures are usually treated with corrective shoeing (wide-web shoes, shoes and full pads, or shoes with rim pads) and stall or paddock rest for several months.[29] Strict immobilization with bar shoes and quarter clips is not necessary. Prolonged rest appears to be required for the best fracture healing, but this often depends on the size of the fracture. If the cause is secondary, then treatment is directed at the underlying cause initially, followed by management of the solar margin fracture.[29]

Type VII

Affected foals with this fracture are usually treated satisfactorily with confinement alone for 6–8 weeks. Exercise should be restricted until radiographic evidence of bony union is evident, which is usually observed at about 8 weeks after the diagnosis.[22,62] Application of restrictive external coaptation (e.g. bar shoe or acrylic) to the hoof is not recommended because of the severe heel contraction that can occur and the potential for the hoof to slough.

OA of the DIP joint is a common sequela to articular P3 fractures. All racehorses with articular wing fractures that had follow-up radiographs had evidence of OA on the radiographs.[47] However, this did not preclude horses from racing, and the authors warned to not overinterpret radiographic abnormalities within the DIP joint. Nonetheless, secondary abnormalities within the DIP joint subsequent to articular fractures may limit future athletic endeavors.[5] Secondary OA of the DIP joint appears to be more likely to develop with type III fractures than type II P3 fractures. If lameness persists, a neurectomy of the PD nerves may permit horses to resume athletic activity. In racehorses with primarily type II fractures, 18% had a PD neurectomy performed, which completely resolved the residual lameness and permitted them to race.[47]

Prognosis

In general, horses with hindlimb P3 fractures have a better prognosis than those in the forelimb, and the prognosis for nonarticular P3 fractures (types I, V, VI, and VII) is usually very good for all ages of horses if sufficient rest is given.[5,55] One recent study of 223 horses reported a 91% return to expected level of activity for horses with type I P3 fractures and 80% for horses with type VI fractures.[49] Foals with type VII P3 fractures usually have an excellent prognosis for return to performance, and fracture healing is expected in about 8 weeks.[32,60]

A range from 50% to 70% return to soundness has been reported for horses with type II wing fractures treated conservatively [44,49,56] However, a much better success rate has been reported in Standardbred racehorses with 81% returning to training and 63% racing.[43] However, 89% that returned to training without a bar shoe refractured at the same site, and 60% of horses returning to training with a bar shoe raced successfully.[43] This is in contrast to nonracehorses, of which 69% returned to their previous level of use and did not wear a bar shoe when they returned to training.[44]

The prognosis for small extensor process fractures treated by arthroscopic removal is generally considered to be very good to excellent. However, a recent study reported that only 46% of horses undergoing arthroscopic debridement of extensor process fragmentation remained sound after 4 years.[11] Removal of chronic large extensor process fragments also results in a good prognosis; 8 of 14 cases returned to their intended use in one report[15] and 14 or 17 in another.[9] The prognosis for large extensor process fractures treated by internal fixation also appears reasonably good in the small number of cases reported.[36,46]

The type of P3 fracture with the most variable prognosis is type III fractures. It remains unknown if affected horses have an improved prognosis with lag screw fixation compared with confinement and corrective shoeing.[3] Variable success of surgical treatment has been reported.[3,24] In one study all fractures healed, but only 2 of 4 horses returned to athletic activity and surgery did not reduce the convalescence time required. An older study using internal fixation reported that 11 of 11 horses older than 3 years of age became sound, and the most recent study that utilized CT guidance reported that 4 of 5 horses with sagittal/parasagittal fractures became sound.[24] In contrast, conservative management of horses with type III fractures was reported to have a 74% success rate for horses returning to their intended use.[49] Regardless of the treatment used, horses with type III fractures have a worse prognosis to return to performance than the other types of P3 fractures, and refracture of the bone is more likely to occur.[46]

SUBCHONDRAL CYSTIC LESIONS OF THE DISTAL PHALANX (P3)

SCLs of the distal phalanx are uncommon and can affect a wide variety of horse breeds of all ages.[4,60] Affected horses are usually intermittently lame, and the forelimbs are more frequently affected than the hindlimbs. Verschooten reported on 15 cases of SCLs involving the distal phalanx, 14 of which were located in the forelimb.[60] In another study 27 of 28 cases involved the forelimbs.[27] Most SCLs communicate with the joint surface,[28,59] although communication with the adjacent joint space can be variable.[60]

Etiology

Regardless of their location, the cause of most SCLs in the horse is considered to be either trauma or developmental.[4,48] Damage to the subchondral bone in the stifle has been shown to cause SCLs of the medial femoral condyle, and SCLs can occur at sites of previous IA

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fractures.[48] Damage to the cartilage and subchondral bone is thought to permit passage of joint fluid through the opening, resulting in resorption of the subchondral bone.[4,60] Alternatively, a developmental osteochondrosis lesion predisposes to the lesion because SCLs are often found in young horses and are frequently bilateral.[4,5] See Chapter 10 for more information about SCLs and osteochondrosis.

Clinical Signs

A history of an acute onset of lameness may be present, but more often the lameness is chronic and intermittent. The lameness may subside with rest and recur with exercise, and the severity can be variable. There are often no palpable abnormalities (including hoof tester examination), but effusion within the DIP joint may be present. The digital pulse rate may be elevated and the phalangeal flexion test is usually positive.[27] A PD nerve block improves the lameness in most cases. Intrasynovial anesthesia of the DIP joint also eliminates the lameness, especially if the SCL communicates with the joint.[28,60]

Diagnosis

Radiography is necessary for a definitive diagnosis of an SCL of the distal phalanx. The SCLs are variable in size and are usually identified within the body of the distal phalanx (Figure 4.40). The majority of SCLs communicate with the joint.[28,59] In one study, 18 of 27 SCLs were located centrally in the distal phalanx, and communication with the DIP joint was observed in all cases.[27] In a more recent study, all 11 horses had SCLs that were IA.[59]

Treatment

Recommended treatments of SCLs of the distal phalanx have included (1) confinement followed by increasing exercise, (2) IA medications, (3) transcortical drilling, (4) extra-articular surgical curettage, and (5) arthroscopic debridement.[28,58–60]

Most horses respond only transiently to IA medications, and debridement through hoof wall windows has been complicated by recurrent abscessation and lameness. A dorsal arthroscopic approach for IA debridement recently has been described in 11 horses.[59] This technique is performed with the DIP joint extended and distracted to permit access to the SCL.[59] Because of the morbidity associated with extra-articular debridement techniques, arthroscopic debridement should be considered the treatment of choice.[38,59] However, some SCLs in the distal phalanx may be inaccessible with the arthroscope.[28]

Prognosis

Variable results have been reported with extra-articular debridement of these lesions, and these techniques are often complicated by infection and continued lameness.[28] However, there are reports of successful debridement of SCLs through the hoof,[60] and 1 horse treated with an autogenous bone graft returned to race training after 6 months.[58] Ten of eleven (91%) horses treated with arthroscopic debridement of a distal phalanx SCL returned to athletic soundness.[59] All of these horses were treated between 16 and 33 months of age, so results may vary depending on the age at diagnosis.[59] However, arthroscopic debridement of SCLs of the distal phalanx should provide a superior outcome in any age horse compared with extra-articular approaches.

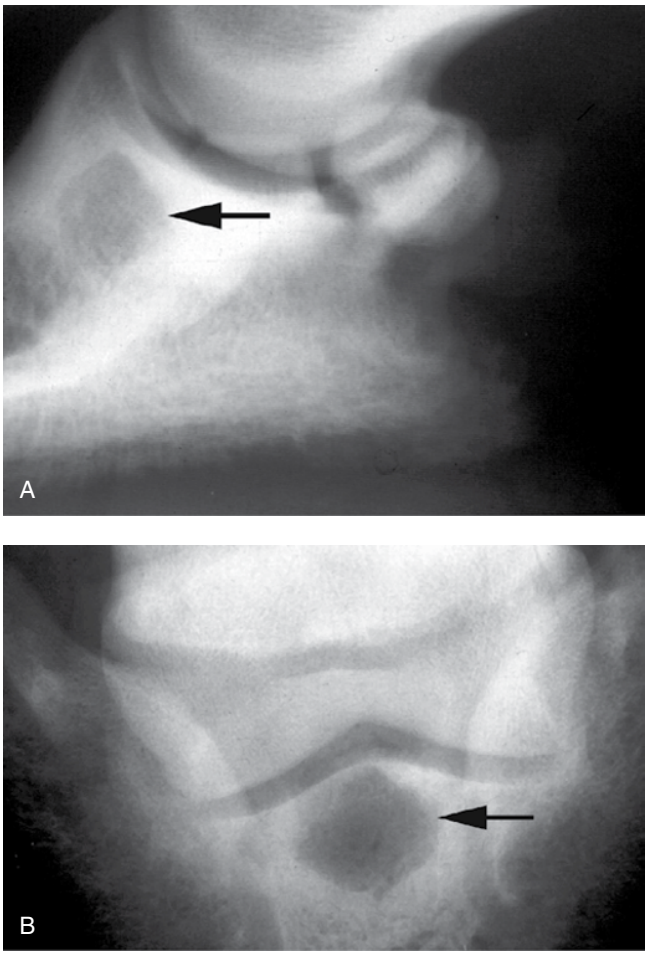


Figure 4.40. Lateral (A) and dorsopalmar (B) radiographs of a horse with SCL of P3 (arrows).

COLLATERAL LIGAMENT INJURIES OF THE DISTAL PHALANX

Desmitis of the CLs of the DIP joint is one of several soft tissue injuries that can occur within the foot. Lesions of the CL of the DIP joint were the second most common soft tissue injury in one MRI study, and they can occur alone or together with other injuries.[18–21] The medial CL of the forelimb is the most common site of injury, and although uncommon, they can also occur in the hindlimb.[19,26] These lesions may have concurrent osseous damage to the distal phalanx at the ligament insertion site (Figure 4.41).[12,13] Horses with extensively ossified collateral cartilages are also thought to be more prone to injuries of the CLs of the DIP joint.[21]

Etiology

Asymmetrical foot placement or foot imbalances may cause sliding and rotation of the distal phalanx relative

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to the middle phalanx, contributing to injuries to the CL of the DIP joint. This can occur as the result of an acute injury or from repetitive-type trauma. However, lesions within the CL of the DIP joint have recently been reported to be a primary degenerative process rather than inflammatory.[20] Histology revealed extensive fibrocartilaginous metaplasia and development of multiple intercommunicating fissures within the degenerate collagen in severe lesions.[20] This was thought to explain the poor response to conservative treatment in many horses with desmitis of the CL of the DIP joint. There are no known predisposing factors, but a recent study of 52 lame horses reported a correlation between a reduced palmar angle of the foot detected with radiography and alterations in the CLs of the DIP joint with MRI.[14]

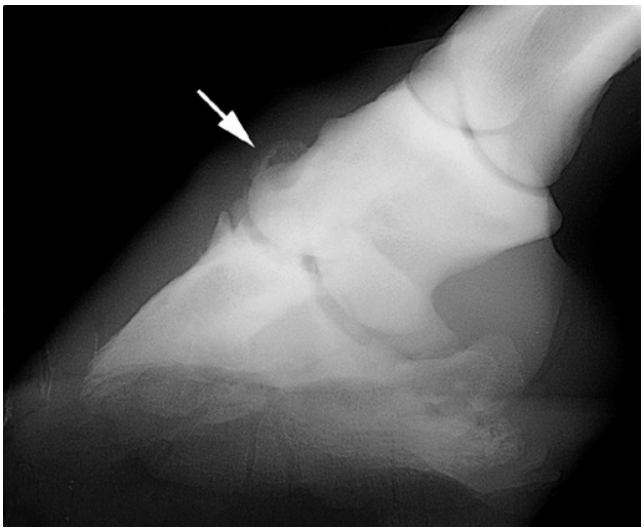


Figure 4.41. Bony proliferation on the dorsolateral aspect of P2 visible on this oblique radiograph (arrow) may be suggestive of a chronic injury to the CL of the DIP joint.

Clinical Signs

In general, horses that have primary soft tissue injuries in the foot such as injuries to the CLs of the DIP joint are more likely to have a history of an acute onset of lameness and be unilaterally lame compared with horses with injuries to the navicular region. There are often few localizing clinical signs in horses with injuries to the CL of the DIP joint.19, 26 Horses often have a history of a chronic forelimb lameness of variable severity that is worse in the circle. Palpable swelling and pain of the CL at its proximal attachment to the middle phalanx may be present above the coronary band in severe cases. However, this is uncommon and effusion of the DIP joint is also not a consistent clinical finding. Most horses (87% in one study) improve with a PD nerve block but may not be completely sound until a more proximal block is performed.19 Only 40% of horses improved with IA anesthesia of the DIP joint in one study.19

Diagnosis

A definitive diagnosis of an injury to the CL of the DIP joint is best determined with MRI in either the recumbent or standing patient. Lesions within the CL of the DIP joint are identified by the alteration in size and signal intensity (Figure 4.42). In addition, some horses may have abnormal mineralization and fluid within the distal phalanx at the insertion of the ligament[18] (see Chapter 3 for more information on MRI). Ultrasound may also be helpful, but certain aspects of the CL of the DIP joint are inaccessible with ultrasound, suggesting

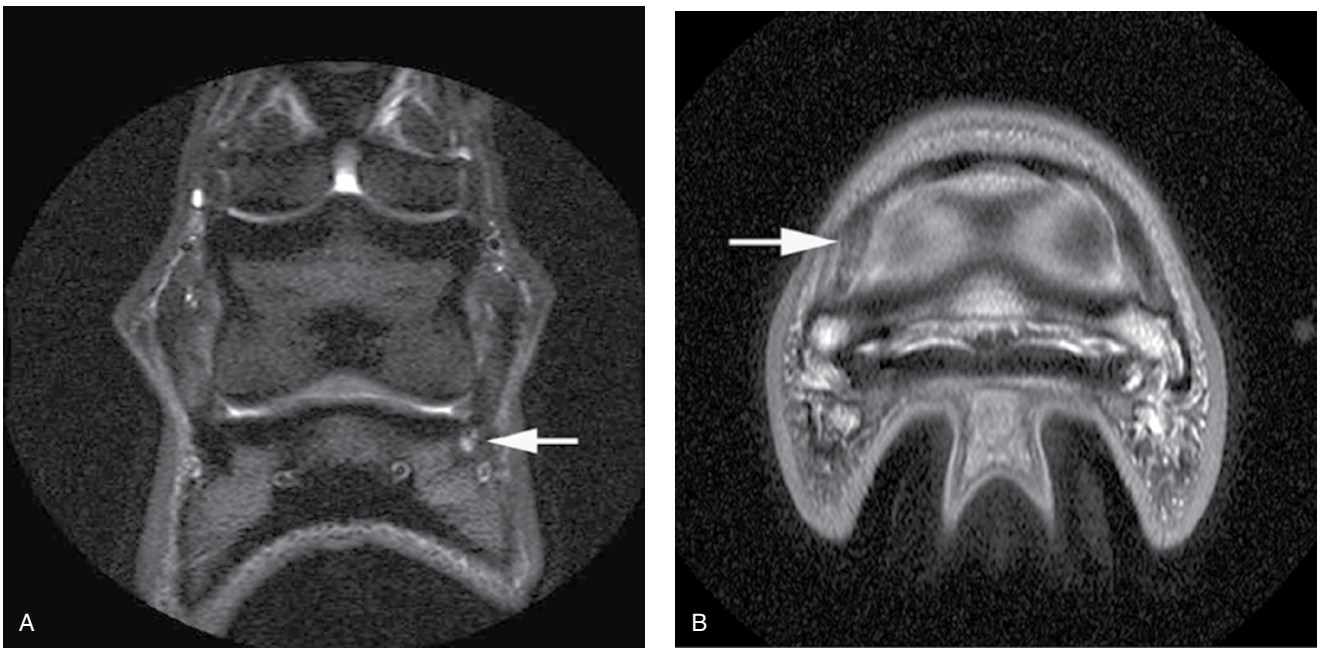


Figure 4.42. Frontal STIR (A) and PD axial (B) MR images of the foot of a 12-year-old Quarter horse with abnormalities within the medial collateral ligament of the DIP joint (arrows).

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that negative findings do not rule out the presence of a lesion.[19] Although concurrent osseous damage at the origin and insertion sites of the ligaments can be present in approximately 50% of these horses, scintigraphy is usually not recommended as an additional diagnostic tool because MRI can usually provide more specific information about the injury.

Treatment

The most important aspects of treating any soft tissue injury of the foot are rest, rehabilitation, and corrective foot care.[26,54] Foot imbalances that may have contributed to their occurrence such as low heels, long toes, mediolateral imbalance, reverse angle of the distal phalanx, etc. should be corrected if possible. For example, one study that correlated radiographic findings with MRI abnormalities found that a reduced palmar angle of the foot was correlated with alterations in the CLs of the DIP joint. Horses should be shod to decrease tension on the side of the injury by increasing the width to the branch of the shoe on the affected side and beveling the shoe on the opposite (unaffected side). Rest and rehabilitation and corrective shoeing are usually performed over a minimum of 6–8 months. Additional treatments for desmopathy of the CL of the DIP joint include extracorporeal shockwave therapy, application of a half-limb or foot cast, and medication of the DIP joint.[26] However, one large study found no benefit of using extracorporeal shockwave therapy or radial pressure wave therapy on outcome.[13] Ultrasound-guided intralesional therapy with stem cells, platelet-rich plasma, and other biological products may also be used, especially if the injury is accessible above the hoof wall (see Chapter 8 for more detailed information on these treatment modalities).

Prognosis

The reported prognosis for horses with desmopathy of the CL of the DIP joint is variable. One study reported that only 29% of horses treated with rest and rehabilitation returned to athletic function,[19] while another study indicated that 60% of treated horses (12 of 20) returned to their previous level of exercise.[26] Another study that followed horses for 2 years after presentation determined that only 27% of horses with CL injury alone and 34% of horses with CL-related osseous injury returned to their previous performance.[13] Horses with multiple soft tissue and osseous injuries within the hoof capsule did substantially worse. In general, these horses have a poor prognosis for future performance.

OSSIFICATION OF THE COLLATERAL CARTILAGES OF THE DISTAL PHALANX (SIDEBONE)

Ossification of the collateral cartilages of the distal phalanx is relatively common in certain breeds of horses, including most larger breeds such as Warmblood horses, draft horses, Finnhorses, and Brazilian jumpers.[40,51–53,61] The forefeet appear to be more commonly involved than the hindfeet, and the clinical significance of the condition remains questionable.[61] Female horses appear to be more susceptible to development of this condition, and the lateral cartilage often shows more ossification than the medial cartilage.[51] The ossification can begin at the base of the cartilage or originate as a separate area in the center of the cartilage.[8,40] In Brazilian jumpers, 7% of the horses had no ossification, 86% had ossification beginning at the base, and 7% had a separate center of ossification.[40] In either case, the palmar/plantar aspect of the cartilage is likely to be spared from the ossification process.[51]

In a radiographic study of 462 Finnhorses, 80% had evidence of sidebones, and large sidebones or separate centers of ossification were significantly more common in females than males.[53] Ossification of the cartilages in the forefeet was present in 93% of Brazilian jumpers.[40] Ten percent of Warmblood horses and 80% of draft horses had ossification of the cartilages of the distal phalanx, and the ossification was more extensive in draft horses than in Warmblood horses.[61] The lateral and medial cartilages were ossified equally in draft horses, but the lateral cartilages were more commonly involved in Warmbloods. In this same study it was concluded that ossification of the cartilage had no clinical significance.[61]

Etiology

The specific cause(s) of sidebones is(are) not clear.[53] It has been suggested that the tendency to develop sidebones is partly hereditary in certain horse breeds in Australia, Finland, and Sweden.[53,61] Hoof concussion causing trauma to the cartilage, poor conformation (particularly base narrow), and poor trimming and shoeing have also been proposed as inciting causes. One study documenting the incidence of sidebones in Finnhorses found that very few horses were base narrow and most horses were base wide and toed out.[53] Another study concluded that ossification of the cartilages was neither the cause nor the result of conformational adaptations of the front feet.[52] It has also been suggested that prolonged exercise and/or racing may have some preventative influence on ossification of the collateral cartilages. The amount of weight placed on the foot may also be contributory because primarily larger-breed horses develop sidebone.

Clinical Signs

Lameness resulting from sidebones is considered rare, and the clinical significance of radiographic apparent ossification is questioned.[5] One study found no correlation between the extent of ossification of the collateral cartilages and the onset of lameness.[61] Furthermore, despite the development of large sidebones in many Finnhorses, most horses performed satisfactorily without lameness.[53] However, large sidebones have been seen in horses associated with type I or II distal phalanx fractures by the author and were thought to contribute to the fracture and chronic lameness (Figures 4.35 and 4.43). A recent MRI study of horses with ossified ungual cartilages and a P3 facture found that all fractures occurred at the base of the ossified cartilage extending into the distal phalanx suggesting a biomechanical cause or focal stress point from cycling. However, these fractures were nonarticular and nondisplaced, but the ligaments and soft tissues adjacent to the ossified ungular cartilages were abnormal.[57]

In a scintigraphic study, a radiographically identified separate center of ossification could be detected with scintigraphy in 12 of 17 feet.[21,41,42] The authors suggested

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Figure 4.43. Standing dorsopalmar radiograph demonstrating a large uniaxial sidebone that was thought to contribute to lameness in this horse.

that scintigraphy may be useful to determine the clinical significance of ossification of the cartilages, prompting further clinical investigation such as uniaxial PD nerve block or advanced imaging.[41] However, another study by the same authors found considerable variation in the radiographic, scintigraphic, and MRI appearance of the palmar processes of the distal phalanx.[42] The study concluded that further investigation was needed to determine the clinical significance of the MRI abnormalities within the palmar processes. Currently, the clinical relevance of ossification of the collateral cartilages has not been documented but should be considered when clinical abnormalities associated with sidebones are found in lame horses.[5,21,42]

Sidebones may be visually apparent as an enlargement of the lateral and medial dimensions of the pastern region if the ossification is extensive. If the ossification involves the proximal extent of the cartilage, palpation may reveal an obvious firmness to the cartilage. Rarely is pain elicited with digital pressure. If present, the enlarged sidebone may contribute to the lameness or may be associated with a secondary fracture of the distal phalanx. A fractured sidebone reportedly causes more acute signs of lameness.[5] However, sidebones may accompany other lameness conditions of the palmar heel region (e.g. navicular syndrome) and may be mistaken for the cause. On the other hand, pain in the palmar heel region may originate from the axial projections of the ungual cartilages that extend toward the midline and have an abundant sensory nerve supply.[7]

Diagnosis

Radiographic examination of the foot usually reveals the extent of the ossification of the cartilage or cartilages (Figure 4.43). Occasionally a sidebone may appear fractured; however, the radiolucent defect must be differentiated from the junction between a separate ossification center and that part of the cartilage that is ossifying from the palmar process of the distal phalanx.[5,51] A fracture in the ossified cartilage can occur but is rare.

Documenting that sidebone is the cause of lameness in horses can be difficult. Asymmetrical swelling of pastern region, pain on palpation of the collateral cartilage, and improvement of the lameness with a uniaxial PD nerve block are suggestive of a problem in this region. However, the mere presence of an ossified cartilage without any other radiographic abnormalities is not diagnostic. The clinical relevance of these findings should be further documented with scintigraphy and/or MRI.[41,42]

Treatment

If sidebone is suspected as the cause of lameness, conservative treatment with rest, topical 1% diclofenac sodium cream (Surpass®), and oral administration of NSAIDs is recommended initially. External compression of the site together with cold-water therapy may also be beneficial. Any contributing foot problems such as foot imbalances should be addressed. Surgical removal of suspected fractured sidebones is not recommended. If lameness persists and sidebone is considered the cause of the lameness, a PD neurectomy can be performed but is usually unnecessary. Horses with sidebone and a secondary distal phalanx fracture are treated with corrective shoeing and confinement similar to a horse with a distal phalanx fracture alone (see type I and II P3 fractures).

Prognosis

The prognosis is difficult to predict because this condition is thought to rarely contribute to overt lameness.

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MISCELLANEOUS CONDITIONS OF THE FOOT

Gary M. Baxter

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SOLE BRUISES, CORNS, AND SUBSOLAR ABSCESSES

A bruise results from the rupture of blood vessels in the dermis (corium or sensitive tissue) beneath the sole, frog, or hoof wall. With time the hemorrhage spreads into the deep layers of the epidermis and becomes visible as the hoof grows. Accordingly, the discoloration associated with a sole bruise is most often seen several weeks after injury, whereas the same injury occurring in the hoof wall may take months before it becomes apparent.[27] Logically, bruises are most visible when the hemorrhage is superficial and the hoof is nonpigmented. It is likely the pain associated with the injury is due to the inflammatory response as well as the increased subsolar pressure.[31] If the bruised site becomes infected, a subsolar abscess is likely to develop.

A corn is a bruise that involves the tissues of the sole at the angle formed by the wall and the bar (Figure 4.44).[31] This site is often referred to as the seat of the corn. Corns occur most frequently on the inner angle of the front feet and are rarely found in the hindfeet.[31]

Etiology

Trauma to the sole is the cause of most sole bruising. However, sole bruising at the toe region may be secondary to an underlying condition such as chronic laminitis and flexural deformities.[27] Horses with flat feet, thin soles, and soft soles appear to be predisposed to sole bruising.[9] Also, horses that are barefoot, have their hooves trimmed too short, or have the sole protruding below the hoof wall appear more likely to develop sole bruising. Horses housed in muddy pens in freezing conditions, whether shod or not, often bruise their soles when the lumpy mud freezes hard.[31] Flat-footed horses that have repeated concussion to the sole adjacent to the white line because the sole contacts the inner aspect of the shoe may also develop sole bruises.[3] Any form of shoeing that concentrates weight-bearing on the sole is likely to cause bruising.

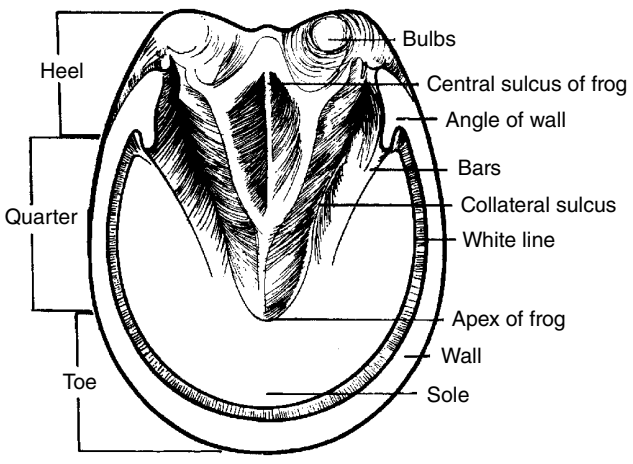


Figure 4.44. Normal forefoot showing anatomic structures.

Corns usually are caused by pressure from horseshoes or when a stone becomes wedged between the shoe and sole. They are rare among horses that are not shod. When shoes are left on too long, the heel may overgrow the shoe, causing selective pressure on the sole at the angle of the wall and the bar leading to a corn. Additionally, bending the inside branch of the shoe toward the frog to prevent pulling or stepping off the shoe can result in direct pressure to the sole, leading to bruising.[31] The application of a shoe that is one-half to one full size too small for the foot also increases the pressure on the sole area at the angle of the heels.[31] Heel calks usually enhance this effect.

Abscesses within the foot can develop from a variety of causes but are usually associated with bacteria entering a defect in the sole–wall junction, penetrating injuries into the foot, or occur secondary to another problem within the hoof (i.e. laminitis, keratoma, necrosis of the collateral cartilage, etc.). For example, "quittor" is the term used to describe necrosis and infection of the collateral cartilage of the distal phalanx characterized by multiple fistulous draining tracts proximal to the coronary band,[15,31] and "gravel" is the layman's term used to describe drainage proximal to the coronary band associated with ascending infection along the white line.[22,31]

Clinical Signs

The clinical signs associated with sole bruising or corns are often similar and variable. Most sole bruises occur at the toe or quarter regions and corns occur at the angle of the wall and bar. Occasionally the frog can be bruised as well.[25] The horse may show varying degrees of lameness (usually mild to moderate) depending upon the severity and type of the bruise or corn. The characteristics of the lameness and foot placement vary according to the location of the bruise or corn. If the bruise is acute or infected, the hoof may appear warmer and an increased digital pulse is often present.[25,31] Hoof testers often identify a focal site of pain unless the lesion is underneath the shoe at the white line. Perineural anesthesia may be required in some cases to exclude other sources of pain causing lameness.

Horses with foot abscesses are typically very lame and often non-weight bearing. Increased heat is often palpable in the foot and distal limb and an increased digital pulse is commonly found. Hoof tester pain is typically severe, and in some cases digital pressure at the site of the abscess causes a painful response. Increased swelling at the coronary band (especially at one heel bulb) may be present if the abscess has migrated up along the hoof wall. For example, ascending infection of the white line occurs when an opening at the sole–wall

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junction that permits infection to invade the laminae, which follows the path of least resistance to break open and drain at the coronary band. However, the condition may go undiagnosed until drainage at the coronary band is observed. Signs of lameness may also vary depending on the severity and location of the infection.

Diagnosis

A tentative diagnosis can often be made based on the history and clinical signs. If pain is localized to the foot but there are no obvious external abnormalities, the shoe should be removed and the sole explored by removing the exfoliating (flaky) sole with a hoof knife. Acute sole bruises may not be readily apparent because the hemorrhage has not migrated far enough distally. Chronic bruises are usually visible as a stippled reddened region.[31] In some cases the discoloration may be bluish, especially if a sole abscess is developing. Sole abscesses may have a small defect in the sole where the abscess is trying to break through the sole. Alternatively, a large-bore needle can be inserted through the soft sole to confirm the presence of purulent material beneath (Figure 4.45). Removing a small area of sole around this defect may also reveal purulent material, confirming a subsolar abscess. Hoof tester pressure at the site usually causes purulent material to exit the sole defect. Focal swellings at the coronary band (dorsally and at the heels bulbs) may suggest ascending infections along the white line even when no defects can be detected on the solar surface of the foot. In these cases, a definitive diagnosis often is not made until the abscess breaks out at the coronary band.

Acute sole bruises may not be evident radiographically unless a serum pocket or abscess has developed. Many subsolar abscesses may be seen radiographically as a gas pocket within the sole (Figure 4.46). However, many are not visible, depending on the radiographic projections, and the lack of radiographic abnormalities does not rule out an abscess. Chronic sole bruising may be associated with demineralization, increased vascular channels, and irregularity of the solar margin of the distal phalanx.[27] Chronic abscessation may be due to an underlying condition such as laminitis or keratoma and may contribute to osteolysis of the distal phalanx and sequestrum formation.



Figure 4.45. This horse was non-weight-bearing lame with no evidence of a hoof abscess or fracture. Focal pain was detected near the lateral heel and insertion of a 16-gauge needle confirmed the suspicion of an abscess.



Figure 4.46. This horse with a type II P3 fracture became acutely lame 2 months after diagnosis. A dorsopalmar radiograph demonstrated a fluid pocket (arrow) consistent with an abscess.

Treatment

Many bruises often resolve without treatment if the source of the trauma is removed. The horse should be rested from heavy work, especially if the soles are abnormally thin. When possible, the environment should be changed so that the horse is not worked on rough ground. If the horse must be used, the sole can be protected with a full pad applied under the shoe. The pad should be placed to avoid pressure to the bruised site. Wide-web shoes may also be beneficial to relieve pressure on the sole. Light paring of the sole overlying the bruise often relieves the pressure and makes the horse more comfortable.[25]

Drainage is the key to treating suppurative bruises and other subsolar abscesses. A small amount of sole overlying the abscess should be removed to permit ventral drainage. Removing a large amount of sole should be avoided because this is usually not necessary for drainage and prolongs the healing time. The foot can then be soaked in antiseptic solution if desired, and the foot bandaged. Once the abscess has resolved, the sole can be protected with protective boots or shoes until the defect has completely keratinized. If an ascending infection of the white line is suspected but cannot be confirmed (no drainage at the coronary band), soaking or poulticing the foot may draw the infection to the surface. When the abscess comes to a "head" just proximal to the coronary band, drainage can be established by lancing the abscess.[3,25,31]

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In cases in which shoeing contributes to the bruising, removal of the shoe may be all that is necessary. The horse should be rested and should not be reshod until symptoms disappear. If the horse must be used, the wall and bar in the region of the corn should be removed to prevent pressure by the shoe. A complete support shoe can be applied to allow the frog to absorb the concussion that would normally be distributed to the corn area. Another option is to apply a wide-web shoe that has been concaved out at the damaged region to decrease the pressure on the bruised site. To prevent shoes from causing corns, the heels of the shoes should extend well back on the buttresses and should fit full on the wall at the quarters and heels.[31]

Prognosis

The prognosis is usually very good for horses suffering from a single traumatic episode and in those with good foot conformation. The prognosis is reduced in horses with poor hoof conformation that are continually worked on hard ground because recurrence is common. Many of these cases develop chronic sole bruising, which eventually may lead to chronic abnormalities within the distal phalanx. Horses with routine foot abscesses also have a very good prognosis, provided the infection does not involve deeper structures in the foot. It should always be remembered that subsolar abscesses may be associated with other conditions of the foot such as keratomas, infection of the collateral cartilages, chronic laminitis, and septic pedal osteitis (PO) (Figure 4.47).

SEPTIC PEDAL OSTEITIS

Septic PO refers to bacterial infection within the distal phalanx. It can occur as an extension of a subsolar abscess, secondary to penetrating injuries or from hematogenous spread in foals. Radiographically the condition usually appears as a focal or diffuse radiolucency of the bone (osteolysis).[6,9] The distal phalanx does not have a medullary cavity, so inflammation/infection of this bone is referred to as osteitis rather than osteomyelitis.[11]



Figure 4.47. This horse had what was thought to be a routine abscess at the toe (arrow), but a lateral radiograph revealed chronic laminitis.

Etiology

Environmental microbes gain access to the distal phalanx either by direct introduction of bacteria by deep penetrating injuries, extension of infection from the soft tissues of the foot into the distal phalanx, or from hematogenous sources in foals.[23,31] Septic PO in foals should be considered as a potential site for hematogenous spread of infection associated with the septic arthritis/joint ill syndrome.[24] One study found that 8 of 22 foals with septic PO had multiple other foci of infection and none of the foals had a history of a penetrating wound or subsolar abscess.[23] Other causes of septic PO in adult horses include chronic severe laminitis, subsolar abscesses (most common), solar margin fractures, deep hoof wall cracks, avulsion hoof injuries, and penetrating wounds of the foot.[6,11,19] A sequestrum may develop in the distal phalanx as the osseous infection progresses.[6,16] In a review of 63 horses treated for septic PO, subsolar abscesses were the most common cause (56%) followed by solar margin fractures (25%) and penetrating wounds (13%).[19]

Clinical Signs

Septic PO occurs most commonly in the forelimbs in adult horses and in the hindlimbs in foals.[6,23] The forelimbs were affected twice as commonly as the hindlimbs in one study,[7] and 11 of 18 cases involved the forelimbs in another study.[6] The severity of lameness in horses with septic PO is usually severe (in one study over 50% of the horses were grade 4 of 5 lame), and the lameness may be chronic (average duration of lameness prior to presentation in one study was 18.5 days).[6,19] Increased hoof temperature and prominent digital pulses are common in the affected limb. Hoof tester exam may be beneficial to localize the site of pain as well as to promote abscess drainage in some cases. Perineural anesthesia of the PD nerves may not eliminate the lameness in all horses with septic PO. No or minimal response to PD nerve block was seen in 5 of 5 cases of septic PO, and all 5 cases improved after an abaxial sesamoid nerve block.[6]

Diagnosis

Radiographic signs suggestive of septic PO are usually straightforward and are consistent with areas of bone infection (Figure 4.48). CT examination of the foot can often provide more detailed information but is usually unnecessary to make a diagnosis. Generally, there is a loss of trabecular detail (osteolysis) with indistinct margins fading into the surrounding bone.[31] Sequestra may develop but marginal sclerosis is rarely observed in these cases (Figure 4.49).[19] Radiographic examination of 18 horses diagnosed with septic PO revealed discrete osteolysis at the margins of the distal phalanx (13 of 18 cases), gas density adjacent to the bone on 2 different radiographic projections (15 of 15 cases), focal (9 cases) or diffuse (3 cases) decrease in bone density, generalized roughening of the solar margin of the distal phalanx (7 of 18 cases), and widening of the vascular channels (13 of 18 cases).[6] Sequestra were identified in 4 horses.[6] In foals with septic PO, evidence of localized lysis or focal loss of bone density was observed

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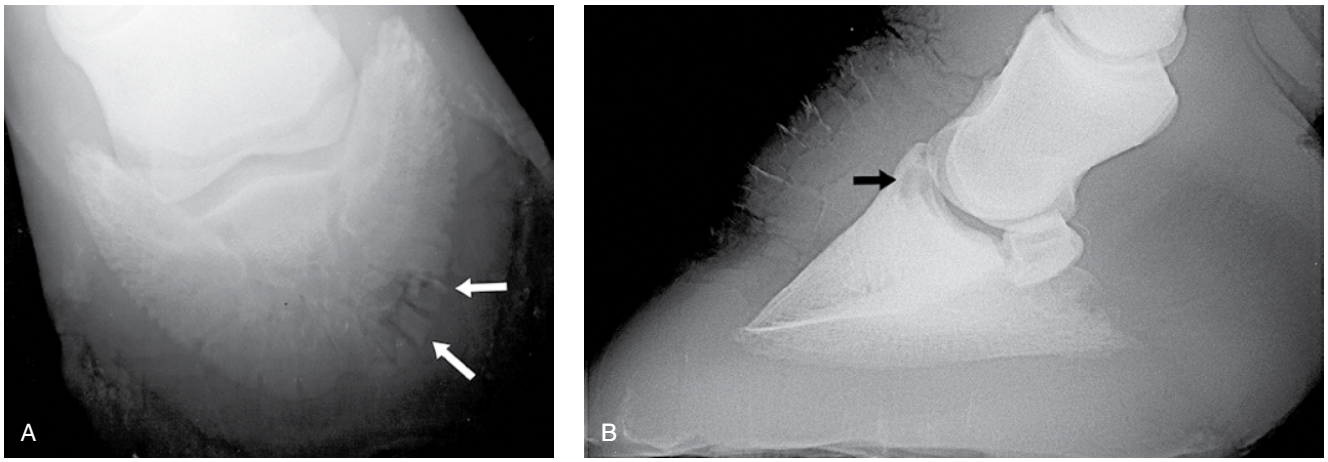


Figure 4.48. Dorsopalmar (A) and lateral radiographs (B) of the feet in two adult horses with severe lameness due to infection of the distal phalanx. Osteolysis of the solar surface (A; arrow) and extensor process (B; arrow) of the distal phalanx is evident consistent with infection.

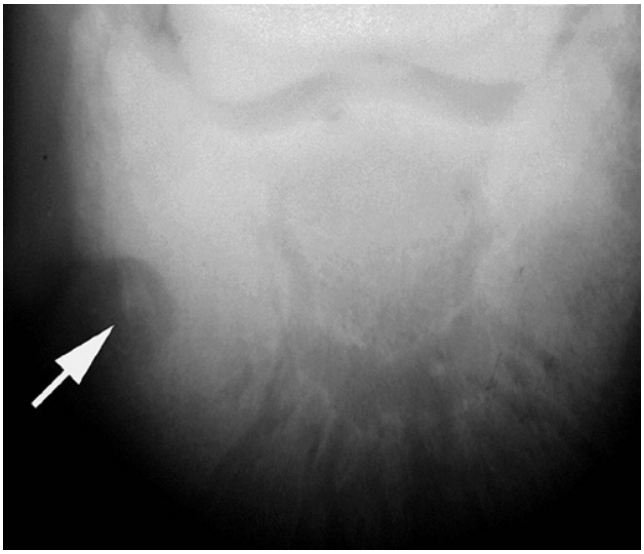


Figure 4.49. Dorsopalmar radiograph of a horse with a chronic draining tract from the solar surface of the foot. Lysis and a sequestrum can be seen along the solar surface of P3 (arrow).



Figure 4.50. The sequestrum in image 4.49 was removed through the solar surface of the foot.

at the toe (14 of 22 cases), extensor process (5 of 22 cases), or the palmar/plantar process (3 of 22 cases) of the distal phalanx.[23]

Treatment

Treatment of septic PO usually involves systemic and local antimicrobials (regional limb perfusion) and surgical debridement of the infected bone.[6,7,9,11,23] Surgical debridement may not be indicated in all cases depending on the location within the bone (i.e. extensor process in foals).[23] Debridement can be performed with the horse standing if the solar surface of the distal phalanx is involved (Figure 4.50). However, general anesthesia is often necessary to permit accurate location of the lesion, especially if a draining tract is not present, and for thorough debridement. In most cases the site of infection is approached through the draining tract, which is usually located on the sole. The abnormal bone is debrided and a sample submitted for culture and sensitivity. Regional IV limb perfusions can be repeated as needed after surgery to help treat the infection (see Chapter 12 on musculoskeletal emergencies for more details). One study reported that up to 24% of the distal phalanx could be removed without long-term adverse effects.[11]

Prognosis

The prognosis for horses with septic PO appears very good to excellent if the infection can be controlled.[7,31]

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Of 33 cases of septic PO in which follow-up was available, all 33 horses returned to their intended use.[19] In other studies, 7 of 9 horses returned to their intended use,[11] and 13 of 18 had a favorable outcome despite a high incidence of sequestra and fractures.[6] Foals with septic PO also have a good prognosis; 86% of the foals survived, 16 of 22 foals reached racing age, and 11 of these raced.[23,24]

PENETRATING INJURIES OF THE FOOT

Penetrating injuries of the foot are commonly seen in equine practice. The injury is often sustained by the horse stepping on (bottom of the foot) or contacting (coronary band, heel bulbs, and pastern region) a sharp object. Although any deep penetrating injury to the foot can potentially be serious, those that penetrate the central third of the frog, the coronary band, and the heel bulbs are at greatest risk to involve deeper vital structures. Structures at risk with injuries to the central third of the frog include the navicular bursa, navicular bone, DDFT, or distal phalanx. Coronary band injuries may involve the DIP joint, distal phalanx, and collateral cartilages of the distal phalanx. Injuries to the heel bulb region are most likely to damage the digital flexor tendon sheath, DDFT, or palmar/plantar aspect of the DIP joint. Injuries elsewhere on the bottom of the foot most likely only damage the digital cushion or the solar surface of the distal phalanx and are usually less problematic. Early identification of the involvement of a deeper vital structure and aggressive medical and surgical treatment greatly affect the outcome of horses with these injuries.[2,3,9,29] See Chapter 12 for further information about treating septic synovial structures.

Clinical Signs

The clinical signs caused by penetrating injuries may vary depending on the depth (superficial vs. deep), location (sole vs. coronary band), and duration (acute vs. chronic) of the injury.[3] In general, the more superficial the injury, the less severe the clinical signs. Horses with minor injuries to the sole that may remain asymptomatic for several days until a subsolar abscess develops are the exception. These types of injuries penetrate the cornified layer of the sole into the digital cushion but do not cause clinical signs unless an abscess develops. Deep injuries that penetrate completely through the hoof wall and contact a bone, tendon, or synovial cavity typically cause severe and acute lameness. Also, horses with wounds in the frog region that involve vital structures usually become rapidly symptomatic.

Affected horses often point the foot and resist contacting the heel (walk on the toe) when walked.[3,32] Palpation of the hoof in any horse with a penetrating injury may reveal increased heat, and a prominent digital pulse can usually be palpated.[2] Careful examination of the sole (visual, hoof tester, and probing) and coronary band is important. It has been stated that any penetrating wound of the foot deeper than 1 cm should be considered serious (Figure 4.51). The approximate reported depths of a perpendicular penetration before vital structures become involved were 1 cm for the sole, 1.5 cm for the frog, and 1.2 cm for the hoof wall.[3]



Figure 4.51. This horse had a history of stepping on a farriery nail with its hindfoot 24 hours previously. Exploration of the tract revealed that it had penetrated through the digital cushion to the solar surface of the third phalanx.

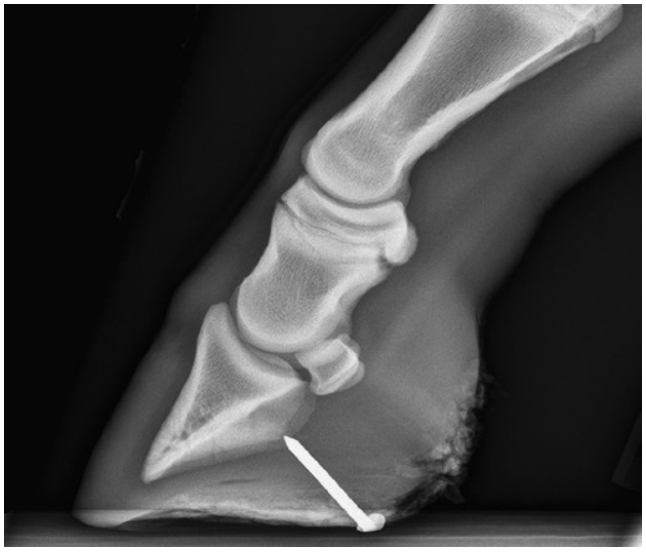


Figure 4.52. Taking radiographs of the foot prior to removing the foreign body can help identify the direction and depth of the puncture.

If a foreign body such as a nail is present in the bottom of the foot, it is ideal to take a radiograph to determine the exact depth and direction of the nail's path before removing it (Figure 4.52).[1,29] However, this is not always possible, and care should be taken to avoid deeper penetration of the nail during the process. If a wound is not obvious, careful application of hoof testers may help identify focal pain, which may indicate the site of penetration. Once a focal site of pain is found in the sole or frog, it is explored with a hoof knife. If the injury is acute (before infection) and involves the sole, a crack or small hole may be the only abnormality found.[3] In most cases, puncture wounds of the sole appear black at

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the entry site. With more chronic penetrating injuries, small areas of granulation tissue may be evident on the sole (Figure 4.53).

Wounds that penetrate the frog can be particularly difficult to locate because the softer and more elastic tissues of the frog tend to collapse and fill in the tract. Careful removal of the frog is often required to visualize the entry site. Probing of the tract can help identify both the depth and direction of the injury. A radiograph can be taken with the probe placed into the tract to further verify its location (Figure 4.51). If infection is present, gentle pressure with the thumbs or hoof testers around the entry hole may cause purulent exudate to exit the tract.[3] Perineural anesthesia is usually not needed to localize the site of lameness but is very beneficial to facilitate close examination of the injury site and removal of the frog or sole if needed.

Palpation of the coronary band for heat, pain, and swelling may also be helpful to identify the location of a penetrating wound to this region. A penetrating wound of the coronary band can be overlooked if the hair is long or if local swelling and wound drainage are not present. Once identified, wounds at the coronary band should be carefully probed and explored because they may contain foreign material such as wood.

Heat, pain, and swelling of one heel bulb are often seen with migration of a subsolar abscess. Effusion of the digital tendon sheath or DIP joint may suggest infectious synovitis.[1,14,29] Synovial fluid analysis can usually be used to confirm the diagnosis. An increased white blood cell count (more than 30,000) with neutrophilia, a pH below 6.9, and an increased protein (more than 4.0 g/dL) are highly suggestive of a septic process.[1,3,29]

Diagnosis

Additional diagnostics that can be performed to confirm the location and depth of a penetrating injury include distension of a synovial cavity with saline to detect leakage from the wound, plain radiographs, radiographs with a metallic probe inserted in the wound, contrast radiography (fistulogram), or ultrasound.[1,29] Plain radiographs may reveal the presence of gas that can be seen with a subsolar abscess or penetration of a synovial cavity. Concurrent fractures and osteolysis due to infection may also be visible. Placement of a metallic probe confirms depth and direction of the injury (Figure 4.51), and contrast radiography often confirms penetration of a synovial cavity (Figure 4.54). Ultrasound may be helpful to document injuries to the DDFT and involvement of the digital flexor tendon sheath. Cross-sectional imaging (CT and MRI) can also be very helpful to confirm both the location and the presence of infection especially when other diagnostics are equivocal (Figure 4.55).

Treatment

Treatment of superficial penetrating wounds that do not involve vital structures (bone, tendon, or synovial cavities) is generally uncomplicated. Treatment is aimed at providing adequate drainage, removing infected and necrotic tissue, and protecting the site from further contamination.[25,31] The majority of cases can be treated in the standing sedated horse using the help of perineural anesthesia. Drainage is established by removing a small amount of adjacent sole or frog with a sharp hoof knife (loop hoof knives work well) and/or a hoof groover. Underlying necrotic/infected tissue should be removed with a standard curette, hoof curette, or nail hole curette.[31] An antiseptic dressing is applied and the foot is protected to minimize further contamination. More extensive superficial infections may require periodic flushing or soaking of the foot together with bandaging and foot protection.



Figure 4.53. Chronic penetrating injuries to the foot may be identified by small areas of granulation tissue on the sole (white arrow).



Figure 4.54. Contrast radiography can be helpful to document synovial involvement in horses with chronic penetrating injuries of the frog and sole. The puncture in this horse had completely healed, but the horse developed an abscess just above the heel bulb. Contrast injected into the abscess communicated with the navicular bursa.

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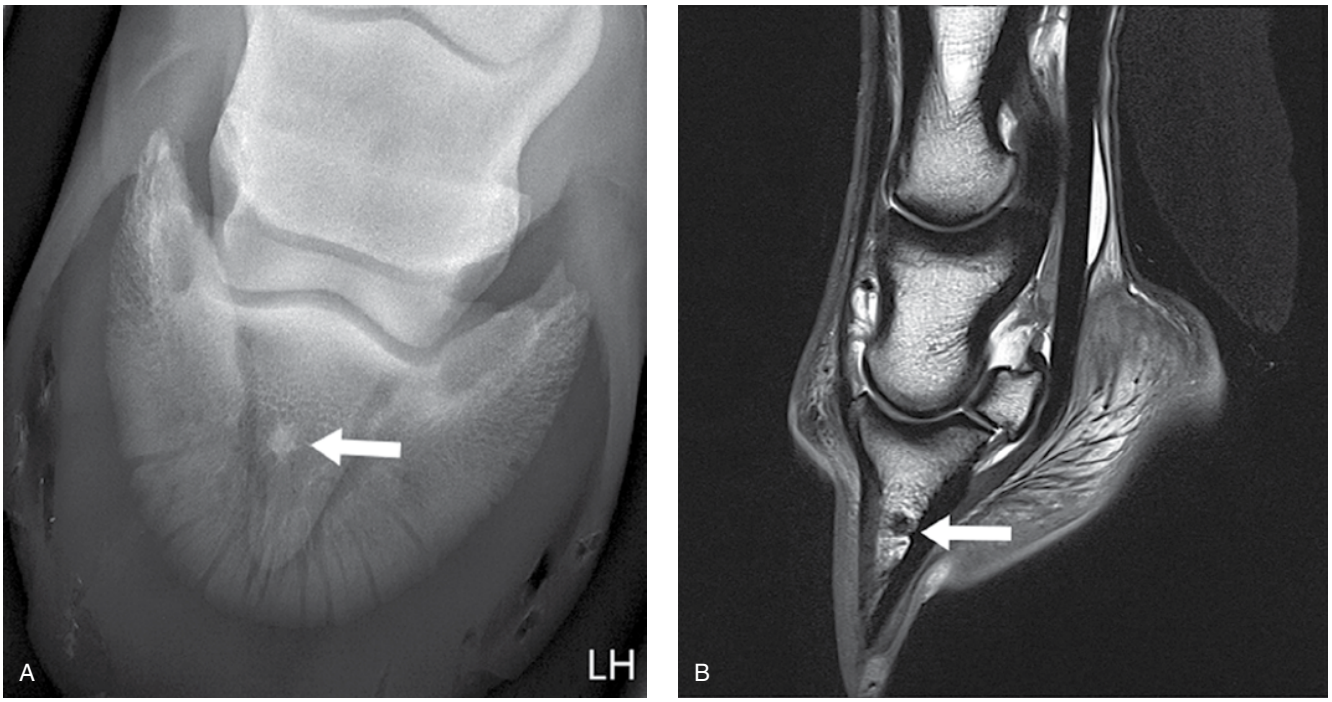


Figure 4.55. Dorsopalmar radiograph of the distal phalanx of a lame horse revealed a focal radiodense area in the center of the distal phalanx (left; arrow) that was confirmed with MRI (right; arrow). The lesion was later identified as a well-circumscribed P3 sequestrum.

Penetrating injuries that involve bone, tendon, or synovial cavities require more aggressive treatment depending on the deeper structure that is involved. Typical treatments include both systemic (IV) and local (IV regional perfusion and intrasynovial) antimicrobials, NSAIDs, local debridement of the wound, and lavage, endoscopy, or arthroscopy if a synovial cavity is involved. Wound debridement may be performed in the standing patient, but it usually best performed with the horse under general anesthesia. Lavage, endoscopy, or arthroscopy of a synovial cavity usually requires general anesthesia. Wounds that penetrate the distal phalanx should be enlarged and the distal phalanx curetted if possible as further progression of the infection into the bone is not uncommon (Figure 4.56). Soaking the foot to lavage deep wounds of the foot is generally not recommended. Involvement of the navicular bursa is best treated with endoscopy or lavage and local debridement of the defect in the frog.[1,2,33] See the section on musculoskeletal emergencies in Chapter 12 for further information.

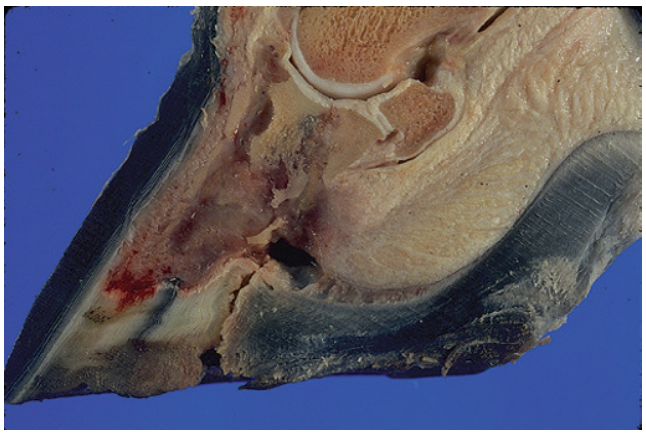


Figure 4.56. Sagittal section of P3 in a horse that sustained a puncture wound to the solar surface of P3. Subsequent infection of P3 contributed to a pathologic fracture of the bone and involvement of the DIP joint.

Prognosis

Horses with penetrating injuries that do not involve bone, tendon, or a synovial cavity typically do very well.[18] Horses with penetrating injuries outside the frog or frog sulci and centrally located in the foot also do well. A recent study reported that 91% of horses with penetrating injuries of the foot without synovial involvement and treated conservatively returned to their previous level of soundness.[18] In another study of 50 horses with deep puncture wounds of the foot, 95% of horses with injuries outside the frog or frog sulci regained full athletic soundness.[32] Horses with synovial involvement following solar foot penetration have a poor prognosis (36% and 29% in two recent studies) for return to preinjury athletic function.[10,18] In general, prompt treatment of any penetrating injury to the foot regardless of the structure(s) involved improves the chance of a successful outcome.[2,18]

KERATOMA

Keratoma is an uncommon condition of the hoof that is characterized by keratin-containing tissue growing between the hoof wall and the distal phalanx. Although the term implies a neoplastic process, histologic

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examination reveals abundant keratin, squamous epithelial cells, occasionally granulation tissue, and inflammatory cells.[13,20] The growth usually begins near the coronary band, but it may extend to the solar surface anywhere along the white line.[31] A visible deviation of the coronary band and/or hoof wall is often present, and the most commonly affected areas of the foot are the toe and quarter.[20] Occasionally a keratoma may be located at a focal site between the coronary band and sole. Lameness and the radiographic changes are thought to arise from the growth of the keratoma and the subsequent pressure that is applied to the sensitive lamina and distal phalanx.[20] Keratomas have been observed in horses ranging from 2 to 20 years of age and should be differentiated from other growths that can occur in the hoof such as squamous cell carcinoma, canker, and melanoma.[20,31] In addition, multiple keratomas may be present in the same foot, but this is uncommon.[8,17]

Etiology

Trauma and chronic irritation in the form of sole abscesses or direct hoof injuries are the cause in the majority of cases.[20,31] However, a keratoma can develop without a history of previous injury, and the initiating cause often cannot be determined.[17,31]

Clinical Signs

A history of a slow onset of intermittent lameness is common. The lameness is often seen before the distortion at the coronary band and hoof wall becomes obvious. Moderate to severe lameness is commonly observed at presentation.[17,20,31] The coronary band and hoof wall may or may not be abnormally shaped, and close examination of the foot may be required to identify any abnormality. In some cases a fistulous tract may develop in the sole or hoof wall, mimicking a subsolar abscess.[5,16] Common clinical signs of keratomas in one retrospective study were lameness and the presence of a subsolar abscess.[5] Hoof tester examination often elicits a painful response when pressure is applied over the lesion. Although perineural anesthesia of the PD nerves at or below the level of the collateral cartilages often improves the lameness, a basisesamoid or abaxial sesamoid block may be required to completely eliminate the lameness.

Diagnosis

A definitive diagnosis of keratoma is usually made based on the characteristic radiographic features. A discrete semicircular defect in the distal phalanx is often seen (Figure 4.57).[5,31] However, the absence of a discrete radiolucency cannot be used to rule out the presence of a keratoma.[17,20] The radiographic signs of a keratoma can usually be differentiated from lysis due to infection because of the smooth borders and lack of a sclerotic margin. Ultrasonographic imaging of a keratoma has been reported, and a hypoechoic, well-delineated soft tissue mass between the hoof wall and the articulation of the distal and middle phalanges was seen.[30] However, cross-sectional imaging (CT or MRI) is currently the preferred technique to both definitively diagnose and accurately localize keratomas within the hoof wall



Figure 4.57. Dorsopalmar radiograph of P3 demonstrating a smooth margined lytic defect within the bone that is characteristic of a keratoma. *Source*: Courtesy of Dr. Scott Katzman.

(Figure 4.58).[12,17] The use of CT to accurately localize keratomas was recently reported to minimize the amount of hoof wall that needed to be removed and reduced postoperative complications in operated horses.[17]

Treatment

Treatment usually involves complete surgical removal of the abnormal growth. In one study only 42% of horses treated without surgery (12 horses) returned to performance compared to 83% that were treated with surgery (23 horses).[4] Incomplete removal of the keratoma is thought to result in recurrence of the growth.[17,20,31] Surgery may be performed with the patient under general anesthesia or while the patient is standing using regional anesthesia and sedation.

Partial hoof wall resection directly over the location of the keratoma is the preferred technique. Using CT to localize the keratoma preoperatively was recently shown to improve the accuracy of hoof wall removal and facilitate complete removal.[17] Windows within the hoof wall can be made with a motorized burr, a cast cutting saw, oscillating saw, or an osteotome (Figure 4.59). Alternatively, the hole in the hoof wall can be made with a large Galt trephine. Multiple trephinations can be performed to adequately expose the lesion. The major advantage to this approach is the relative lack of disruption of the hoof wall.[16,17] Regardless of the technique, the overall goal of surgery should be to remove as little hoof wall as possible to facilitate complete removal of the keratoma.[5,16,17]

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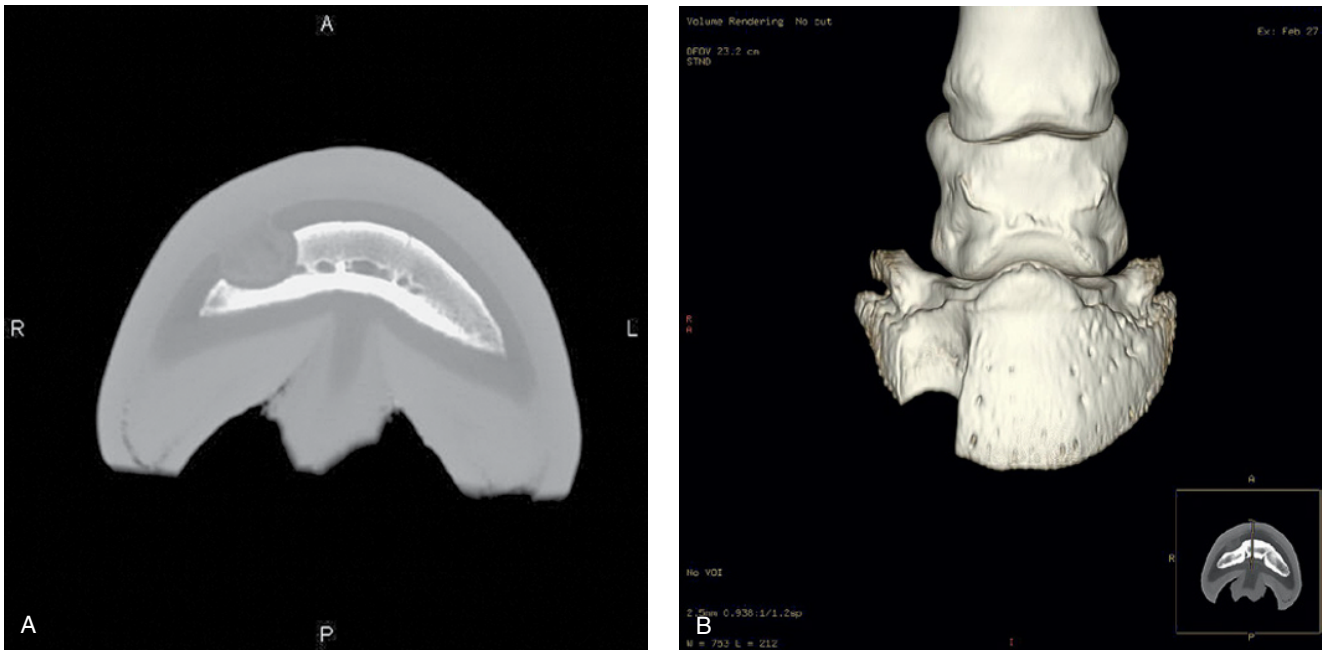


Figure 4.58. Single CT image of a foot (A) demonstrating a defect within the distal phalanx consistent with a keratoma and CT reconstruction of the CT images (B) that illustrates the exact location of the keratoma within the distal phalanx. *Source*: Courtesy of Dr. Scott Katzman.

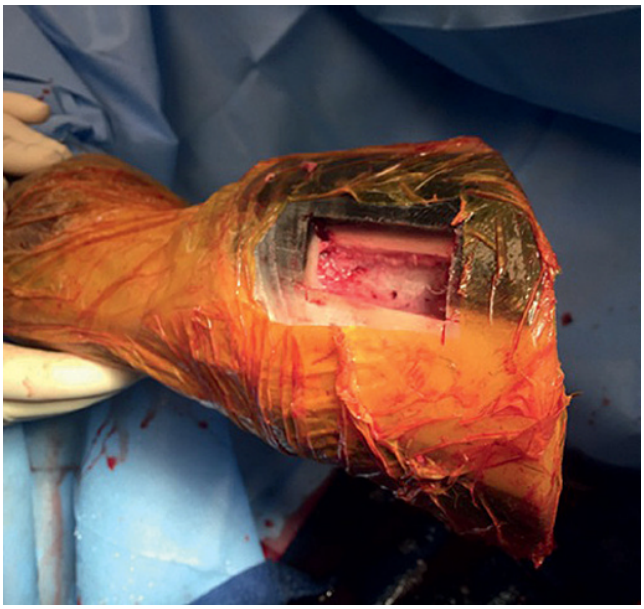


Figure 4.59. Hoof wall removal directly over the keratoma that was located with CT. *Source:* Courtesy of Dr. Scott Katzman.

Prognosis

The prognosis is generally very good for return to performance if the abnormal tissue is completely removed.[4,5,12,17] Using cross-sectional imaging (CT or MRI) is thought to facilitate more accurate localization of the keratoma, enabling a smaller hoof wall resection and an improved prognosis.[12,17] Postoperative complications may be decreased by creating smaller hoof defects, filling the defects with antimicrobial-impregnated polymethylmethacrylate, and placing a shoe early in the postoperative period.[12] In one large study, 25 of 26 horses returned to their previous exercise level,[5] and the most recent study indicated that 28 of 31 horses returned to their previous activity level.[17] Complications occurred in only 4 of 31 (13%) horses.[17] Adequate stabilization of the hoof defect and complete removal of the lesion are important for a successful outcome.[5,12,17]

AVULSION INJURIES OF THE HOOF

Avulsion injuries of the foot region are relatively uncommon in horses but can be very serious and difficult to manage. The injuries can be categorized as complete (when the tissue is totally removed) or partial or incomplete (when the tissue remains intact on at least one border).[26,31] These injuries may involve the hoof wall, coronary band, pastern region, sole, and structures deep to the hoof capsule (e.g. distal phalanx, digital cushion, laminae, synovial cavities). In general, the seriousness of the injury is related to the depth of the avulsion and the amount of tissue that is removed. However, even with significant loss of germinal tissue, the foot has the capacity to heal, although slower than other tissues, with complete reformation of hoof wall structures if treated properly for a long enough period.[26,27,31] The foot region has limited ability for wound contraction; therefore, the wound heals primarily by epithelialization and reformation of the corium.[28,31] These processes require a healthy bed of granulation tissue and a stable, clean environment to prevent permanent hoof defects.

Complete or incomplete avulsion injuries of the coronary region may result in deformities and/or permanent hoof wall defects if not treated properly (Figure 4.60). However, if treated promptly and correctly, the majority heal without problems.[21,28,31] In addition, partial loss of the distal phalanx, digital cushion, or collateral cartilages does not appear to reduce the prognosis of affected

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Figure 4.60. Abnormal coronary band and hoof growth associated with a previous injury to the medial aspect of the coronary band.

horses for future soundness. However, secondary infection of deeper hoof tissues usually reduces the chances of a complete recovery. The time required for healing depends on the size and extent of the avulsion injury and the method of treatment. Generally, 3–5 months are needed for second intention healing of complete avulsion injuries, compared with 3–4 weeks for incomplete avulsions that are surgically repaired.[21,28]

Etiology

Incomplete avulsion of the hoof wall of the heel can be caused by vertical tears of the hoof wall, kicking or stepping on sharp objects, continued foot imbalance, and improper shoe removal in which nails are torn out of the heel and quarter regions.[28,31] Other avulsion injuries of the foot and pastern are usually caused by lacerations from sharp objects. The horse either steps on or kicks at a sharp object, or the foot becomes entrapped, resulting in the avulsion. These are commonly seen as heel bulb lacerations that often involve the hoof.

Clinical Signs and Diagnosis

The degree of lameness usually varies with the duration, extent, and location of the avulsion injury. Moderate lameness is usually seen with an acute superficial injury that does not involve deeper structures. More extensive avulsion injuries usually cause severe lameness. Gentle manipulation of the foot and phalanges can provide important information regarding the status of support structures. Involvement of deeper structures such as the DIP joint, navicular bursa, and the digital tendon sheath should be identified.[3]

For chronic avulsion injuries, varying degrees of lameness may be present. If the wound heals without problems, lameness usually subsides with time. However, if lameness and purulent exudate persist, further diagnostics such as probing the wound with a sterile probe, radiography, contrast radiography, and ultrasonography should be performed to determine the cause of the continued drainage and lameness.[3] A chronic nonhealing wound with drainage usually suggests continued infection of deeper structures or involvement of a synovial cavity.

Treatment

Incomplete Avulsion (Coronary Band Not Involved)

Incomplete avulsions of the hoof wall at the heel and quarter without involvement of the coronary band are usually best removed.[28] Attempts to salvage the hoof wall are often unrewarding and contribute to continued lameness and infection beneath the avulsed hoof wall (Figure 4.61). Removal also prevents the wall from being snagged and continually traumatized, which is often very painful.[3]

The hoof wall can be removed with the horse standing for limited involvement or under general anesthesia for larger lesions. Sharp hoof knives, nippers, and a handheld electric drill (Dremel tool) to burr the hoof wall at its attachments may be used for removal.[28,31] The dorsal attachment of the unaffected hoof wall should be beveled flush to the wound so there is little tendency for it to be snagged, resulting in further separation. The wound should be bandaged and protected from contamination and trauma until the exposed tissues become keratinized. A full-support shoe can be used to provide hoof wall stability and to reduce weight-bearing on the hoof defect by "floating" the involved heel.

Incomplete Avulsion (Coronary Band Involved)

Incomplete avulsion injuries of the coronary band alone or the coronary band and hoof wall are best managed by suturing the wound whenever possible. Reapposition of the coronary band is important to prevent future hoof deformities.[26,27] Often the hoof wall cannot be salvaged and will require removal, but the coronary band should be reconstructed in any way possible (Figure 4.62). Lacerations of the coronary band without loss of hoof wall should be sutured primarily and immobilized in a foot cast (Figure 4.63). Avulsion injuries that affect the coronary band and a small portion of the hoof wall can also be sutured and immobilized. The hoof wall adjacent to the defect can be thinned with a hoof rasp, and the separated piece of hoof wall can be thinned with a motorized burr to permit suturing.[28,31]

When the avulsion injury extends from the solar surface proximally through the coronary band, the majority of the hoof wall can be removed to within 1 cm of the coronary region, and the coronary band and soft tissues sutured if possible. Alternatively, the hoof wall can be included in the closure by thinning the walls adjacent to the defect with a Dremel tool. Regardless of the technique, accurate approximation of the coronary band is important. If left untreated, these incomplete avulsion injuries of the coronary band often remain elevated, eventually producing a horny spur at the distal extremity of the avulsion, while the remaining underlying tissue heals by scarring and epithelialization.[26,28,31] Invariably these avulsions protrude above the skin and hoof wall surface, making them susceptible to further trauma and painful to palpation. If the avulsed tissue is just removed, a permanent hoof wall defect will often develop (Figure 4.60).

Complete Avulsion

With complete avulsion injuries of the hoof, there is usually no tissue to appose and the wound and hoof defect heal by second intention (Figure 4.64). The initial

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Figure 4.61. Before (A) and after removal (B) of an incomplete hoof avulsion of the medial quarter of the hindfoot.



Figure 4.62. Before (A) and after (B) reconstruction of an incomplete hoof avulsion that involved the coronary band, sole, and frog. Realignment of the coronary band should be performed whenever possible.

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Figure 4.63. A sharp laceration of the coronary band (A) was debrided and closed primarily (B) to prevent aberrant hoof wall growth.

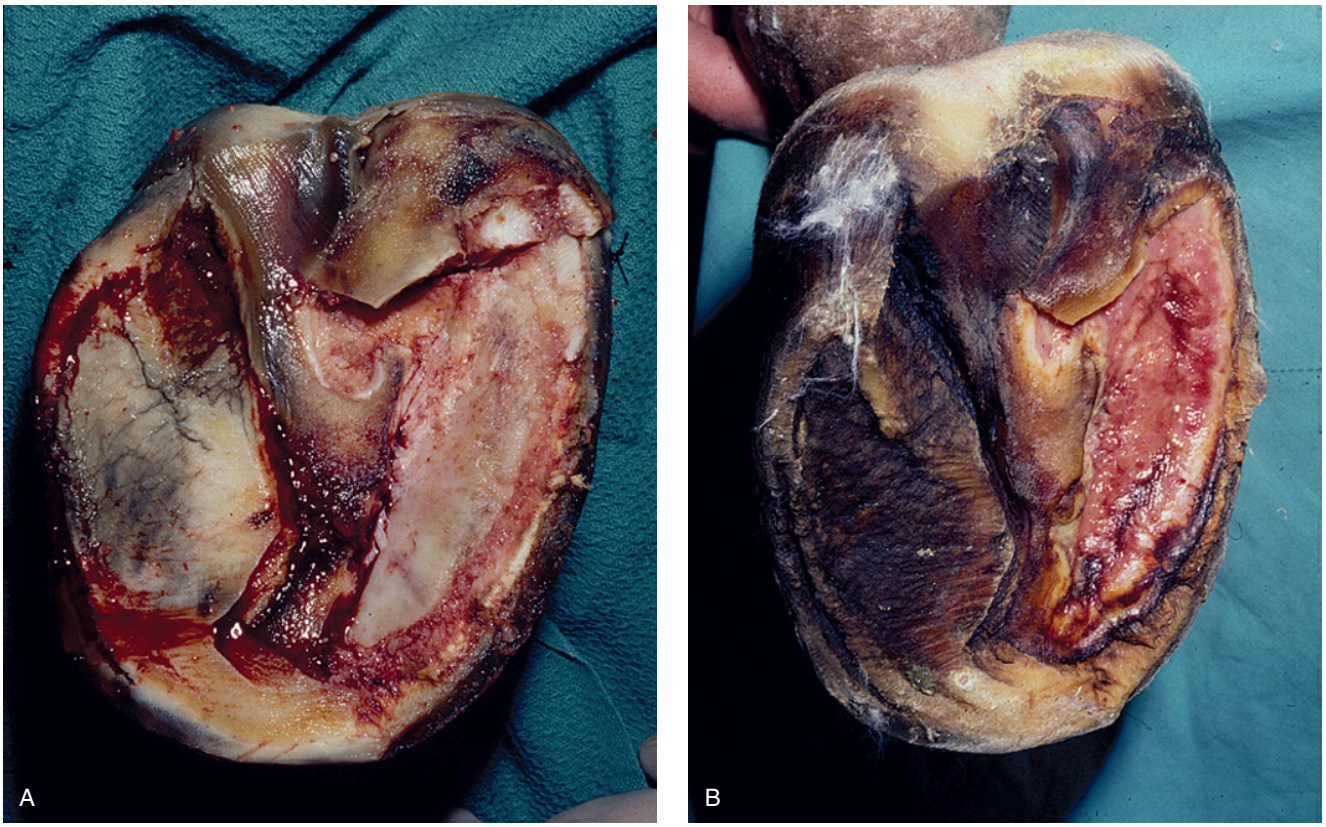


Figure 4.64. Complete avulsion of the sole with exposure of the solar aspect of P3 (A) and the same defect after it has filled with granulation tissue (B).

clinical picture is usually that of a hemorrhagic wound with obvious tissue loss.[26] The depth of the defect and potential involvement of other structures should be determined prior to treatment because their involvement affects both the type of treatment recommended and the prognosis. The practical treatment of these wounds in terms of bandaging, the use of systemic and local antimicrobials, and local wound care follow the same guidelines used to treat other limb wounds in horses.[26] Many of these wounds can be bandaged initially to control

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hemorrhage and permit repeated lavage and debridement of the wound. However, immobilization with a foot cast is often very beneficial in the long-term management of these injuries. Casts are usually left in place for 3–4 weeks and may need to be reapplied depending on the size and location of the avulsion. Wound stability is thought to improve the chances of complete reformation of the hoof wall.[31]

Hoof avulsions heal by similar processes to other open wounds, but healing is often protracted because wound contraction is limited in the foot. Wounds must heal be epithelialization and reformation of the corium, which often requires 3–5 months, depending on the size of the defect.[28] There are several types of germinal epithelial tissues in the foot (skin, limbic, coronary, parietal, and solar), and all can contribute to epithelialization of the defect.[26,27] The structure and quality of the hoof that forms is related to the type of epidermis that migrates over the surface of the wound.[26] Epithelial cells that migrate aberrantly can lead to hoof deformities.[26,27]

For instance, if epidermis from the parietal integument grows into the space formerly occupied by the coronary band, the wall generated in that location will not resemble wall generated from the coronary integument. If epidermis from the hoof migrates proximally to the coronary band, a horny spur will form in the pastern region.[26] One of the goals of treating complete hoof avulsion injuries is to prevent aberrant migration of epithelial cells and hoof wall deformities (Figure 4.60).

Prognosis

Generally, incomplete avulsion injuries of the hoof wall alone and/or including the coronary band have a very good functional and cosmetic outcome if they are sutured.[21,28,31] The prognosis for deeper avulsion injuries is often difficult to predict until complete healing has occurred. Complications such as fracture of the distal phalanx, osteomyelitis, septic arthritis, and OA of a damaged DIP or PIP joint obviously reduce the prognosis for a sound horse. Chronic hoof deformities are one of the most common sequelae of hoof avulsion injuries but do not always cause a clinical problem.[27]

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LAMINITIS

James Belknap, Andy Parks, and Katy Dern

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OVERVIEW

The disease generally termed "laminitis" likely exists solely due to the unique mechanical demands placed upon the equine epidermal cell in the digital lamellae, namely, the suspension of the distal phalanx (and thus the entire weight of the horse) within the hoof wall. These lamellar epidermal cells are not only responsible for adhesion of the hoof wall to the underlying connective tissue of the basement membrane and dermis/corium (which attaches to the distal phalanx); it is now clear that the cells must maintain their cellular structural integrity (i.e. "stiffness") to resist the distracting forces normally placed upon them in their function of suspending the distal phalanx—and therefore the entire musculoskeletal system—of the horse. Although dysadhesion of the epidermal lamellae from the dermal lamellae (leading to lamellar separation) was traditionally described as the primary cause of lamellar failure, stretching of the epidermal lamellae through loss of structural integrity of the lamellar epidermal cells is now realized to be a central component to lamellar failure. When either the structural integrity of the epidermal cells or the adhesion of these cells to the underlying dermis is compromised, the resulting loss of structural strength of the digital lamellae, combined with the forces on the distal limb, leads to a displacement of the distal phalanx. Three types of displacement of the distal phalanx are described: (1) symmetrical distal displacement in which the entire distal phalanx displaces distally, (2) asymmetrical distal displacement in which the medial (most common) or lateral aspect of the distal phalanx displaces distally, and (3) dorsal rotation in which the dorsodistal tip of the distal phalanx displaces in palmarodistal direction away from the hoof wall (discussed in detail later in the chapter). Throughout this discussion, the term palmar used for the forelimbs is intended to be inclusive of plantar for the hindlimbs.

The term laminitis has been used to describe all clinical presentations of equids in which structural failure of the lamellae occurs. As laminitis occurs as a sequela to other diseases, the types of laminitis are categorized according to the general predisposing disease states associated with lamellar failure: systemic sepsis/endotoxemia (sepsis-related laminitis [SRL]), systemic endocrinopathies (endocrinopathic laminitis), or a disease or injury to one limb that necessitates preferential weight-bearing on the opposite limb (supporting limb laminitis [SLL]). Although the predisposing disease states are readily recognized, there has been a great deal of controversy for decades regarding the specific pathophysiologic mechanisms leading to lamellar failure in each type of laminitis, and, as therapies for diseases are based on the pathophysiology known for that disease, disagreement about the cascade of events leading to lamellar failure has led to confusion regarding therapeutic regimens for the disease. The somewhat recent establishment of the three primary types of laminitis has led to more rapid advancements in our understanding of systemic and local pathophysiologic mechanisms through the more accurate description of clinical cases and through the establishment of experimental models for all three types of laminitis. Concomitant with our increased understanding of the pathophysiology of the different types of laminitis, effective management and treatment tools are now being discovered.

This chapter will review the relevant anatomy, our current understanding of pathophysiologic mechanisms occurring in the different types of laminitis, the clinical presentations of the animals, and finally, a discussion of current diagnostic and therapeutic procedures for the different types of laminitis.

RELEVANT ANATOMY

The distal phalanx of the equine digit is surrounded by the hoof, consisting—similar to the skin—of the deeper dermal tissues and more superficial epidermal tissues. The dermal tissue surrounds and attaches to the outer cortex of the distal phalanx through components of the extracellular matrix (e.g. collagen fibers). In addition to providing important structural functions through the connective tissue attachments to the distal phalanx, the dermal tissue also contains all of the vasculature and innervation of the hoof. The dermis of the hoof wall consists of the sublamellar dermis (also described in the literature as "deep dermis," which attaches to the distal phalanx axially and continues abaxially as the dermal lamellae [containing the microvasculature upon which the epidermal lamellae depend for nutrition]). Importantly, the deep dermis also extends on the palmar/ground surface of the distal phalanx, providing the vasculature and innervation upon which the solar epidermis is dependent.

Similar to the skin, the epidermal component of the hoof consists of both keratinized nonviable components externally and live epidermal cells internally. The outer hoof wall, consisting of the stratum externum and stratum medium, provides both protection to the digit and a critical support function due to fact that, through the lamellar attachments to the distal phalanx, the hoof wall bears the majority of weight of the horse normally via the hoof wall's contact with the ground. The epidermal lamellae, part of the stratum internum of the hoof wall, extend vertically from the coronary groove of the proximal hoof wall distad to merge with the sole; they are placed around the periphery of the distal phalanx, interdigitating with the same number of dermal lamellae. There are approximately 550–600 primary epidermal lamellae, each of which consists of a keratinized, acellular core (continuous with the keratinized hoof wall portion of the stratum internum). Approximately 150–200 secondary epidermal lamellae emanate from each primary epidermal lamella, interdigitating with secondary dermal lamellae. The secondary epidermal lamellae are made up of two types of live epithelial cells, basal

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epithelial cells interfacing with the lamellar dermis, and parabasal epithelial cells on the interior of each secondary epidermal lamella. These cells appear to function similarly to skin epithelial cells, with basal epithelial cells attaching to the matrix fibers of the underlying basement membrane (continuous with the matrix fibers of the dermal lamellae) primarily via the same protein complexes present in the basal epithelial layer in the skin: hemidesmosomes. The lamellar epithelial cells also have an intricate cytoskeleton, likely providing the same "stiffness" to the lamellar cells as is reported in epithelial cells in other species. In lamellar failure in laminitis, it is likely that dysregulation of epithelial cytoskeletal dynamics leads to lamellar stretching of the epidermal lamellae, whereas dysregulation of hemidesmosome complexes (attached to the intermediate filament aspect of the cytoskeleton) appears to lead to dysadhesion of the lamellar basal epithelial cells from the basement membrane and therefore to separation of the epidermal and dermal lamellae.

PATHOGENESIS: PATHOPHYSIOLOGIC CONSIDERATIONS

The delineation of the three general types of laminitis has allowed more focused investigations into systemic and local lamellar events occurring in these different types of the disease. Additionally, the availability of state-of-the-art research tools to assess lamellar events has allowed rapid advancement of knowledge over the last decade. Finally, although we have had models of SRL for several decades, we have only recently developed experimental models of endocrinopathic laminitis and SLL.

Experimental models for SRL include both carbohydrate overload models and the black walnut extract model. These models were developed in order to closely emulate the clinical cases of laminitis caused by either consumption of excessive carbohydrate or of black walnut shavings (used for bedding). The first CHO overload model established included the gastric administration of a mixture of corn starch and wood flour (commonly termed the starch gruel model).[34,35] Today, this starch gruel model has largely been replaced by the oligofructose model in which a gastric bolus of oligofructose in solution is administered.[21,22,53] Whereas the black walnut extract model was useful for examining early events in the disease process,[7,47] the fact that most animals did not progress to structural lamellar failure somewhat decreased the clinical relevance of (and interest in) the model.[7] Using these models, a profound inflammatory response was detailed in the lamellar tissue (much greater than other tissues in the body).[7,46,47] Interestingly, similar inflammatory events were discovered as occur in organ injury in human sepsis, including the extravasation of leukocytes into the lamellar tissue[26,28,29] and the marked increased expression of inflammatory mediators/enzymes in the lamellar tissue including cytokines,[46] chemokine,[27,30] and cyclooxygenase (COX)-2.[46,70] Although lamellar ischemia due to decreased blood flow has been purported as the cause for lamellar failure in SRL, conflicting results are present in the literature, primarily due to the lack of a technique to accurately assess lamellar perfusion. In recent investigations in which a microdialysis probe is inserted into the lamellar tissue to assess lamellar events in the oligofructose model, lamellar perfusion does not appear to change, whereas metabolite changes suggest a similar dysfunction of mitochondrial respiration as reported in models of human sepsis.[51] Due to histologic evidence of lamellar stretching and dysadhesion in the oligofructose model of SRL, and electron microscopic evidence of cytoskeletal and hemidesmosome disarray,[32,43] it is likely that dysregulation of both cytoskeletal dynamics and adhesion dynamics is playing a role in lamellar failure in SRL. Although earlier reports proposed that matrix breakdown by matrix metalloproteases (MMPs) may play an important role in lamellar failure,[31,42,52,59] the lack of presence of activated forms of MMPs of interest[48] and the lack of efficacy of potent a protease inhibitor as a treatment for laminitis in the oligofructose model decrease the likelihood of proteases playing the central event in lamellar failure (Underwood and Pollitt, unpublished data). Finally, the same lamellar growth factor-related signaling (possibly through the proinflammatory cytokine IL-6)[16] as discussed with endocrinopathic laminitis has been documented to occur in the carbohydrate models of SRL (Belknap laboratory, unpublished data) and may play an important role in lamellar epithelial cell dysregulation and lamellar failure.

In endocrinopathic laminitis, including both metabolic syndrome and pituitary pars intermedia dysfunction (PPID), both clinical and experimental studies indicate that hyperinsulinemia (commonly termed insulin dysregulation) is the primary factor inducing lamellar failure. In a study of horses diagnosed with PPID, laminitis only occurred in those animals with hyperinsulinemia.[39] In equine metabolic syndrome (EMS), the term "insulin resistance" has been taken from the human medical literature on metabolic syndrome in which obesity leads to a poorer systemic response to insulin, most likely due to inflammatory mediators released from the excessive adipose tissue. In EMS, this obesity-related systemic inflammation has not been conclusively demonstrated; it appears that the main risk factor for laminitis may be a propensity toward hyperinsulinemia upon ingestion of carbohydrate independent of actual insulin resistance. The role hyperinsulinemia plays in lamellar failure is well supported by the most commonly used experimental model for endocrinopathic laminitis, the euglycemic hyperinsulinemic clamp (EHC) model. In this model, elevated blood insulin levels (in the presence of normal blood glucose concentrations) lead to the same histologic changes to the lamellae as observed in clinical cases of endocrinopathic laminitis.[39,43] In regard to the cause of lamellar failure due to hyperinsulinemia, it has recently been reported that the lamellar epithelial cells in the EHC model of endocrinopathic laminitis undergo the same growth factor-related signaling shown to disrupt normal cytoskeletal and adhesion dynamics in cancer biology when epithelial cells transform into cancer cells (termed epithelial-to-mesenchymal transition).[44] In both clinical cases of endocrinopathic laminitis and in the EHC model, the lamellae do not commonly undergo the dramatic dysadhesion/separation of the lamellar basal epidermal cells from the underlying dermal lamellae that commonly leads to rapid structural failure in SRL. Instead, lamellar stretching plays a more prominent role,[43] likely due to

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loss of rigidity due to cytoskeletal dysregulation/collapse secondary to aberrant epithelial cell signaling. Whereas these lamellar changes take place over 48 hours in the EHC model (likely due to the supraphysiologic blood insulin concentrations maintained),[43] the changes can occur gradually over months to years in the clinical patient.

In SLL, a recently introduced model in which a shoe with a V-shaped insert on one limb causes excessive weight-bearing on the other limb has started to shed light on the events preceding lamellar failure. This establishment of a model for SLL was critical due to the fact that clinical cases of SLL are characterized by a rapid onset of severe lamellar injury/separation, making it difficult to assess early changes in the disease process. Whereas the first study using this model suggested an increase in lamellar hypoxia in SLL due to increased concentrations of a protein induced by hypoxia,[33] a more recent study using the same SLL model in which microdialysis probes were placed in the lamellae more clearly indicated a decrease in lamellar blood flow starting approximately 48 hours after the onset of preferential weight-bearing (A. van Eps, unpublished data). On histology of lamellar samples harvested at 96 hours of preferential weight-bearing, both lamellar stretching and dysadhesion were present (Belknap and van Eps, unpublished data). As the decreased blood flow is likely due to physical factors such as a lack of movement,[23] it is unlikely that pharmacologic vasodilation will be effective in preventing or treating SLL. The breakdown in lamellar epithelial structure in SLL may not only be due to the direct effects of hypoxia on cell metabolism; we have also recently established that the same growth factor-related signaling as discussed above is occurring in the lamellar epithelial cells in the SLL model; this is most likely stimulated by factors activated due to hypoxia (Belknap and van Eps, unpublished data). Thus, in all three types of laminitis, the induction of this same growth factor-related signaling may lead to lamellar failure through disruption of epithelial cell cytoskeletal dynamics and adhesion to the underlying matrix.

PATHOGENESIS: STRUCTURAL CONSIDERATIONS OF THE EQUINE DIGIT

The stresses on the lamellae are related to the stresses borne by the two anatomical structures to which the lamellae attach, the hoof wall and the distal phalanx. Different stresses occur due to both weight-bearing and locomotion. The three primary forces affecting the lamellae include:

1. The downward force of the horse's weight through the distal phalanx
2. The torque (or moment) around the distal interphalangeal (DIP) joint created by the ground reaction force (the force exerted by the ground on the digit; also termed extensor moment)
3. The tension of the deep digital flexor tendon (DDFT) exerted on the caudal aspect of the distal phalanx (also termed the flexor moment; Figure 4.65)

It has been demonstrated in a horse with healthy feet that the sole of the foot can support the weight of the limb (i.e. counteracting the downward force on the distal phalanx from the horse's weight), but it is unknown how the forces associated with weight-bearing are distributed from the sole to the distal phalanx. More specifically, the question is how much of the weight borne by the sole is directly transferred to the distal phalanx and how much is redirected through the lamellae to the distal phalanx. The following discussion assumes that there is some direct support of the distal phalanx by the sole, but this remains unproven. In the horse shod with standard shoeing that is standing on a firm flat surface, all the stress of weight-bearing is borne by the lamellae because the only relationship between the foot and the ground surface is through the part of the foot contacting the shoe, the distal hoof wall. On the assumption that significant weight is transferred directly from the sole to the distal phalanx, then a decrease in solar support of the distal phalanx will be exacerbated by the elevation of the sole off the ground surface due to the thickness of the shoe; in the unshod foot, the character of the ground surface would therefore affect the amount of stress

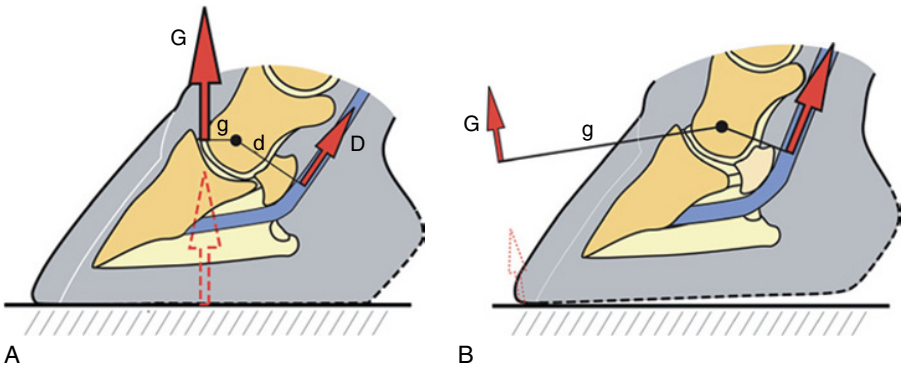


Figure 4.65. (A) At rest, the foot is stable with respect to the ground. The ground reaction force is approximately vertical, and positioned approximately in the center of the foot, slightly in front of the center of rotation of the distal interphalangeal joint. The product of the magnitude of the GRF (G, large red arrow) and the length of its moment arm (g) is the extensor moment, which is opposed by the flexor moment, which is the product of the force in the deep digital flexor tendon (D, small red arrow) multiplied by the length of its moment arm (d). (B) At break-over, the position of the foot is dynamic, and the magnitude of the ground reaction force (G, small red arrow) is decreased as the horse moves off the leg, but the length of the moment arm (g) is increased because the GRF is positioned at the toe. To cause the foot to move from the stable position at rest to the dynamic state, the flexor moment exceeds the extensor moment.

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borne by the lamellae. On a hard surface (including artificial surfaces commonly used in stables), only the distal hoof wall and adjacent sole contact the ground. This results in a similar great degree of stress on the lamellae similar to the foot with standard shoes. This is due to the combined effects of concentration of stress through the hoof wall to the lamellae and the lack of support of the distal surface of the distal phalanx. In both of these instances, the distal phalanx can be thought of as being suspended within the foot by the lamellar attachments to the hoof wall. When placed on a softer surface such as sand (or possibly an elastic flooring material), the entire solar surface can be engaged to directly support the distal phalanx in addition to being suspended by the lamellar attachments to the hoof wall. This helps to distribute the digital support through both anatomic structures and thus decrease the stress on the lamellae.[37] As is discussed later, addition of putty to the solar surface in the shod horse has the same end result.

At rest, the center of weight-bearing is approximately at the center of the ground surface of the foot. During the majority of locomotion, the stresses associated with weight-bearing are also centered on the ground surface of the foot. However, during the breakover stage of the stride, ground reaction forces are localized to the toe of the foot, resulting in an increase in the length of the moment arm (and hence the torque or moment; see Figure 4.65) and thus the stress on the dorsal lamellae.

In a healthy horse, the strength of the lamellae greatly exceeds the stresses applied, thereby allowing the distal phalanx to be retained in its normal position. However, in horses with laminitis, the lamellar attachments to the distal phalanx are compromised, as discussed above. Partial loss of the lamellar integrity due to epithelial cell stretching and loss of cellular attachments to underlying connective tissue causes the remaining intact lamellae to endure the entire load of weight-bearing and locomotion, so the remaining lamellae are potentially exposed to a cycle of excessive mechanical stress leading to further stretching and dysadhesion or, possibly, tearing and subsequent failure of suspension of the distal phalanx.

The pattern of displacement of the distal phalanx within the hoof capsule varies between horses and sometimes between an individual horse's feet. The distal phalanx may displace evenly around the circumference of the hoof wall, resulting in distal displacement or sinking (Figure 4.66).

Alternatively, the distal phalanx may displace unevenly in relation to the hoof wall. A unilateral distal displacement results in either medial or lateral displacement or sinking of the distal phalanx (Figure 4.67). Although this type of asymmetric displacement is more uncommon than the dorsal rotational types of distal phalangeal displacement, it is important to assess for this because it is a devastating displacement that requires specific foot care.

The most common pattern of asymmetric displacement, usually termed rotation of the distal phalanx, occurs when the dorsal distal margin of the distal phalanx moves distally and the dorsal parietal surface of distal phalanx diverges from the dorsal surface of the hoof wall, with the distal aspect of the parietal surface of the phalanx being further displaced from the dorsal hoof wall than the proximal aspect. Rotation is categorized as:

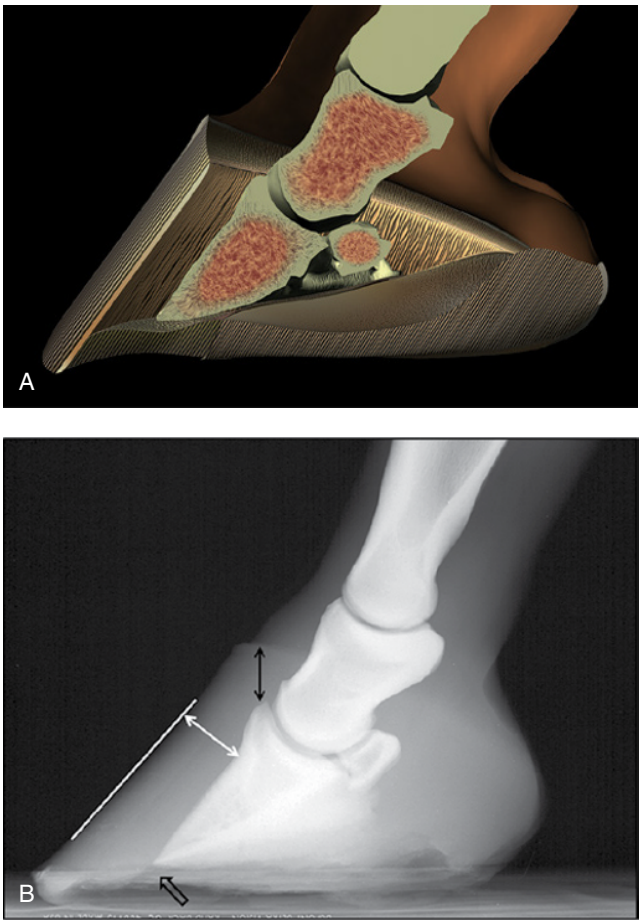


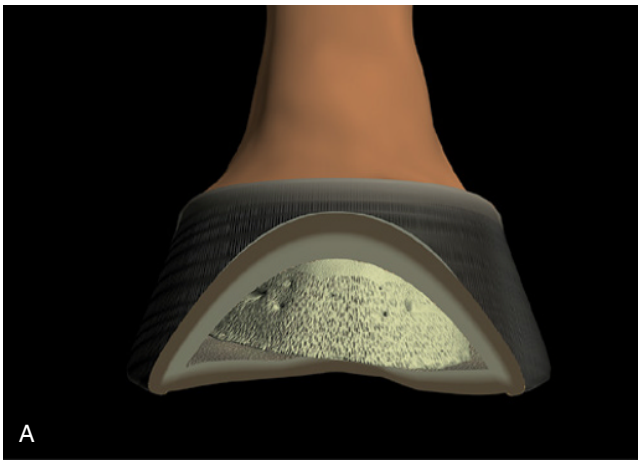
Figure 4.66. In symmetrical distal displacement, the distal phalanx descends within the hoof capsule (A). Therefore, the distal phalanx retains its alignment with the more proximal phalanges and the hoof capsule (B), but the distance between the parietal surface of the distal phalanx and the hoof wall (white arrow) and the distance between the proximal extensor process and the proximal border of hoof wall (black double arrow) increase, and the distance between the distal phalanx and the sole and ground decreases (open arrow).

1. Capsular rotation (Figure 4.68): Divergence of the parietal surface of the distal phalanx from the dorsal hoof wall (with or without flexion of the DIP joint)
2. Phalangeal rotation (Figure 4.68): Palmar rotation of the distal phalanx (flexion of the DIP joint with or without capsular rotation) in relation to the axis of the phalanges

In most horses, the pattern of displacement is a combination of distal displacement and rotation of the distal phalanx, although one pattern is usually dominant.

In horses with acute lamellar failure (most common in SRL and SLL), the forces upon the hoof wall may rapidly exceed the support provided by the remaining intact interdigitating dermal and epidermal lamellae. This results in tearing of these remaining lamellae and displacement of the distal phalanx. The potential space

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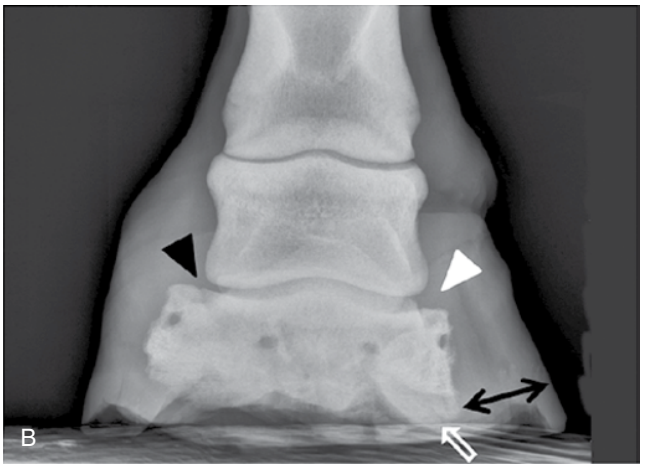
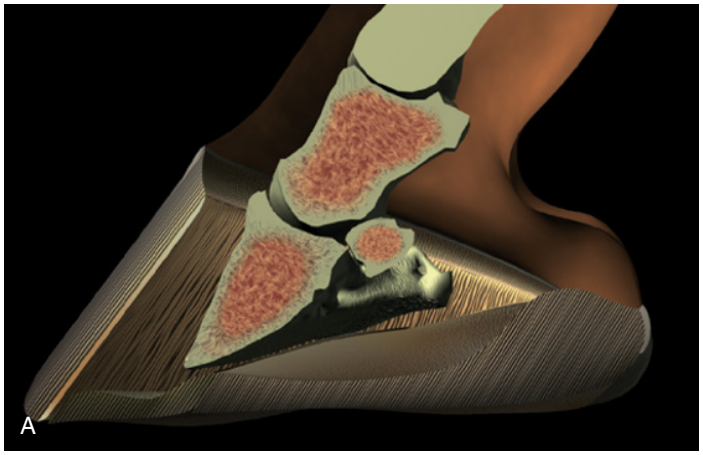


Figure 4.67. In medial or lateral asymmetrical displacement (A), one side of the distal phalanx descends (white open arrow in B). The distance between the wall and the distal phalanx increases (black double arrow), and the distance between the distal phalanx and the sole and ground decreases on the affected side (white open arrow). Additionally, the DIP joint becomes asymmetrical when viewed on a dorsopalmar radiograph; the joint space is increased on the affected side (white arrowhead) and decreased on the unaffected side (black arrowhead).



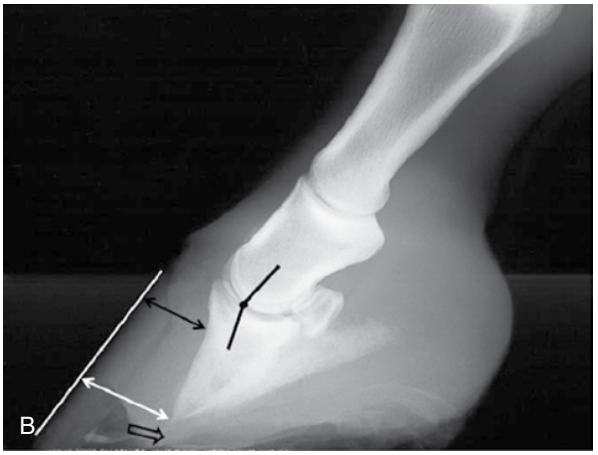


Figure 4.68. (A and B) In horses with early chronic laminitis, the surface of the hoof wall is unchanged, but the distal phalanx rotates about the DIP joint (the joint flexes, black lines in B); consequently, the normal alignment of distal phalanx with the other phalanges is changed (phalangeal rotation). Additionally, the distance between the dorsal distal parietal surface of the distal phalanx and hoof capsule increases (white double arrow) while it remains close to normal proximally (black double arrow), i.e. there is divergence of the surfaces (capsular rotation). Also, the distance between the dorsal margin of the distal phalanx and the sole and ground is decreased (open arrow).

left between the hoof wall and the displaced distal phalanx is likely to fill with blood and necrotic tissue (this area on radiographs may appear radiolucent for a short time after displacement).

After separation of the lamellae, some germinal epithelial cells remain on the deep surface of the separated epidermal lamellae, and some remain attached to the surface of the dermal lamellae. Similar to wound healing in other tissues, remaining viable germinal epithelial cells proliferate. However, the underlying damaged dermis is unable to exert normal control of epithelial proliferation.[60,71] Therefore, remaining epithelial cells undergo aberrant proliferation in the space created by displacement of the distal phalanx, resulting in a lamellar wedge of disorganized epithelial tissue, the apex of which is proximal and the broader base distal (Figure 4.69). This wedge most likely impedes the ability of the clinician and farrier to realign the distal phalanx with the dorsal hoof wall.

THE CLINICAL PRESENTATION: THE HORSE AT RISK OF LAMINITIS

As we can now place most laminitis cases into one of the three types of the disease, we realize that the clinical presentation can differ greatly not only between animals but also between the different types of laminitis. Classically, the literature has attempted to fit the laminitic horse into one of three categories: developmental, acute, or chronic laminitis. The developmental/prodromal stage was defined as the stage prior to the horse exhibiting clinical signs (i.e. the septic horse not exhibiting digital signs); the acute stage has been defined as the animal exhibiting clinical signs of digital pathology but no radiographic signs of displacement of the distal phalanx; and the chronic stage describing the animal in which displacement of the distal phalanx is present on radiographs. The authors do not find these categories useful for two

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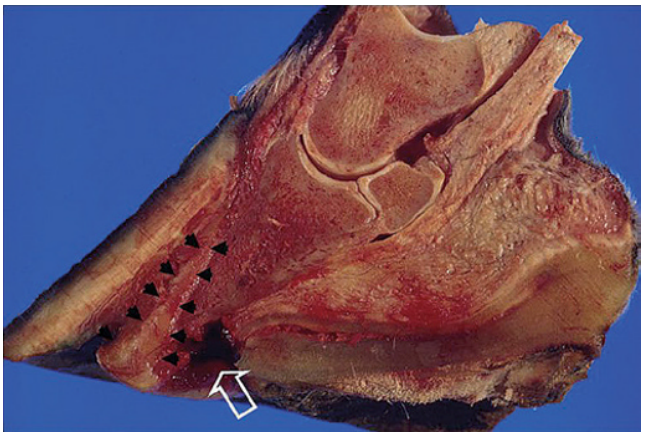


Figure 4.69. A sagittal section of the hoof wall of a horse with capsular rotation of the distal phalanx. Note the lamellar wedge (black arrows) of epithelial tissue that forms from remaining lamellar epithelial cells following displacement of the distal phalanx. Also note the penetration of the sole by the dorsodistal tip of the distal phalanx immediately dorsal to the apex of the frog (white arrow).

reasons: they do not accurately describe the range of clinical cases (not all patients with sepsis are actually in the developmental stage of SRL), and, furthermore, they do not lend themselves to treatment recommendations. Rather than these categorizations, it is best to describe the initial period as an "at-risk" period instead of a prodromal or developmental period. In addition to defining the animal at risk for the different types of laminitis (for prophylactic management of the animal), it may be best to define the acute and chronic stages in a temporal sense, with the acute stage being the first 3–4 weeks after signs of laminitis first occur (regardless of the placement of the distal phalanx) and the chronic stage being the animal treated from 4 weeks to years later.

The equid at risk of SRL is an animal suffering from an infection (e.g. enterocolitis, pneumonia, acute endometritis) or from a compromised viscus (e.g. volvulus of the large colon) that results in the animal exhibiting systemic signs of sepsis/endotoxemia (e.g. fever, injected mucous membranes, tachycardia). Although SRL research has demonstrated that lamellar inflammatory events begin long before the animal exhibits clinical signs of laminitis,[28,47] it has also been documented in both the clinical and research setting that effective intervention with digital hypothermia (discussed later) even early into the onset of clinical lameness can protect the lamellae from structural failure.[18,25] Thus, it is critical to detect these animals at risk of SRL for initiation of prophylactic therapy. Most animals with SRL will exhibit signs of laminitis prior to lamellar failure/displacement of the distal phalanx, including throbbing digital pulses, increased hoof warmth, response to hoof testers, and lameness especially exacerbated on tight turns. Again, in the authors' experience, this is likely the last possible time point to initiate therapy to avoid lamellar failure, as, once the animals are exhibiting severe lameness (i.e. Obel grade 3–4 laminitis), severe lamellar injury and displacement of the distal phalanx are commonly already present. In the interest of identifying patients early in the disease process, it is important to understand that although the forelimbs are usually most severely affected in SRL, it is also the subcategory of laminitis in which patients stand the greatest risk of having all four limbs affected. Whereas, with only forelimb involvement, the animal places its forelimbs and hindlimbs farther forward than normal (to get weight off forelimbs and place more weight on hindlimbs), the animal with involvement of all four limbs may place the forelimbs forward and the hindlimbs further caudad than normal (appearing "camped out").

The equid at risk of endocrinopathic laminitis is now realized to not always be as straightforward to identify as those at risk of the other types of laminitis. The two primary types of endocrinopathies putting the horse at risk of endocrinopathic laminitis, EMS and PPID, can have disparate presentations. For EMS, similar to the human component of metabolic syndrome, obesity is a major risk factor. Additionally, and also in parallel with findings in human medicine, genetics are recently being ascribed a larger role in EMS. This interplay between obesity and genetics is most clearly demonstrated by overweight animals that never exhibit signs of EMS-associated laminitis (EMSAL). Conversely, there are cases of athletic animals with only a moderately elevated body condition score (e.g. show jumpers) that develop EMSAL. Similar to EMS, it has recently been reported that not all animals diagnosed with PPID are at risk of laminitis, only those with insulin dysregulation.[39] Thus, diagnostic testing needs to be combined with physical signs to establish those animals at risk of endocrinopathic laminitis. Animals with pasture-associated laminitis (animals that develop laminitis when exposed to lush pasture) are included in EMSAL; these animals may exhibit the classic signs of laminitis (i.e. digital pulses, hoof warmth) after a short period of time on pasture or may undergo an insidious displacement of the distal phalanx that may go unnoticed for extended periods of time. These animals may exhibit no clinical signs of foot pathology while a gradual displacement of the distal phalanx is occurring (likely over months to years). Thus, in the authors' experience, the first sign of foot disease detected by the owner or trainer may be lameness from solar bruising due to the pressure of a moderately to severely displaced distal phalanx on the solar tissue. For this reason, it has been suggested to obtain radiographic films of the forelimb digits once a year to detect any changes in the relationship of the distal phalanx to the hoof wall (see chapter on imaging component).

The equid at risk of SLL is the animal preferentially bearing weight on one limb due to injury/disease to the contralateral limb causing pain or inability to place weight on that limb. This can occur due to fracture, synovial sepsis, nerve injury leading to paresis, or even long-term subsolar sepsis. Although SLL usually occurs in the animal with severe unilateral lameness, it can occur in the animal with moderate lameness. In a survey of equine surgeons in private and university practices, they reported that the most common cause of lameness in the contralateral limb in SLL cases was sepsis of synovial structures.[73] Similar to endocrinopathic cases, there is not a defined developmental period, as SLL may develop any time from 2 to 3 days to over 60 days of preferential weight-bearing on one limb. The animal at risk of SLL may exhibit signs of the disease within a few days of becoming lame on the opposite limb, although signs of laminitis commonly occur after the animal has been exhibiting lameness on the opposite limb for several weeks. Once signs of laminitis occur, the SLL is

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commonly characterized by a rapidly progressing catastrophic failure of the lamellae (with extensive separation of the dermal and epidermal lamellae), resulting in rapid displacement of the distal phalanx. Earlier signs of a milder lameness may not be apparent due to the lameness already present on the opposite limb.

CLINICAL SIGNS: THE ACUTE ONSET OF LAMINITIS

The clinical signs assessed in laminitis mainly refer to the degree of lameness noted and the digital exam. The lameness varies in severity from being barely detectable to an animal that is recumbent the majority of the time secondary to digital pain. Due to this variability, Obel introduced a grading system to describe the degree of lameness (Table 4.2) more than 60 years ago. Although the Obel grading system does not describe all cases of laminitis (it appears to be primarily based on cases of SRL), it is still used to describe the degree of lameness in both the clinics and when describing time points assessed in laminitis research.

Table 4.2. Obel grades of laminitis.

Grade	Associated lameness and gait abnormalities
1	At rest the horse alternately and incessantly lifts the feet, often at intervals of a few seconds. Lameness is not evident at the walk, but a short stilted gait is noted at the trot
2	The horse moves willingly at a walk, but the gait is stilted. A foot can be lifted off the ground without difficulty
3	The horse moves very reluctantly and vigorously resists attempts to have a foot lifted off the ground
4	The horse refuses to move and will not do so unless forced

Clinical signs of SRL are most commonly observed 24–72 hours following the onset of a septic disease process. The onset of disease in endocrinopathic laminitis is extremely variable; whereas an insidious, "subclinical" displacement of the distal phalanx may take place over months to years in the horse with EMS, pasture-associated laminitis may exhibit an acute and severe onset similar to SRL. In SLL, disease progression is extremely inconsistent between animals, with clinical signs of SLL occurring within days, weeks, or months after the onset of excessive weight-bearing in the affected digit.

The clinical signs of acute laminitis are characterized by lameness, an increase in the temperature of one or more hooves, increased digital pulses, and elicitation of a painful withdrawal response to hoof testers. In EMS and sepsis, the forelimbs are most commonly affected (most likely due to more weight being borne by the forelimbs); the hindlimbs may also be involved (usually in severe septic cases). SLL occurs on the supporting forelimb or hindlimb opposite the non-weight-bearing limb. The characteristic stance of a laminitic horse with both forefeet affected is placement of the forefeet well in front of the normal position and anterior placement of the hindfeet in order to shift more weight to the hindlimbs (Figure 4.70).[37]

The characteristic gait of a laminitic horse is stiff limb movement while maintaining the same laminitic posture of both fore- and hindlimbs being placed anteriorly. Horses in which all 4 feet are affected may have a more normal stance or may appear "camped out" with



Figure 4.70. Classic stance of a horse with laminitis with the forelimbs placed abnormally far forward and weight shifted toward the hindlimbs. *Source*: Courtesy of L. Brandstetter.

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forelimbs placed more anterior than normal and hindlimbs placed more posterior than normal. Similarly, their gait may be more normal in appearance than the animal with only forelimb involvement. Importantly, patients with distal displacement of the third phalanx in all four limbs are usually intensely resistant to movement and more commonly recumbent due to severe pain. It is at times difficult to determine whether the forelimbs or both forelimbs and hindlimbs are affected; short-lived anesthesia (using lidocaine) of the forefeet with abaxial sesamoid nerve blocks helps determine whether any lameness exists in the hindfeet or if any abnormality noted in hindlimb gait is merely compensation for the painful forelimbs.

Increased hoof temperature and the strength of digital pulses must be interpreted in the context of the lameness and other systemic symptoms, because it is not unusual for an increase in hoof temperature to be transient, and the interpretation of digital pulses is highly subjective. Application of hoof testers is useful to confirm the presence of pain around the dorsal margins of the sole in cases with mild lameness in which laminitis is suspected. The clinician can also use hoof testers to detect unilateral (more accurately described as uniaxial) pain (medial vs. lateral pain) in unilateral distal displacement. Less commonly, hoof testers can be used to detect heel pain in some chronically affected horses with secondary heel contracture. The veterinarian must keep in mind differential diagnoses such as sole bruising that, in the early stages, can present similarly with digital pulses and bilateral pain in response to hoof testers.

Horses with chronic laminitis have different degrees of hoof capsule deformation and lameness. Changes to the hoof capsule vary with the duration of the disease and type of displacement and may be visible in both the wall and the sole. In the case of a patient with chronic laminitis and rotation, the wall is unchanged in the immediate period after displacement of the distal phalanx, but commonly assumes a dorsally concave appearance as the disease progresses due to disparate growth between the toes and the heels (slower growth in the toe region) and the presence of a lamellar wedge. This disparate growth is also reflected in abnormal growth rings, which are more widely spaced in the heel than the toe (Figure 4.71). The sole dorsal to the apex of the frog may be soft and flattened (or convex) due to downward pressure on the sole from the dorsodistal aspect of the displaced distal phalanx secondary to either rotation or distal displacement. The flat, soft character of the sole may lead to bruising around its dorsal margin. The white line may be wider than normal, frequently with elongated keratinized lamellae, and show evidence of prior bruising/hemorrhage. In cases of distal displacement of the third phalanx, there may be a palpable (and sometimes visible) groove at the junction of the skin of the pastern and the coronary band. The lameness in the long-standing chronic case is commonly only from excessive sole pressure by the displaced distal phalanx, with minimal lamellar pain present due to healing of the lamellae (i.e. the lamellae are stable and therefore pain-free even if abnormal morphologically). The most common exception to this is the horse with recurrent laminitis due to either PPID or EMS, in which bouts of lamellar injury/instability commonly recur.



Figure 4.71. This foot from a horse with chronic laminitis demonstrates abnormal hoof growth with wider growth rings at the heel where there is more growth (small arrows) and converging to narrowly placed rings at the toe (large arrow) where minimal hoof growth is occurring. *Source*: Courtesy of Dr. Gary Baxter.

Diagnosis

The diagnosis of laminitis may be obvious in acute severe cases due to the history, characteristic stance, and digital exam. The diagnosis may be much more difficult in chronic mild cases, especially in older horses that may have bilateral distal limb arthropathies contributing to the lameness. In the acute case with severe lameness of the forelimbs, one author (JKB) performs an abaxial sesamoid nerve block with lidocaine in order to:

1. Enable the clinician to rapidly obtain good quality radiographs without undue stress to the horse
2. Enable thorough examination of the solar surface
3. Assess for involvement of the hindlimbs (by limited walking of the horse around the stall in each direction)
4. Enable the application of therapeutic shoes or pads to the affected limbs

It is important to note that lidocaine is the local anesthetic of choice for this perineural anesthesia as its short duration of action will ensure that the patient will not put excessive stress on already tenuous lamellar attachments.

In the chronic, mild case, it is best to initially block the digit with a palmar digital (PD) nerve block, which blocks solar pain due to a displaced distal phalanx,[65,66] a common cause of pain in the chronic case of laminitis. Although it has recently been established that the PD nerve (not the dorsal branch) provides the majority of the innervation to the lamellae in normal horses,[58] the PD block does not appear to block a lameness due to dorsal lamellar pain in most laminitic horses. The reason for this disparity may be that lamellar pain in laminitic horses is likely due to lamellar instability, which

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commonly results in a severe pain that may not block out with local analgesia. Thus, if the PD nerve block abolishes the lameness on the affected digit, it is likely that the pain may be due to solar pain primarily and that the lamellae are stable.

In the horse with suspected chronic laminitis in which it is unknown whether the lameness is due to laminitis or other musculoskeletal issues, it is important to perform distal limb flexion prior to nerve blocks; if the horse is severely lame, the response to flexion can be assessed at a walk. A response to flexion usually indicates the presence of an arthropathy unrelated to laminitis, the most common of which is arthropathy of the proximal interphalangeal joint.

To delineate lamellar pain from proximal interphalangeal joint pain in the horse that does not respond to a PD nerve block, the clinician can assess any remaining pain emanating from the foot by performing a modified ring block immediately proximal to the coronary band to block the dorsal branches of the PD nerve. When attempting to rule out the other common cause of bilateral forelimb lameness, namely, navicular syndrome/palmar heel pain (cases with severe degeneration of the navicular bone can mimic the lameness of a moderately severe case of chronic laminitis), the history, presentation, and radiographs are valuable in addition to the response to nerve blocks. Because PD nerve anesthesia can block both palmar heel pain and solar pain and likely a great deal of lamellar pain,[58] radiographs should be used to help differentiate between them. As with any type of severe musculoskeletal pathology, the lameness may not block out entirely with local perineural anesthesia in some severely painful cases.

Radiography

Radiography is not only critical as a diagnostic tool to determine the presence of the disease but is also critical to monitor progress of the disease and guide treatment. Radiographs should be taken at the first sign of acute laminitis to serve as a baseline for subsequent radiographic comparisons and determine if radiographic changes suggestive of previous laminitis are present. The most important views are the lateral and the dorsopalmar/plantar projections (Figures 4.67, 4.72, and 4.73). For both of these projections, it is important that the foot is placed on a block and that the X-ray beam is centered as close as possible to the solar margin of the distal phalanx (approximately 1.5 cm proximal to the surface of the block).

Early radiographic signs suggestive of distal displacement of the distal phalanx include: 1) increased dorsal hoof wall thickness (e.g. widening of the distance between the dorsal surface of the hoof wall and the parietal surface of the distal phalanx)[14] and 2) increased vertical distance from the proximal aspect of the extensor process to the firm proximal border of the hoof wall located immediately distal to coronary band (sometimes termed the founder surface of the hoof capsule). It is important to ensure that the lateral radiographic view is a true lateral; rotation of the axis of the foot by more than 10° causes the degree of rotation to be underestimated.[40]

A radiopaque object or paste can be applied to the mid-dorsal hoof wall and should end at the level of the coronary band to help identify it (Figure 4.72). The distance between the dorsal surface of the hoof capsule and the parietal surface of the distal phalanx is best measured as the shortest distance between the two immediately distal to the base of the extensor process (in Figure 4.72b) to limit the effect of any rotation on the measurement. This measurement is most valuable when pre-disease radiographs are available and then it is a relatively reliable indicator of displacement in horses in which the distal phalanx has undergone distal displacement. In normal horses (approximately 450 kg), the distance should be less than 18–20 mm (reported means of 14.6–16.3 mm). With digital radiographs it is possible to identify two layers of the hoof wall, the outer being more radiodense than the inner layer. In horses with distal displacement, the inner layer increases in thickness, while the outer layer is unaffected, at least initially. Therefore, an increase in the ratio of the inner layer to the entire thickness of the wall should also increase with distal displacement and is independent of the size of the horse. The normal ratio is approximately 40%.[36]

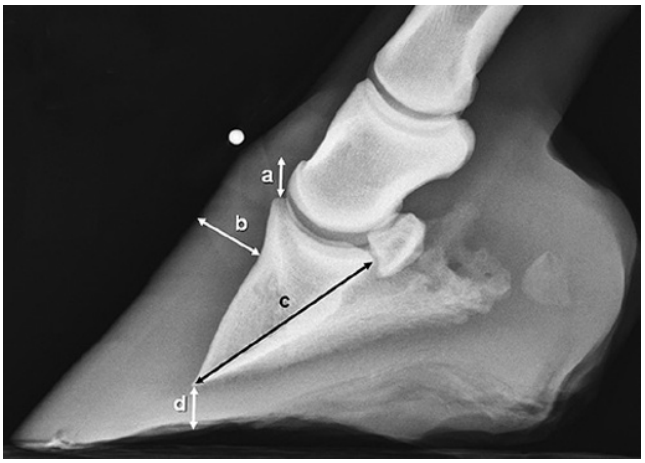


Figure 4.72. Several measurements obtained from lateral radiographs of the digit can be used to assess horses with distal displacement of the distal phalanx. a = distance from the proximal extensor process to the proximal aspect of the hoof wall (immediately distal to coronary band), b = the distance from the dorsal parietal surface of the distal phalanx to the dorsal surface of the hoof capsule, b/c = the ratio of the distance from the dorsal parietal surface of the distal phalanx to the dorsal surface of the hoof capsule (b) to the length of the palmar cortex of the distal phalanx (c), and d = the distance from the dorsodistal tip of the distal phalanx to the ground surface of the sole.

Additionally, an accurate measurement of the dorsal hoof wall thickness that takes into account the magnification and the size of the foot is the ratio between this dorsal measurement and the palmar cortical length of the distal phalanx measured from the dorsodistal tip of the bone to its articulation with the navicular bone (in Figure 4.72c). This ratio, which should be less than 28% in the normal horse, indicates possible distal displacement from 28% to 32% and indicates likely displacement if greater than 32%. The vertical distance from the extensor process to the firm proximal border of the hoof wall is also well used and is –2 to 10 mm in normal horses, depending on size (Figure 4.72a).[14] Due to the variability of this distance (–2 to 10 mm) in normal horses, it is most valuable to compare this

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measurement to pre-disease baseline radiographs to accurately assess any changes in the individual horse. Unilateral distal displacement can only be reliably assessed on a dorsopalmar/dorsoplantar projection (Figure 4.67).

Palmar/plantar rotation of the distal phalanx away from the dorsal hoof wall resulting in an angle greater than 5° confirms the diagnosis of laminitis due to capsular rotation (normal horses can have angles less than 4°). Two types of rotation can be assessed. Capsular rotation, the degree of rotation between the dorsal hoof wall and the parietal surface of the distal phalanx (α in Figure 4.73), is best reserved for assessment in the acute stage because the measurements can be difficult to interpret in horses with chronic laminitis due to deformation of the hoof wall. The angle of solar margin of the distal phalanx to the ground surface (β in Figure 4.73) is more useful in horses with chronic laminitis because it will remain unaffected by dorsal hoof wall deformation. Serial radiographs should be taken to monitor the progression of the disease and determine the success of selected treatments. Digital venography in the standing horse has been developed as a prognostic aid to assess the vasculature of the digit. A venogram in which there is no filling of contrast of the lamellar vessels, the circumflex area, and the terminal arch is reported to indicate an extremely poor prognosis for recovery.[63]

Treatment

The goal in the treatment of the acute laminitis case is to stabilize the digit in the short term regardless of the degree of displacement. In the authors' opinion, if the clinician can attain stabilization of the digital lamellae for approximately 3 weeks, distal phalangeal displacement can be addressed with other techniques including corrective shoeing and possibly deep digital flexor tenotomy in nonresponsive cases. In regard to foot support, the veterinarian must be willing to try a variety of techniques (discussed below) in an attempt to find the type of digital support that the animal responds to favorably.

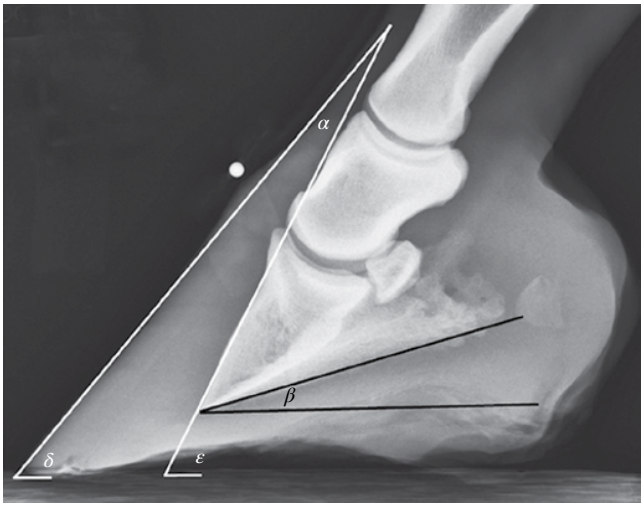


Figure 4.73. For assessment of rotation of the distal phalanx, the clinician can assess the degree of capsular rotation (angle α) at the intersection of the dorsal capsular and dorsal phalangeal lines or can measure the difference between the dorsal angles δ and ε. The relationship of the solar margin of the distal phalanx to the ground surface of the foot can be assessed by measuring angle β.

Anti-inflammatory Therapy

Anti-inflammatory therapy has endured as a central component of laminitis pharmacotherapy over the years. As discussed above, there is compelling evidence to use nonsteroidal anti-inflammatory drugs (NSAIDs) due to marked inflammatory events occurring both prior to and at the onset of lameness in the laminitic horse. Therefore, aggressive, prudent use of NSAIDs is indicated in the horse known to be at risk of laminitis (i.e. colitis, grain overload, etc.) until approximately 48–72 hours after the animal is no longer showing clinical signs of systemic inflammation/toxemia.

In addition to blocking COX enzyme activity, high doses of NSAIDs recently have been found in other species to block inflammatory pathways including some controlling basic inflammatory gene expression, some of which are upregulated in the early stages of laminitis.[9] COX-2 has recently been shown to be an important mediator in the synapses of sensory neurons; therefore, COX-2 inhibition is likely to not only decrease lamellar inflammation but also decrease central pain sensation. Due to the gastrointestinal (GI) and renal toxicity caused by NSAIDs, close attention must be paid to the animal's history (i.e. a history of gastric/colon ulcers or renal disease), the animal's hydration status, and laboratory work in the critical case to assess renal function.

The four main NSAIDs available to the equine clinician are three nonselective COX-1/COX-2 inhibitors (flunixin meglumine, phenylbutazone [PBZ], and ketoprofen) and one COX-2 selective NSAID (firocoxib). There is some question whether, during treatment with a COX-2 selective NSAID in the peracute phase of laminitis, the vascular inflammation/injury occurring in the lamellae may place the digit at risk of the same vascular accidents (thrombosis leading to myocardial infarction and stroke) that have resulted with the use of COX-2 selective drugs in humans. Thus, until proven otherwise, it may be best to use a nonselective drug in the peracute stage and consider firocoxib in the chronic, long-term case in which the drug's decreased incidence of side effects is more important. Meloxicam is available in countries other than the United States and may be of value due to slight COX-2 selectivity (approximately two- to threefold), which appears to make it a very safe option due to a low incidence of side effects and possibly fewer chances of unwanted vascular events.[10]

In the horse still demonstrating signs of systemic illness with a possible ongoing bacterial toxemia (i.e. colitis), flunixin meglumine is indicated due to its increased efficacy against endotoxemia. In the animal that has a stable hydration status and no indication of renal compromise or intestinal ulceration, the use of high-dose (1.1 mg/kg IV TID) flunixin may be indicated for up to 3–5 days; the authors decrease the dosage after 3 days if the source of bacterial toxemia appears to be resolving. If the lameness does not improve with flunixin, it is indicated to either add other types of analgesics (see constant rate infusion [CRI] below) or possibly lower the flunixin dosage by half and add 4.4 mg/kg PBZ SID.

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In the animal demonstrating renal compromise or clinical signs of GI ulceration, ketoprofen is indicated due to its reported efficacy in ameliorating both endotoxin effects and lameness[4,57] and a markedly increased margin of safety when compared with flunixin or PBZ.[49] Due to the poor response when the drug is only administered BID and the incredible safety of the drug, the BID dosage of ketoprofen can be administered QID (2.2 mg/kg IV QID). Since a "peak and trough" analgesic effect between ketoprofen dosages has been observed in some animals, it may be prudent to administer 2.2 mg/kg PBZ SID in order to have a more consistent NSAID effect.

Analgesia in the chronic case most commonly consists of PBZ therapy, with long-term doses usually in the range of 2.2–4.4 mg/kg/day to avoid complications. An important reason for PBZ's higher incidence of toxicity when compared with the other NSAIDs (almost every reported case of NSAID-related right dorsal colon ulceration has been due to PBZ) is that the drug has a longer half-life and accumulates in the tissues to a much greater degree than either flunixin or ketoprofen. One way to avoid toxicity in animals on long-term PBZ therapy is to cease administration of the drug for 24 hours once every 5–7 days to allow clearance of PBZ from the system. If the animals are too painful to be without an NSAID for 24 hours, either ketoprofen or flunixin may be given without interfering with the clearance of PBZ. Firocoxib has the advantage of once-a-day treatment and is much more protective against GI concerns than PBZ; however, in the authors' opinion, it commonly does not provide an adequate amount of analgesia in many laminitis cases.

Pentoxifylline is reported to be anti-inflammatory due to its "anti-TNF" effect[3]; preliminary results from a study using the carbohydrate model indicate a decreased incidence of lamellar failure in animals with pentoxifylline therapy initiated at the time of intragastric administration of carbohydrate.[17] It is unknown whether the efficacy is due to inhibition of inflammatory signaling, inhibition of matrix metalloproteases, or any hemorrheologic effect (not likely as requires prolonged therapy to achieve this effect in other species). Although digital hypothermia ("cryotherapy") has been demonstrated to protect the lamellae from structural failure when instituted at multiple time point in models of SRL (discussed below),[18,20,24,25] it has a variable effect on inhibition of inflammatory signaling depending on when it has been instituted.[15,16,20] Hypothermia has an inhibitory effect on a broad array of inflammatory molecules (multiple cytokines and chemokines, adhesion molecules, COX-2) if initiated prior to the administration of a carbohydrate overload,[24] but a much more focused effect (primarily IL-6 and COX-2) when initiated later in the disease process (onset of sepsis),[16] and, when instituted at the onset of lameness, no consistent anti-inflammatory effect (although still protective against lamellar failure).[15] Although intravenous lidocaine infusion, similar to that used for treatment of ileus in the equine patient, has been proposed as a treatment to decrease inflammatory signaling in laminitis, no anti-inflammatory and actually some proinflammatory properties of constant rate intravenous lidocaine infusion were reported in a laminitis model.[72]

Therapy Regarding Matrix Metalloprotease Inhibition

Although many practitioners have used tetracyclines in laminitis cases based on the drugs' reported MMP inhibitory properties, preliminary data from an equine study indicate that doxycycline is a poor inhibitor of equine MMPs of interest (MMP-2 and MMP-9) and oxytetracycline inhibits MMPs *in vitro* but was ineffective in treating the disease in a model of laminitis.[33] Additionally, further studies have decreased the interest in MMPs as a therapeutic target including (1) a study demonstrating that MMP-2 and MMP-9 were neither in an active state in lamellar extracts from clinical cases of laminitis[48] and (2) the inability to block lamellar pathology with marimastat—a potent broad-spectrum MMP inhibitor—in using an experimental model of laminitis (Underwood and Pollitt, personal communication).

Drugs Affecting Blood Flow

The majority of drugs that have been introduced for treatment of possible decreased lamellar blood flow have been demonstrated to be ineffective in increasing lamellar blood flow in the horse (i.e. isoxsuprine, pentoxifylline, and nitroglycerin paste). The only drug demonstrated to increase digital blood flow is the phenothiazine tranquilizer acepromazine, which only increases flow for a short period of time (approximately 30 minutes) when administered intramuscularly.[45]

Anticoagulant Therapy

Although there are data supporting the role of platelets in SRL,[2] there is no consensus on the use of any therapies to address this area. There are confounding data from retrospective clinical studies on the efficacy of heparin as a prophylaxis in horses at risk of laminitis.[8,13] Furthermore, experimental treatment with heparin 24 hours after carbohydrate overload (CHO) administration did not ameliorate signs of laminitis or lamellar lesions.[50] One problem with previous heparin studies is that unfractionated heparin was used, which induces autoagglutination of equine red blood cells and becomes lodged in capillaries (including lamellar capillaries); this event may further compromise affected lamellar capillaries in laminitis. Low molecular weight heaparin (LMWH) may be a valuable alternative because it does not cause equine RBC autoagglutination and has recently been reported to reduce the incidence and severity of laminitis in postoperative colic cases.[61] Unfortunately, no further studies have been published on LMWH in laminitis in the past decade.

Analgesic Therapy

The mainstay of analgesic therapy in laminitis cases is still the use of NSAIDs. Whereas PBZ is still the NSAID providing the greatest analgesia in the laminitic horse exhibiting digital pain, flunixin meglumine or COX-2 selective NSAIDs (e.g. firocoxib) may be used if there are ongoing systemic concerns in which the other NSAIDs may either be more effective or less harmful (i.e. concomitant renal disease or GI ulceration, ongoing endotoxemia).

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Due to the common inability to control digital pain with a safe dosing of NSAIDs, CRI of analgesic drugs has been used to supplement NSAID therapy in decreasing the level of pain suffered by hospitalized laminitis patients. The use of "Pentafusion" (combination of ketamine, morphine, lidocaine, detomidine, and acepromazine; Eric Abrahamsen, BEVA Proceedings, 2005) has been described for use in severely painful cases of laminitis. The CRI is a valuable addition to laminitis therapy because it takes some of the humane concerns away from the owner and veterinarian treating an animal in severe pain. Some of these drugs also affect GI motility; therefore, the animals must be monitored closely for large intestinal motility/impaction. Additionally, the animals may become unstable or overly sedate if the CRI rate is excessive. For a more focused approach, an epidural can be considered for analgesia in hindlimb laminitis.

Therapy to Limit Structural Failure of the Lamellae

The obvious overarching goal of therapies for the horse at risk of or suffering from laminitis is limiting the structural failure of the lamellae. The only therapy to consistently inhibit—or limit—lamellar structural failure in both the research and clinical setting is digital hypothermia (also termed cryotherapy).[19,25,41] Although original work demonstrating efficacy of hypothermia maintained the horses' forelimbs in an ice/water slurry to the level of the carpus,[20] more recent work has demonstrated that immersion of the foot up to the level of the pastern region is adequate to keep the hoof temperature less than 10 °C.[18] Although many clinicians use 5-L intravenous fluid bags taped to the limbs to maintain the digits in the ice slurry, commercial alternatives exist (e.g. Ice Spa Pro, Soft-Ride, Inc.). Continuous hypothermia in this manner has been demonstrated to protect the lamellae from structural failure even when hypothermia is not initiated until the onset of lameness.[25] Although most experimental studies demonstrating efficacy of digital hypothermia have been performed in models of SRL, the same protective effect was recently demonstrated to occur when hypothermia was assessed in EHC model of endocrinopathic laminitis (Belknap and van Eps, unpublished data). In the clinical setting, digital hypothermia is most commonly used as a prophylactic therapy in the septic equid at risk of laminitis. In a multicenter retrospective clinical study of the use of digital hypothermia as a prophylactic therapy against laminitis in 130 equine enterocolitis cases, horses treated with digital hypothermia were ten times less likely to develop laminitis than those not treated with hypothermia.[41] In that study, there was a trend for a worse prognosis if hypothermia were used intermittently than if not used at all (S. Holcombe, personal communication), indicating that it is very important that the digits need to be continuously maintained in the hypothermic state. Although it varies between animals, ice usually needs to be replenished every 1–2 hours to maintain the hypothermic state. Many practitioners only maintain the front digits in ice due to the increased incidence of laminitis in the forelimbs; however, it is best to maintain all four limbs in ice if possible. Although no studies have been performed to ascertain the optimal time period to maintain the digits in the ice slurry, most clinicians maintain animals at risk of SRL in ice for 24–48 hours past the abatement of clinical signs of sepsis.

Hoof Care in the Treatment of Acute and Early Chronic Laminitis

In the acute and early chronic laminitis case, it is likely that lamellar instability remains a prominent factor. Ideally, in horses with severe acute and early chronic laminitis, the force of the horse's weight on the feet should be reduced. To this end, slings have been used to enforce partial weight-bearing, but availability and inadequate tolerance by the horse frequently result in failure of this effort. Therefore, many treatment measures are aimed directly at the foot. The two main objectives of hoof care are to redistribute the force of weight-bearing away from the hoof wall and to decrease the extensor moment about the DIP joint.

Several measures of varying efficacy attempt to redirect weight-bearing away from the wall by recruiting the frog, bars, and all or part of the sole to bear weight. However, as discussed in Chapter 8, it must be remembered that the physiological role of the sole in weight-bearing is not fully understood. The amount of the ground surface that can be recruited to bear weight is highly variable between horses. In general, once the distal phalanx has displaced, pressure over the sole under the displaced distal phalanx increases discomfort. Thus, the clinician must weigh the benefits of possibly decreasing lamellar stress by applying solar support with the risk of causing excessive solar pressure and worsening the pain (and possibly the pathologic process). Before considering other measures, the clinician must decide whether to leave shoes on the feet if the horse is shod. Shoes on firm surfaces concentrate stress around the perimeter of the foot (e.g. the hoof wall and lamellae), and therefore removing them will likely be beneficial by removing this effect. Removing shoes also makes other therapeutic measures more feasible, including recruiting other parts of the ground surface of the foot for weight-bearing and modifying break-over. The potential disadvantage of removing shoes in horses with laminitis is that the removal process itself may cause additional trauma to the lamellae.

The simplest way to recruit the sole, frog, and bars for weight-bearing is to place the horse on bedding material that readily conforms to the shape of the foot. In this regard, sand is significantly better than shavings. Peat has also been used. Deep deformable substrates also permit the horse to adjust the angle of its foot to optimize comfort. However, the deep deformable substrates are not selective in how pressure is applied to the ground surface of the foot. Various devices and materials have been placed under the frog, sole, and bars to recruit varying amounts of weight-bearing. These include rolled gauze, Lilly pads, silicone putty, Styrofoam insulation board (usually 2-inch board), closed cell foam (1.5 inches thick, 4 lbs/ft^3 density), and commercial pad systems such as the Soft-Ride boots (Soft-Ride, Inc., Vermillion, OH). The closed cell foam has the advantages of not crushing like Styrofoam does, and it can be cut to bevel the ground surface to move the break-over palmarad. The softer materials may offer the additional

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advantage of softening impact during locomotion. Rigid materials may transfer weight-bearing more effectively but are discouraged because they are more likely to cause focal pressure under the margin of the distal phalanx and pain and should therefore be generally avoided or only used with caution.

The moment or torque about the DIP joint at rest can be decreased by elevating the heels. This decreases the tension in the DDFT and therefore the tension in the dorsal lamellae. This is more likely to benefit horses that have or are about to develop capsular rotation and is unlikely to help (and may hurt) those that have or are prone to distal displacement. The arm of the extensor moment at break-over can be shortened by moving the point of break-over palmar to the dorsal margin of the toe (Chapter 8). This may be done by rasping the ground surface of the hoof wall while being careful to preserve the integrity of the white line or incorporating it into a supporting device. Commercial plastic cuff and pad combinations conveniently combine a wedged heel and eased break-over (i.e. the NANRIC Ultimate, NANRIC Co., Lawrenceburg, KY).

Treatment must be titrated to the severity of the disease. Currently it is not routinely possible to assess the degree of injury to the lamellae, so the severity of the disease for each type of displacement is usually determined by clinical parameters such as heart rate, the severity of the lameness, response to treatment, and radiographic changes. In the early stages of therapy, the intensity of therapy initiated depends on the severity of clinical signs (i.e. not all horses need all of the measures described). However, in the horse that has undergone treatment and appears to be improving, it is advisable to gradually discontinue treatments, withdrawing one treatment measure at a time followed by 2–3 days of observation. In more mildly affected horses, treatment may be discontinued over 1–2 weeks. However, in severely affected horses, the treatment should be tapered off over 6–8 weeks as the treatment switches to that for the chronic laminitic patient.

Treatment of Active and Stable Chronic Laminitis

The objectives for maintaining stability of the distal phalanx and pain control are similar to that of the acute and early laminitic horse. An additional goal is to restore the relationship between the hoof capsule and the distal phalanx. The management varies with the type of displacement, severity of lameness, the way in which the horse moves, and stability of the distal phalanx. The treatment of dorsal capsular rotation, symmetrical distal displacement, and asymmetrical displacement are considered separately.

Hoof Care in the Treatment of Horses with Rotation

The challenge of treating horses with rotation, usually both capsular and phalangeal, is to realign the phalangeal axis while simultaneously realigning the hoof wall with the parietal surface of the distal phalanx (while also controlling pain). Complications are common and must be managed as they occur. The mainstay of treatment is hoof care, but various surgical techniques may be required.

Trimming and shoeing of the foot should be planned based on radiographic observations.[54,62] Dorsopalmar and mediolateral radiographs should both be taken (as described earlier) prior to shoeing. The guiding principles are as follows:

1. Realign the phalangeal axis by trimming
2. Preserve the thickness of the sole
3. Move the break-over in a palmar/plantar direction
4. Reduce the tension in the DDFT
5. Recruit weight-bearing by the ground surface of the foot depending on tolerance and type of shoeing to be used

The trim should aim to restore the angle that the solar margin of the distal phalanx forms with the ground to between 0° and 5° (in severe cases, it may be preferable to aim for 5° vs. 0°). At the same time, the sole depth should be preserved to at least 15 mm wherever possible. In horses in which the dorsal sole is greater than 15 mm in depth, the trim is along a single plane from the dorsal margin of the sole all the way to the heels (Figure 4.74A). However, in most horses with rotation, the depth of the dorsal sole is significantly less than 15 mm due to the distally displaced dorsodistal aspect of the distal phalanx.

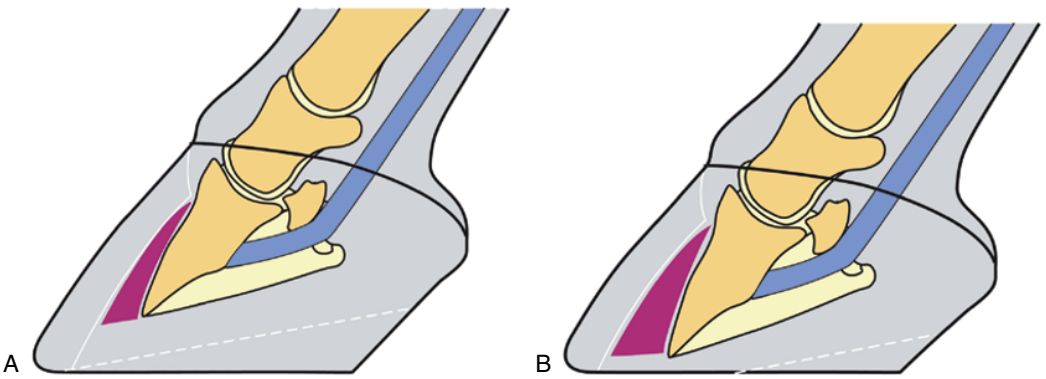


Figure 4.74. (A) When the depth of the sole exceeds approximately 15 mm across the sole, the alignment of the distal phalanx with the solar surface of the foot can be achieved by trimming the entire ground surface of the foot on a single plane (dashed line). (B) When the dorsal sole is less than 15 mm, which is common in horses with rotation, the goal is to preserve the dorsal sole, yet still realign the distal phalanx for shoeing. The palmar ground surface is trimmed (dashed line) on a different plane to the dorsal half (which is not trimmed).

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To attempt to realign the distal phalanx with the ground surface with the sole by trimming, the ground surface of the foot to one plane would inevitably decrease the thickness of the dorsal sole further. Therefore, only that portion of the foot with greater than 15 mm sole depth is trimmed, which is usually the palmar 50%–70% of the sole (Figure 4.74B). This results in two separate planes for the dorsal and palmar portions of the foot (Figure 4.74B). Depending on the shoeing technique to be employed, the dorsal plane of the foot may be brought into line with the palmar plane at the time of shoeing, usually by filling the gap with a synthetic polymer. The plane for trimming can be identified on the radiograph by drawing a line parallel to and 15 mm distal to the solar margin of the distal phalanx (Figure 4.72).

Some horses, particularly those with mild rotation and sufficient sole depth, may be managed with trimming and maintaining them on a soft ground substrate without shoeing.

Once the foot has been trimmed, the clinician must decide whether or not to shoe the horse, and if so, which type of shoe to use and how to attach it. The shoe may be attached to the foot with nails, directly with glue, with glue and a cuff, or, in some instances, with cast material (i.e. the wooden shoe). The type of shoe and the manner in which it is attached vary with duration and severity of diseases and clinician experience and preference. For less severely affected horses, most frequently, shoes are nailed to the foot in the traditional manner. However, when nailing the shoe in place is considered too painful or there is insufficient hoof wall to which to nail, a less traumatic technique using a combination of adhesives, casting tape, and screws may be used. When selecting a shoe, it is important to remember that there are several underlying principles and numerous ways of achieving them. The underlying principles or goals of shoeing include:

1. Moving the center of pressure
2. Changing the area of distribution of force
3. Decreasing the shock of impact
4. Enhancing ease of movement
5. Protecting parts of the foot

Enhancing ease of movement involves decreasing the moment arm about the DIP joint and smoothing out break-over. This is most beneficial in the dorsopalmar plane, in which the objective is accomplished by rolling the toe, setting the shoe back, squaring the toe, or using an open toe shoe. However, to a lesser extent, break-over in a medial or lateral direction can be eased by the use of rails, rounding the medial and lateral branches of the shoe, or using a Steward clog (wooden shoe) with beveled sides.

For horses with rotation, the center of pressure may be moved in a palmar direction in relation to the center of the ground surface of the foot and toward the center of rotation of the DIP joint by using extensions, by recruiting more ground surface to bear weight on the palmar ground surface, or by elevating the heels. In doing so, weight-bearing can be moved away from the most affected laminae.

Increasing the area of distribution of weight across part or all of ground surface of the foot is theoretically performed to transfer weight-bearing from affected lamellae to the sole, bars, and frog to decrease stress in the affected lamellae. However, the role of the sole in weight-bearing to spare weight-bearing by the wall has been questioned (see therapeutic shoeing in Chapter 8). Regardless of the theoretical considerations, some horses appear to benefit greatly from sole support. Support of the ground surface of the foot is most readily accomplished with the use of synthetic polymers such as polymerized silicone (two-part putties such as the impression material [Equine Digit Support System (EDSS), Penrose, CO] or advanced cushion support [NANRIC, Lawrenceburg, KY]) or polyurethane ("pour-in pads") in the space between the branches of the shoe. However, support of the ground surface is also still approached by more traditional means such as pads and heart-bar shoes.

Several shoe types have been used to address these goals including regular keg shoes, egg-bar shoes, reverse shoes, heart-bar shoes, four-point rail shoes, and wooden shoes/clogs. Each deserves consideration to examine how it accomplishes the goals and to discuss its advantages and limitations.

The keg shoe must be modified to be effective in the treatment of active and stable chronic laminitis. Break-over in the dorsopalmar plane can be eased by forging the toe of the shoe to square and roll it and setting the shoe back. Easing break-over in the medial and lateral directions can be achieved to a limited extent by rounding/beveling the outside rims of the branches of the shoe. Heel elevation can be added as needed by using a shoe with a built-in wedge or using a wedge pad, frequently a wedge rim pad. The wedge rim pad allows incorporation of as much of the ground surface of the foot as needed for weight-bearing using a synthetic polymer in the concavity of the foot between the branches of the shoe (and rim pads).

The egg-bar shoe (Figure 4.75) is probably the most common shoe used because it is readily available and many farriers and veterinarians are familiar with its use in other circumstances. It can be modified in all the same ways as the keg shoe, and it has the added benefit of the



Figure 4.75. Egg-bar shoe.

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bar that extends in a palmar direction. The palmarly extended nature of the bar causes it to act as a mild wedge when the horse stands on a soft surface by acting like a "snowshoe" and inhibiting sinking of the heel. This decreases tension in the DDFT and hence theoretically decreases the tension placed on the dorsal lamellae.

The reverse shoe combines the advantages of the egg-bar shoe with an open toe. The open toe theoretically offers superior easement of break-over in the dorsopalmar plane when compared with a closed toe shoe. However, the open toe also has the potential disadvantages of concentrating stress at the dorsal margin of the branches and not protecting the toe in horses with dorsal solar concerns such as solar prolapse dorsal to the frog. While this shoe currently appears to have fallen out of favor, it may offer a simple solution, particularly when used in horses in which access is needed to the dorsal aspect of the sole.

The heart-bar shoe (Figure 4.76) has been the traditional mainstay in the treatment of horses with laminitis.[12] The heart bar is a V-shaped piece of bar stock that has been welded between the branches of the shoe to cover the frog. Alternatively, the heart bar may be forged into the shoe, either by hand or during manufacture. As such, it can be modified in the same manner as the keg shoe. The original rationale behind employing this shoe is that it recruits weight-bearing by the frog and hence decreases weight-bearing by the lamellae. An alternative explanation for the effectiveness of this shoe is that it increases the ground surface area of the shoe in the palmar half of the foot and hence either acts as a mild wedge or at least decreases the descent of the heels into a soft substrate.

The overall principles involved in trimming and setting the shoe are similar to those of the keg shoe. However, care must be taken to ensure that there is not undue pressure from the heart bar on the frog that would result in bruising and increase lameness. Because of this concern, many farriers leave a small space between the frog and heart bar and apply a synthetic polymer underneath the heart bar; synthetic polymer can also be placed alongside the heart bar between the branches of the shoe. The heart bar may also be used in conjunction with an open toe or egg-bar shoe.

The four-point rail shoe is based on the pattern of an open-heeled shoe.[62] The toe of the shoe is squared, aggressively rolled, and broadened. Additionally, narrow wedges, called rails, are applied to the axial side of the ground surface of each branch to raise the heels. Thus, this shoe by design incorporates both ease of break-over at the toe and elevation of the heel. The use of the rails applied to the axial side of the branch of the shoe also provides improved medial and lateral break-over compared with other designs of shoe used for laminitis.

While the shoe may be hand forged or modified (Figure 4.77), the majority of those used are one of two commercially available designs: the Aluminum Four-Point



Figure 4.77. A hand-forged aluminum four-point rail shoe. Note the squaring and rolling of the toe. *Source:* Shoe courtesy of Neal Baggett.

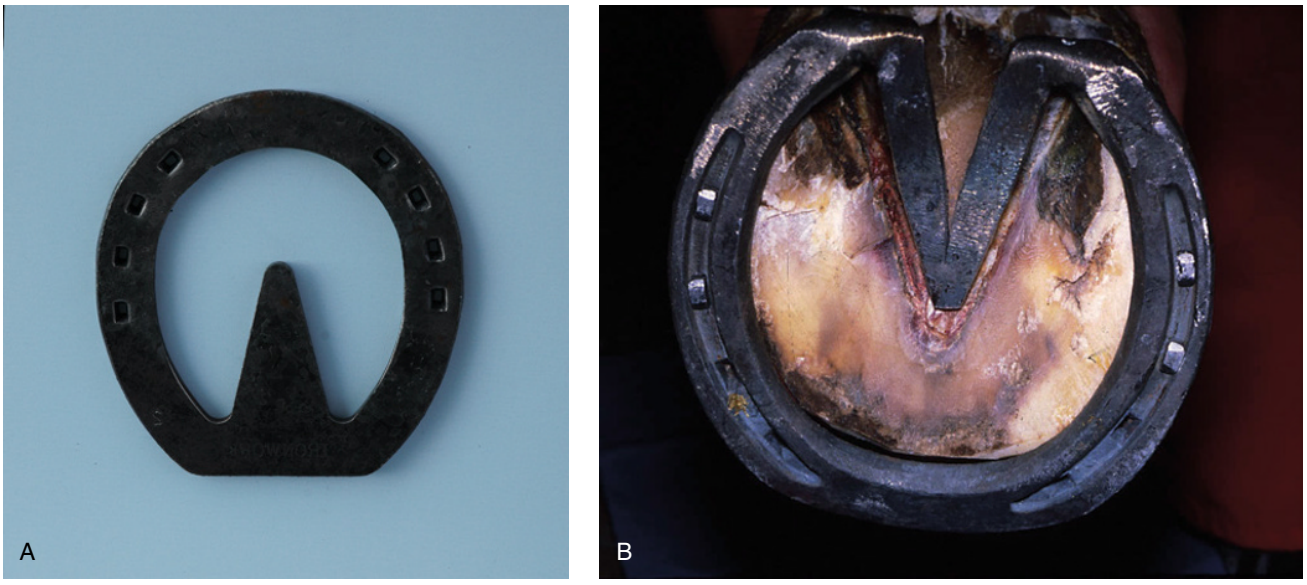


Figure 4.76. Heart-bar shoe (A) with correct placement on the foot (B).

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Rail Shoe and the EDSS (Figure 4.78). The Aluminum Four-Point Rail Shoe is manufactured to incorporate all the features of the four-point rail shoe in a single unit, with incorporation of the rail into the shoe (NANRIC Co., Lawrenceburg, KY). The EDSS is marketed in the form of a kit (Figure 4.78) (Equine Digit Support System, Inc. Penrose, CO). The kit includes a flat shoe with the break-over previously described and a range of rails of different heights that can be attached to the ground surface of the shoe. Additionally, the kit contains pads, frog-shaped inserts, and silicone putty.

The advantage of the Aluminum Four-Point Rail Shoe is simplicity of construction and application; recruitment of the sole, frog, and bars to bear weight is achieved with a synthetic polymer. The disadvantage of this shoe compared with the EDSS is that the rails are of fixed height (although they can be ground down as needed) and cannot be readily adjusted once the shoe is on the foot. The advantages of the EDSS are that the rails can be applied after the shoe is on the foot and the height of the rails can be selected/adjusted. The kit is designed to recruit weight-bearing by applying a pad between the shoe and foot in conjunction with silicone putty placed between the pad and foot; plastic frog inserts can be screwed to the ground surface of the pad. Alternatively, the shoe and rails can be used with a synthetic polymer placed in the concavity of the foot and between the branches of the shoe instead of the pad and frog insert.

The Steward clog/wooden shoe (Figure 4.79) as originally described is cut from 0.75-inch plywood to correspond to the size of the foot, with a squared toe and a 45° bevel all around its perimeter so that the ground surface is smaller than the foot surface.[67] The thickness of the shoe may be increased by the addition of an additional three-quarters inch of plywood or rubber similarly beveled and added to the ground surface of the shoe. The shoe is set back from the toe so that the dorsal margin of the shoe is approximately 1.5 inches dorsal to the apex of the frog. Silicone putty may be applied between the palmar aspect of the hoof and the shoe and the shoe is attached with screws. Usually, an adhesive or a 2-inch roll of casting tape is applied over the screw heads and distal wall to improve stability. Furthermore, the shoe is open to almost infinite customization, more common forms of which include adding a wedge or recessing the dorsal solar surface for horses with solar prolapse. This shoe is simple to make and apply. By varying its customization, it can achieve all of the goals previously outlined. It is likely that the properties of the wood itself offer some advantages in relation to comfort during weight-bearing. In addition, it has been postulated that the thickness of the shoe may have at least two other advantages: (1) it permits greater adjustability for the point of and curvature of break-over, and (2) it has been postulated that the height may elevate the forelimbs sufficiently to transfer weight to the hindlimbs. Although the shoe is primarily used on forelimbs, Steward has used a shorter variety of the clog on hindlimbs (Mike Steward, personal communication).



Figure 4.79. A commercially available ethylene vinyl acetate (EVA)/wood laminated clog shoe. *Source*: Equicast, Inc.





Figure 4.78. (A) The Equine Digital Support System is a kit that provides a convenient way to adjust the break-over, support the ground surface of the foot, and elevate the heels. (B) As an alternative to using all the components of the kit, ground support may also be provided with polymerized silicone.

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The Sigafoos Series II shoe (Figure 4.80) is a kit that is composed of a cuff bonded to a rim that is attached to an aluminum plate that covers the ground surface of the foot (Sound Horse Technologies LLC, Unionville, PA). The aluminum plates may be flat or wedged and have a urethane bonding surface to which additional wedges may be bonded. This shoe can be highly customized to achieve the principal objectives. The attachment of the shoe with a cuff is atraumatic compared with nailing a shoe on, and placement of the acrylic on the outside of the wall causes less harm to the wall than when the acrylic is placed on the ground surface of the wall. Because the cuffed shoes follow the perimeter of the hoof capsule, they may require more extensive modification to set the toe back compared with other methods of shoe application.

The choice of shoe for each case is in large part personal preference based on cost, experience, and availability. It is important to emphasize principles over technique. There are several complications common to various shoeing techniques. Where weight-bearing has been relieved at the toe and concentrated at the quarters, the wall at the quarters is likely to become thinner and less resilient. Additionally, prolonged heel elevation frequently leads to heel contraction. Finally, prolonged attachment of the shoe with acrylics leads to deterioration in the mechanical properties of the wall, particularly if it is applied to the ground surface of the wall.

The application of the general shoeing principles must be titrated to the specific symptoms of the individual horse. The less damaged the lamellae, the less aggressively eased break-over, ground support, and heel elevation are required. Improving the ease of break-over benefits any horse with laminitis. The amount of area across the ground surface of the foot that should be used to distribute weight-bearing is subjective. Pressure should be limited where compression of the underlying dermis may occur between the sole and an unstable or displaced distal phalanx. Otherwise, the comfort of the horse and the perceived instability of the distal phalanx are the best indicators. The requirement for (and degree of) heel elevation is in part similarly determined by the discomfort of the animal, but also by the way the horse lands. Adjusting the heel height so that the horse lands slightly heel first is likely to result in the greatest comfort and stability of the distal phalanx.



Figure 4.80. Sigafoos Series II shoe.

The treatment time scale is highly variable and based on the severity of the original disease, recurrence of the disease, and the development of complications. However, some generalizations can be made. A more severely affected horse that is amenable to treatment and improves in an uncomplicated manner is usually shod somewhere between 3 and 6 weeks after the onset of the acute disease with eased break-over, ground surface support, and heel elevation as needed. It is desirable to remove heel elevation as soon as possible after the horse is comfortable landing with a flat foot to limit contraction of the heels, usually between 3 and 6 months. Sole support may be removed, usually in a gradual manner, between 4 and 8 months. The most successful cases may return to near normal shoeing or barefoot trimming between 8 and 12 months. Obviously, less severely affected horses that do not develop complications should recover correspondingly quicker. Horses that suffer recurrent acute episodes or develop complications may take much longer to respond or never respond satisfactorily.

Treatment of Chronic Laminitis with Distal Displacement

Horses that suffer distal displacement present different challenges from those with dorsal rotation. First, treatment directed at the acute and early chronic disease frequently lasts considerably longer than that for horses with rotation. Easement of break-over and ground surface support are both potentially beneficial and achieved in the same manner as previously described. However, there is no rationale or perceived benefit to elevating the heels; this commonly makes these horses worse. The wooden shoe/clog has gained a great deal of popularity in the treatment of distal displacement in the forelimbs.

Treatment of Chronic Laminitis with Unilateral Distal Displacement (Medial or Lateral Rotation)

The transition between the hoof care used to treat horses with acute and early chronic disease to that of stable chronic laminitis due to unilateral displacement is more blurred than in the previous scenarios because there is much less experience in treating this condition and complications, particularly separation of the coronary band, are common. However, in line with the overall objective to redirect the load away from the most damaged area of the lamellae, the logical approach is to attempt to redirect the load to the opposite side of the foot in conjunction with ground surface support.[55] This can be accomplished by either placing a 4- to 8-mm extension (concentrating ground support on the extended side; Figure 4.81) or placing a thin wedge on the side of the foot opposite the displacement or changing the weight-bearing area under the foot asymmetrically. Of these treatments, the wedge is the most aggressive and does not appear to be necessary and as such is best avoided. Therefore, starting with an extension or modifying ground surface support is suggested. The application of a wedge to the unaffected side of the foot appears counterintuitive if only the radiographic

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position of the distal phalanx within the hoof capsule is considered because it is natural to try and restore the alignment of the DIP joint. However, consideration of the stresses within the lamellae suggests that restoring the symmetry of the DIP joint is likely to be counterproductive. Unilateral distal displacement frequently occurs with some rotation, in which circumstances heel elevation may also be warranted. While this technique has been beneficial in some horses, more experience is required before firm recommendations are possible.



Figure 4.81. This horse has medial asymmetrical distal displacement, evidenced by disparate growth of the medial and lateral walls. A wooden shoe has been positioned to act as a lateral extension to increase weight-bearing by the healthier lateral side of the foot to decrease pain from compression of the sole and tension in the lamellae medially. *Source*: Courtesy Dr. Stephen O'Grady.

Casting and Splinting

Casting the distal limb is beneficial in horses with acute and early chronic laminitis but is particularly valuable in the treatment of chronic cases with unilateral complications such as subsolar abscessation (Figure 4.82). Half-limb casts, half-limb casts with transfixation pins, and open soled half-limb casts have been used.[6] However, foot or pastern casts may also provide some stability to the lamellae.[11] If fit snugly at the proximal end of the metacarpus, the half-limb casts potentially offer some weight sparing by the digit. However, transfixation pin casts are likely to provide dramatically greater load sparing by the foot. Additionally, by decreasing movement within the distal limb, casts may decrease the pain as the horse moves around a stall. Open soled casts offer the advantage that the ground surface of the foot can be inspected and treated while the foot is in the cast and that load bearing by the ground surface of the foot may be more selectively applied using synthetic polymers. If casts are only placed on one limb, it is important to place some type of support on the contralateral limb to both support the foot (which is usually also affected with chronic laminitis) and equalize the height of the two limbs. Splints offer some of the advantages of immobilization, but offer no relief from load bearing.

Deep Digital Flexor Tenotomy

Tenotomy of the DDFT abolishes or greatly reduces or abolishes the flexor moment about the DIP joint. The direct result is that the center of pressure should move in a palmar direction to a point directly distal to the center of rotation of the DIP joint. The benefits are that it decreases the tension within the dorsal lamellae and compression on the dorsal sole, thus allowing realignment of the distal phalanx with the ground without exacerbating the deleterious forces on the dorsal lamellae and sole (Figure 4.82B and C). This is usually accompanied by decreased pain and increased sole growth dorsally. The disadvantages are that the toe of the foot may become elevated from the ground as the horse rocks back or ambulates and that mild to moderate subluxation may occur in the DIP joint

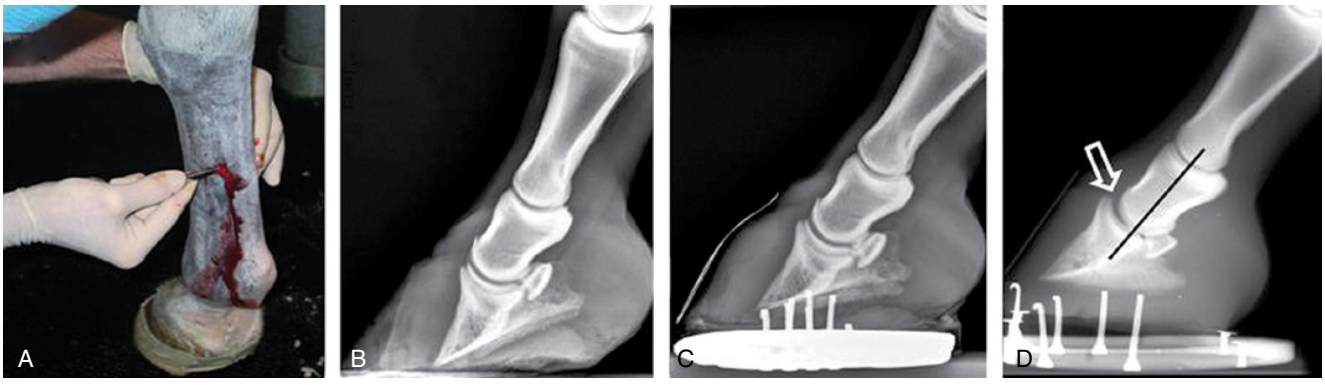


Figure 4.82. Tenotomy of the DDFT is most commonly performed standing at the mid-cannon region using a guarded bistoury (A). The tenotomy allows realignment of the rotated distal phalanx (B) with the ground surface (note realignment of the foot in C after 6 weeks). Subluxation of the DIP can occur following the procedure (D), characterized by dorsal displacement of the extensor process of the distal phalanx away from middle phalanx (arrow) and by caudal displacement of the distal articular surface of the middle phalanx so that a line bisecting the middle and proximal phalanges does not bisect the middle of the articular surface of the distal phalanx (black line).

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(Figure 4.82D). Based on these responses, the surgery is primarily indicated in horses with:

1. Early chronic laminitis that continues to rotate despite all other measures taken
2. Intractable pain originating from the dorsal sole and wall despite stabilization and shoeing
3. Secondary flexural deformities

It does not appear to consistently benefit horses with distal displacement and rarely if ever benefits the horse with unilateral distal displacement of the distal phalanx.

The surgery may be performed in the mid-metacarpal region (Figure 4.82A) or in the midpastern region.[1,69] The surgery is easier to perform in the mid-metacarpal region. Additionally, should a second tenotomy be necessary, it is preferable to perform the proximal one first because adhesions may limit the effectiveness of the second surgery in the metacarpal region if the first surgery was performed in the pastern. Tenotomy in the midpastern appears to provide greater mobility of the foot about the DIP joint, but may also cause more instability of the joint.

The tendon may be cut immediately before or after corrective trimming and shoeing is performed (it is probably best to perform the surgery first to address possible DIP subluxation following DDFT). Because tissue repair at the tenotomy site occurs fairly rapidly and will inhibit any further realignment, it is imperative to obtain the best realignment of the distal phalanx with the ground and phalanges as soon as possible after surgery (i.e. within hours to days). To counter the disadvantages of the surgery, it is advisable to perform radiographs while shoeing immediately following tenotomy of the DDFT to assess both the adequacy of the realignment of the distal phalanx to the ground surface and, importantly, the degree of subluxation of the DIP joint (indicated by both dorsal displacement of the extensor process of the distal phalanx away from the middle phalanx [arrow, Figure 4.82D] and by palmar displacement of distal articular surface of the middle phalanx in relationship to the articular surface of the distal phalanx [line, Figure 4.82D]). Application of increasing degrees of heel elevation can be assessed radiographically until the subluxation is resolved. If radiographic assessment is not available post-tenotomy, the horse should be shod with mild heel extension and elevation (approximately 3°).

Drainage and Debridement

Digital sepsis is a well-recognized complication associated with laminitis. Drainage may occur at the coronary band or through the dorsal sole. In most horses, the infection is confined to the soft tissues of the hoof, but, occasionally, the infection may involve the distal phalanx. If the hoof capsule of the sole is removed, the solar dermis usually prolapses, and the prolapsed tissue is extremely sensitive to pressure (Figure 4.83). Therefore, when subsolar sepsis is present, it is advisable to create an avenue for drainage through the distal wall rather than the sole whenever possible. If solar drainage is performed, very small portals should be made at the opposite sides of the affected area for drainage and to allow insertion of a catheter or teat cannula for lavage with saturated Epsom salts (in contrast to removing excessive sole; Figure 4.83).

Sepsis of the distal phalanx can be difficult to both diagnose and treat. It is difficult to diagnose because the radiographic changes in the distal phalanx associated with sepsis closely resemble those associated with prolonged inflammation, and therefore, septic and nonseptic inflammation is very hard to distinguish. In the majority of subsolar sepsis cases, the sepsis does not involve the bone. However, direct contact of bone after inserting a probe in a draining track conclusively identifies exposure of the bone to sepsis and is highly suggestive of septic osteitis (combined with radiographs indicative of severe focal lysis at point of probe contact).

In the authors' experience, septic osteitis of the distal phalanx is much more refractory to treatment in horses with laminitis than in horses in which the sepsis occurred for another reason. Additionally, surgery, usually consisting of curettage of the suspect bone, exposes the surface of the distal phalanx in a horse that did not have septic osteitis and increases the likelihood that the horse will develop septic osteitis. Therefore, infection should be treated as superficial unless drainage can be directly linked to the bone or prolonged treatment fails to resolve it and septic osteitis is the best explanation for the con-



Figure 4.83. Painful granulation tissue (arrow) covered with fibrin exists due to prolapse of the dermis secondary to displacement of the distal phalanx and excessive trimming of the sole. Removal of the sole from the foot in horses with chronic laminitis should be minimized to avoid this painful complication.

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tinued drainage. In the severely underrun subsolar abscess, one author (JKB) lavages the foot with saturated Epsom salts delivered through a catheter or teat cannula through small portals either in the distal hoof wall (preferred) or sole. If there is moderate to severe capsular rotation also present in the affected foot, a deep digital flexor tenotomy with "derotation" of the distal phalanx is also indicated to improve healing and decrease the chance of recurrence by decreasing the pressure on the affected dorsodistal aspect of the distal phalanx and the adjacent subsolar/solar tissue.

HOOF WALL RESECTIONS

Full dorsal hoof wall resections were at one time widely advocated to decrease the pressure of the wall on the coronary band, debride necrotic material, and encourage the realignment of the distal phalanx.[12] They accomplish these goals to some degree, including enhancing growth of new wall at the coronary band and increasing the likelihood that the newly formed wall will conform to the parietal surface of the distal phalanx. However, resecting the dorsal wall removes any support that the dorsal wall supplied to the distal phalanx, increases the vertical stresses in the wall at the margins of the resection, and causes further instability by removing circumferential tension that spans the dorsal hoof capsule connecting quarter to quarter. Consequently, the remaining dorsal quarters are more likely to become distracted from the underlying tissues. Therefore, more limited/partial hoof wall resections are more commonly performed than total dorsal hoof wall resections to minimize the loss of support to the distal phalanx and stability of the adjacent wall observed in more extensive resections. They are currently most commonly performed to debride necrotic tissues and remove the lamellar "wedge" to encourage new hoof wall growth to follow the contour of the distal phalanx.

CORONARY BAND GROOVING AND RESECTION

Coronary band grooving is designed to take pressure off the coronary band to increase the rate of new hoof wall growth (Figure 4.84).[64] It also creates a discontinuity between older, more distal hoof wall and the newer proximal wall so that the distal wall is less likely to distract the new hoof away from the distal phalanx. The groove is created in the dorsal hoof wall at the level of the base of the extensor process and extends through the full thickness of the stratum medium of the wall from one toe–quarter junction to the other. A modification of this technique involves creation of the groove as described above and then removing all of the stratum medium proximal to the groove to the level of the coronet. In the authors' experience, results with this technique are highly variable.

Resection of the cornified layer of hoof at the coronary band is probably most frequently performed when the hoof wall has separated from the coronary band. In these circumstances, it is done to decrease the chafing by the separated—and therefore somewhat mobile—hoof wall that damages underlying viable germinal tissue and enhance drainage of exudate that has accumulated under the separated tissues. It is advisable to bevel the proximal margin of the remaining hoof wall so that it is thinnest proximally (immediately distal to the coronary band) to minimize the pressure ridge that develops and impinges the underlying dermis of the coronary band and proximal lamellae. The indications and optimal timing for this procedure have not been determined.



Figure 4.84. Grooving of the proximal dorsal hoof wall immediately distal to the coronary band to encourage dorsal hoof wall growth and mechanically dissociate new hoof wall growth from the older distal wall.

Prognosis

There are numerous reasons why treatment of horses with laminitis is unsuccessful, but the three most important reasons are the severity of the original pathology, type of displacement, and severity of the clinical signs.[5,38,55,56] These factors are likely to determine the continued course of the disease and the development of complications. The prognosis following displacement is always considered guarded to poor.

Regarding the different types of displacement, rotation is considered to have a more favorable prognosis than either symmetrical or asymmetrical distal displacement. Increased experience with the treatment of asymmetrical distal displacement suggests that the prognosis for this condition is better than that with symmetrical displacement so long as separation at the coronary band has not occurred. Two radiographic criteria have been documented to determine prognosis: the degree of rotation and the distance from the proximal margin of the extensor process of the distal phalanx to the firm proximal margin of the hoof capsule (immediately distal to the coronet, distance also termed the "founder distance").[14,68] Capsular rotation greater than 11.5° predicted poor survival and capsular rotation less than 5.5° predicted return to performance. A coronet-to-extensor-process distance of greater than 15.2 mm is indicative of poor survival. Both of these reports are dated, and with recent advances in the treatment of this disease, their prognostic value should be reassessed. Additionally, the thickness of the sole and the angle that the solar margin of the distal

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phalanx subtends with the ground may be beneficial in assessing the prognosis for success; thinner soles and greater angles of the solar margin of the distal phalanx are anecdotally associated with decreased prognosis. Finally, financial constraints of the owner and the failure of the owners to follow recommendations, particularly advice regarding exercise and maintaining appointments, are likely to decrease the success of treatment.

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THE PASTERN

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The proximal interphalangeal (PIP) joint or pastern joint is a diarthrodial joint, which is formed from the distal aspect of the proximal phalanx (P1) and the proximal aspect of the middle phalanx (P2). The pastern region is bounded dorsally by the common or long digital extensor tendon together with the dorsal branches of the suspensory ligament. Palmar/plantar support structures of the pastern region are formed by the distal sesamoidean ligaments (DSLs) (straight, oblique, cruciate, and short), superficial digital flexor tendon (SDFT), deep digital flexor tendon (DDFT), and proximal and distal digital annular ligaments within the digital flexor tendon sheath (DFTS). The medial and lateral collateral ligaments provide support in the sagittal plane.

Abnormalities such as dorsal swelling or bony enlargement in the pastern region are often obvious due to minimal soft tissue in the area. The severity of lameness from the pastern region ranges from subtle to severe depending on the injury. Generally, injuries such as fractures that involve the PIP joint or tendinitis within the DFTS cause obvious lameness, whereas lameness due to early osteoarthritis (OA) of the PIP joint or strains of the DSLs may be mild. Pain and lameness in the pastern region is often exacerbated by distal limb flexion or lunging the horse with the affected limb on the inside of the circle.

Complete analgesia of the pastern region by perineural analgesia is variable. Occasionally, horses will have complete analgesia after a palmar digital (PD) nerve block,[9] most will not have complete analgesia until a basisesamoid or abaxial nerve block has been performed, and still others may require a low four-point nerve block. When blocking the pastern region, horses with suspected stress fractures of P1 should not be blocked to avoid further displacement of the fracture.

Radiography is important for the initial characterization of the injury. Ultrasonographic evaluation of the pastern is an integral part of characterizing the extent of any soft tissue injury. Additionally, nuclear scintigraphy, computed tomography (CT), magnetic resonance imaging (MRI), or tenoscopy of the DFTS may all be important to provide additional information necessary for a complete and accurate diagnosis and prognosis.

Differential diagnoses for disorders of the pastern region include PIP joint OA, osteochondrosis (OC), fractures, bone bruises, luxation/subluxation of the PIP joint, infection, lacerations, and soft tissue injuries; however, the types of injuries are often breed or use specific. For the purposes of this discussion, conditions of the pastern will include bone and joint abnormalities of P1 and P2 and the PIP joint and soft tissue injuries of the palmar/plantar aspect of the pastern including the DSLs, digital annular ligaments, distal branches of the SDFT, and the DDFT within the distal aspect of the DFTS.

OSTEOARTHRITIS (OA) OF THE PIP JOINT (HIGH RINGBONE)

The term "high ringbone" is often used synonymously with OA of the PIP joint and "low ringbone" with OA of the distal interphalangeal joint. Most cases of ringbone present with both joint and periarticular pathology.[43] OA or degenerative joint disease of the PIP joint is an important and common cause of lameness in virtually all breeds and ages of horses.[56] Older horses appear to be at greater risk and the forelimbs are more frequently affected than the hindlimbs. Secondary OA from P2 fractures (particularly palmar/plantar eminence fractures) or OC occurs more commonly in the hindlimbs.[31,71]

Etiology

Chronic repetitive trauma of the PIP joint and surrounding structures is thought to be the most common cause of PIP joint OA. Inherent conformational traits and the type of work the horse performs may also contribute to problems in the PIP joint. For example, horses that are base narrow and toe out are thought to be predisposed to OA on the lateral side of the joint, whereas horses that are base wide and toe in are believed to be predisposed to injury on the medial side of the joint. Pasterns that are overly upright may also result in increased concussion to the PIP joint



Figure 4.85. An upright pastern conformation predisposes to pastern OA.

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(Figure 4.85). Poor conformation may predispose to pulling/tearing of the soft tissues surrounding the joint or to incongruences within the joint surfaces. In addition, western performance horses that are required to start and stop suddenly or twist and turn abruptly appear to be prone to PIP joint damage.

Trauma to the periarticular soft tissues and the PIP joint may be repetitive or occur as a single, high-energy event that does not cause a fracture or joint luxation. Because the PIP joint is considered a low-motion/high-load joint, the articular cartilage and subchondral bone are placed under a greater workload, making these structures more susceptible to injury from nonphysiologic loading.[50] Overloading the PIP may cause direct articular cartilage damage and/or subchondral bone bruising that may contribute to the development of subchondral cystic lesions (SCLs) (Figure 4.86) or OA. Trauma may also cause injury of the periosteal attachments of the extensor tendons, ligaments, and joint capsule, resulting in periostitis and new bone formation. In addition, these injuries may cause joint instability or subluxation leading to secondary cartilage damage and OA. The OA may also result from an imbalance between repetitive microtrauma sustained during athletic performance and the adaptive repair mechanism of the skeletal tissue.[50] Whether primary synovitis/capsulitis is a distinct entity involving the PIP joint is uncertain,[43] but it is less commonly recognized than with other joints in the horse. Lame horses that block to intra-articular (IA) anesthesia of the PIP joint that do not have radiographic abnormalities may have PIP joint synovitis/capsulitis, subchondral bone bruising, or other unrecognized bone or cartilage damage that can only be recognized with advanced imaging such as CT or MRI (Figure 4.87). When bone bruising does not involve the subchondral bone, it can be expected to heal without long-term consequences with rest alone (Figure 4.88).

OA of the PIP joint may develop secondarily to a number of abnormalities within the joint. These include OC, unrecognized palmar/plantar eminence fractures of P2, traumatic blows or lacerations, septic arthritis, and selective weight-bearing in young horses.[16,23,71] OA of the PIP joint secondary to OC is usually seen most frequently in the hindlimbs of horses less than 3 years of age, and more than one joint can often be affected.[71] Single eminence fractures of P2 also occur most commonly in the hindlimb and can be the cause of chronic hindlimb lameness if unrecognized at the initial injury. Direct blows to the pastern region and lacerations at the margin of the PIP joint or involving the collateral ligaments may contribute to OA of the PIP joint. Infectious arthritis is often associated with an existing laceration or puncture wound that involves the PIP joint but may also develop after a wound has completely healed.

Clinical Signs

Focal or diffuse enlargement of the pastern region may be evident visually as well as on palpation (Figure 4.89). Palpable heat and pain with firm digital pressure may be appreciated. The affected pastern region may feel larger, particularly the dorsolateral and dorsomedial surfaces, than the contralateral joint. In most



Figure 4.86. Dorsoplantar radiograph of a trauma-induced SCL of the distal aspect of P1 (arrow) that was not apparent radiographically until 3 months after the original injury.

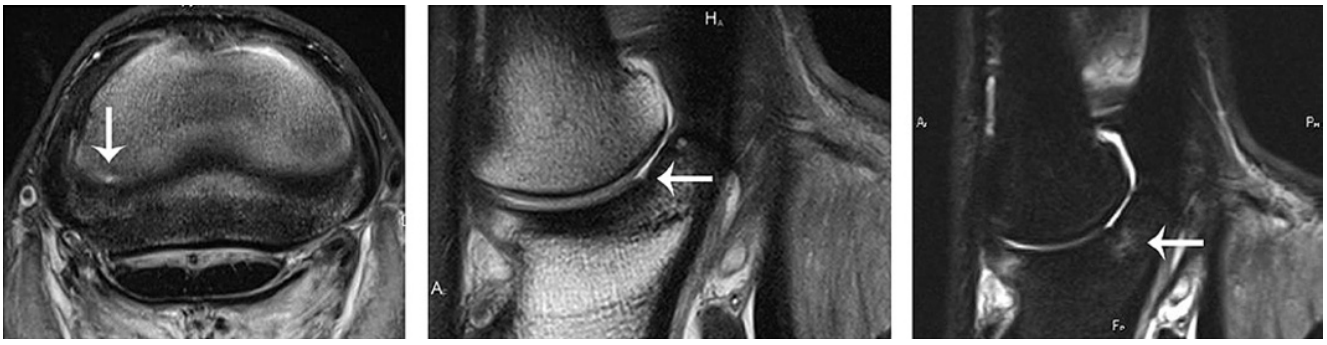


Figure 4.87. Proton density-weighted (left and center) and STIR (right) MR images from a horse with moderate to severe lameness of 1 month's duration that blocked to the pastern joint. There were no radiographic abnormalities. There is an articular cartilage defect (arrow left and center) and fluid signal intensity/bone marrow lesion in the subchondral bone (arrow right). The lameness did not improve with stall rest. The lameness persisted and the joint developed radiographically apparent signs of OA after 3 months of stall rest and it was treated with a pastern arthrodesis.

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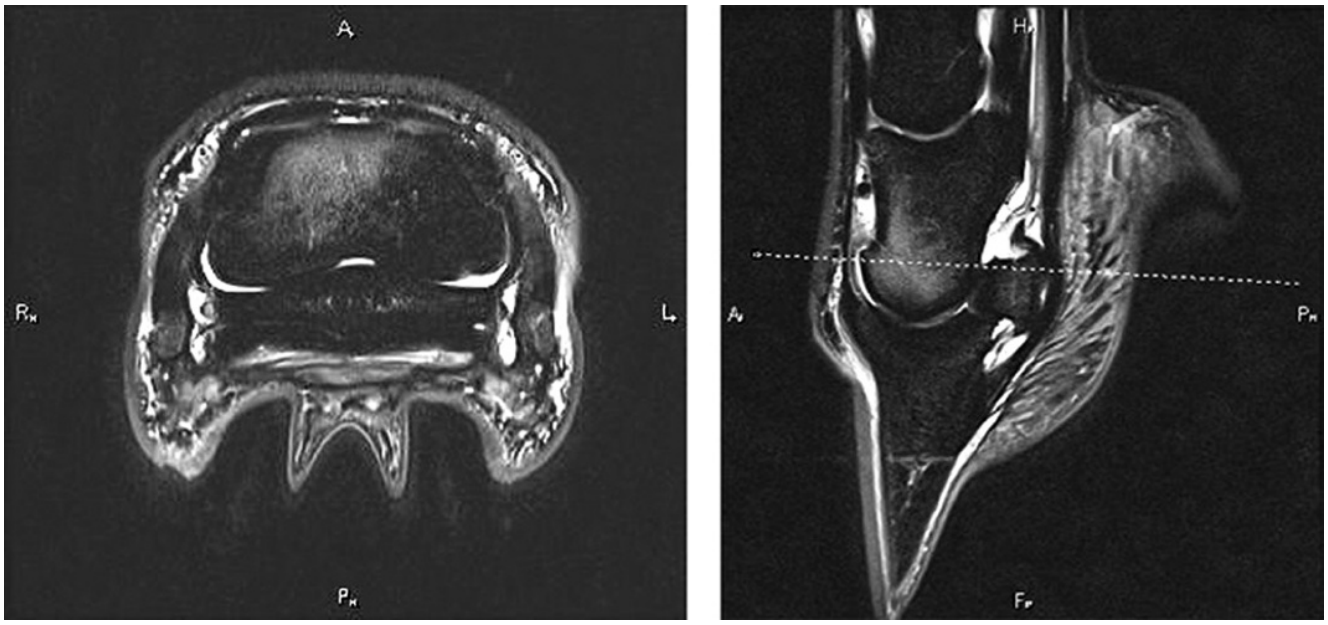


Figure 4.88. Transverse and sagittal STIR MR images with a large bone bruise of the dorsodistal P2. Because this lesion does not involve the subchondral bone, it healed without long-term consequences with rest alone.



Figure 4.89. Typical enlargement of the pastern that may be visible in horses with OA of the PIP joint. This was a 9-year-old Warmblood show jumper that was presented for pastern arthrodesis.

horses there is pain on flexion and rotation of the pastern region, unless the joint has undergone ankylosis. Obvious enlargement of the pastern and/or varus deformity of the phalanges may be present in horses with advanced disease. Horses with mild or early disease may have no visual or palpable abnormalities in the pastern region.

Lameness severity is variable depending on the severity of the disease. When there is radiographically apparent OA, the severity of lameness often correlates with the severity of the OA within the PIP joint, especially when there is joint space narrowing. The lameness is usually exacerbated at a trot, at exercise on an uneven surface (e.g. slope), or by circling at a trot. Except in early cases where there is focal medial injury, increased lameness is usually seen when the affected limb is on the inside of the circle. In some cases during foot flight, an exaggerated extension of the toe may be seen prior to foot placement.[68]

Pastern joint-associated lameness should not improve with a PD nerve block if performed very low in the pastern (just above collateral cartilages) with a small volume of anesthetic (1.5 mL).[62] However, some PD nerve blocks may desensitize the PIP joint. Most horses improve with a basisesamoid or high PD nerve block but may not become completely sound, especially if the disease process is severe. Response to IA analgesia varies depending on the injury, but improvement of lameness by 50% or more usually implicates the PIP joint as the site of the problem. The authors prefer the palmar/plantar approach to block and inject the PIP joint when there is dorsal bony proliferation, which can make the dorsal approaches more difficult.[45]

Diagnosis

A tentative diagnosis of OA of the PIP joint is often made based on physical examination findings combined with the responses to local anesthesia and radiographs. The most common radiographic findings in a study performed on 196 horses (262 joints) with chronic OA of the PIP joint were (1) joint space narrowing or collapse, (2) osteophyte formation, (3) subchondral bone sclerosis, (4) periosteal/periarticular bony proliferation, (5) deformity/collapse of the joint space, and (6) subchondral lysis (Figure 4.90).[15] Other radiographic features that may be identified include SCLs (Figure 4.86) or OC lesions,[71] and chronic eminence fractures of P2.

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Figure 4.90. Lateral radiograph of the pastern demonstrating periosteal reaction and small osteophytes on the dorsal aspect of the joint indicative of mild to moderate OA within the joint. Dorsopalmar radiograph of the same pastern demonstrating enthesophytes, subchondral lysis, and asymmetrical joint space collapse indicative of severe OA within the joint. This horse would not be expected to do well with medical therapy.

Periarticular new bone growth associated with lacerations may be limited to the site of trauma unless the joint was invaded and/or infectious arthritis is present. Infectious arthritis of the PIP joint often causes severe periosteal/periarticular bony proliferation, osteophyte formation, and subchondral lysis seen on radiographs. Older horses with trauma-induced OA or young horses with OC should have radiographs of the contralateral PIP joint performed because these disease conditions are often bilateral.

In early acute cases, there may be minimal to no palpable changes of the pastern region, and even radiographic examination may not reveal abnormalities. In these cases where pastern injury is still suspected, MRI[49,79] and/or ultrasound exam can be performed. MRI findings in cases of OA of the PIP joint include the same radiographic findings of osteophytes, joint space narrowing, subchondral lysis, periosteal proliferation with enhanced ability to detect subchondral bone changes (lysis, sclerosis), bone marrow lesions, SCLs, articular cartilage lesions, and joint effusion (Figure 4.87). Alternatively, repeat radiographic examination can be performed in 1–2 months because evidence of periostitis (periarticular new bone) and peripheral osteophyte formation may be present at that time.

Treatment

Conservative Management

The decision regarding treatment of horses with OA of the PIP joint depends on the severity of the disease, degree of lameness, age and intended use of the horse, and the owner's expectations and financial constraints. Horses with severe lameness and advanced radiographic abnormalities are usually not good candidates for nonsurgical treatments. It is important to note that the most important radiographic finding in grading the severity of OA of the PIP joint is joint space narrowing, especially when narrowing is asymmetrical (Figure 4.90). Horses with quite severe accumulation of periarticular bone but normal joint space width would be more likely to respond to conservative therapy than horses with joint space collapse, despite both groups having so-called severe radiographic changes (Figure 4.91). Horses with mild to moderate radiographic abnormalities of the PIP joint may respond well to conservative treatment, depending on the horse's intended use. Horses with a single traumatic injury to the pastern, such as those with an MRI diagnosis of a bone marrow lesion, may respond well to rest with complete healing and no long-term evidence of joint disease or OA (Figure 4.89), while others with bone marrow lesions may develop OA in the following months (Figure 4.88). There are numerous treatment options and the decision on how to treat horses with OA of the PIP joint is often made on a case-by-case basis.

Conservative management of PIP OA may involve periods of rest, systemic and/or IA anti-inflammatory therapy, trimming and shoeing, and a change in the horse's career. See Chapter 8 for more specific details on these treatment modalities. In acute traumatic cases, stall confinement and rest from exercise are important to prevent further trauma, reduce inflammation, and permit healing to occur.[16,68] Rest periods may extend from weeks to months depending on the severity and type of the injury and the response to treatment. Confinement and rest is rarely effective in horses with chronic OA of the PIP joint.

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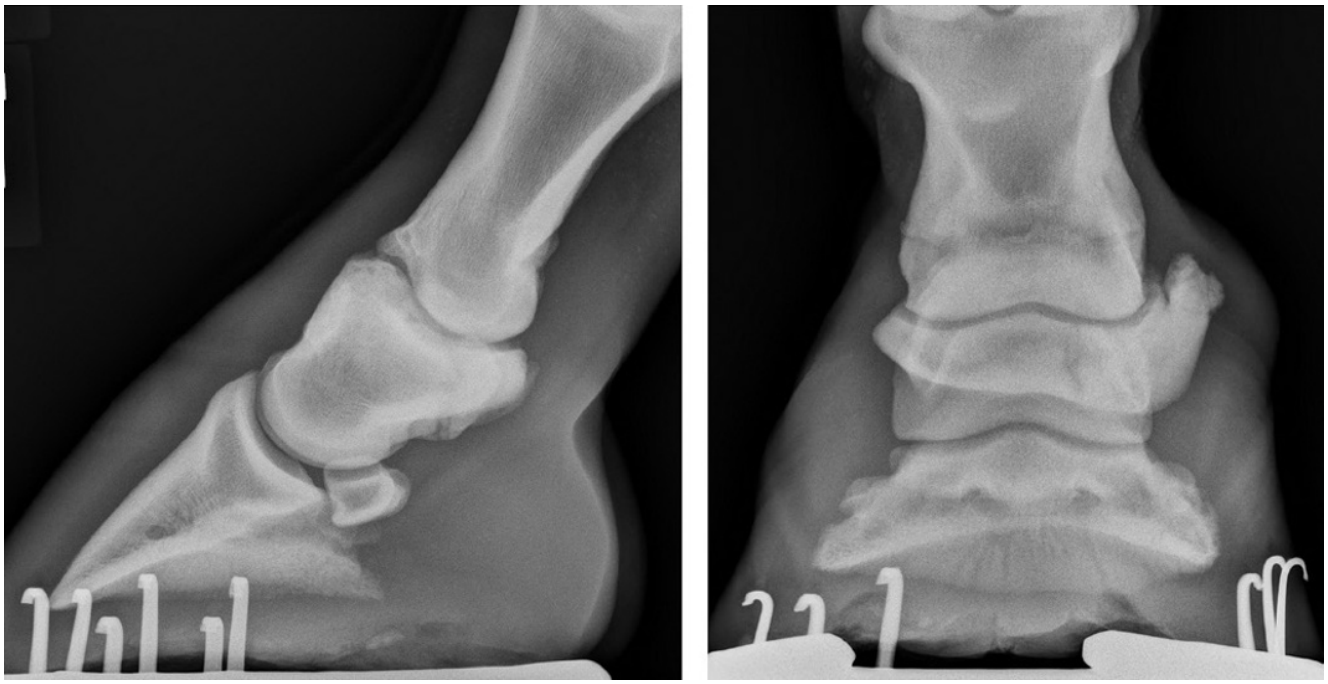


Figure 4.91. Lateral and dorsopalmar radiographs of the pastern with severe periarticular bone dorsally and medially but not joint space narrowing. Radiographs of the contralateral limb were nearly identical. This horse had minimal to no hindlimb lameness and was maintained in full work (reining) for 2 years after these radiographs were made. Recheck radiographs 2 years later were largely unchanged and there continued to be minimal lameness.

Often oral NSAIDs are combined with IA medication (steroid ± hyaluronan [HA]) of the PIP joint in horses with chronic OA to reduce the signs of lameness and improve the effectiveness of both treatments. A combination of a steroid (e.g. triamcinolone or methylprednisolone acetate) and HA can also be used IA in horses with acute disease to reduce inflammation and slow the progression of the disease. Intramuscularly or IA polysulfated glycosaminoglycans (PSGAGs), IV HA, and oral nutraceutical supplements are also commonly used.

There is some debate as to whether corrective shoeing can benefit horses with PIP joint problems. Changing heel height appears to have much more effect on altering flexion and biomechanical forces of the DIP joint than the PIP joint. In addition, raising the heel decreases the strain on the DDFT but increases strain on the SDFT and the suspensory ligament.[34] Therefore, adjusting heel height may not be beneficial in horses with PIP joint problems. However, the hoof-pastern axis should be corrected if abnormal because either a broken-forward or broken-back hoof-pastern axis can contribute to problems within the PIP joint. In general, the feet should be trimmed and balanced, and shoes applied that minimize break-over forces and provide good lateral and medial support. See Chapters 8 and 12 for more information on corrective shoeing.

Surgical Treatment

Surgical treatment for OA of the PIP joint consists of arthrodesis, which is aimed at eliminating motion within the joint, thereby decreasing pain and lameness. Natural ankylosis of the PIP joint may occur, but it is often a long, painful process with variable results. Surgical arthrodesis is generally considered a better solution with more consistent results. Numerous surgical techniques have been proposed over the years to promote arthrodesis of the PIP joint.[6,8,21,23,28,31,37,41,59,66,67,74]

The basic principles of surgical arthrodesis include removal of the remaining articular cartilage; internal fixation of P1 and P2 with screws, plates, or a combination of plates and screws; compression across the joint surface; accurate alignment of the phalanges; and variable periods of external immobilization with a half-limb cast. The authors prefer a single dorsally applied 4.5-mm, narrow, 3-hole LCP with two 5.5-mm transarticular screws in most horses (Figure 4.92). In very large horses two plates with transarticular screws may enhance stability. This technique of a dorsal plate with transarticular screws is thought to improve the comfort level of horses in the immediate postoperative period and requires casting for only 2 weeks postoperatively to optimize surgical wound healing.[31] Cancellous bone grafting is usually unnecessary, but forage or osteostixis of the subchondral bone around the joint may promote faster arthrodesis, especially in older horses that have very dense sclerotic bone. Bilateral forelimb or bilateral hindlimb arthrodesis may also be performed successfully.

Following a routine arthrodesis, the cast is removed after 2 weeks, which is adequate time for the surgical wound to heal.[31] When minimally invasive procedures can be used on moderately to severely affected OA joints, horses can be placed in a bandage only after surgery.[74] Arthrodesis for phalangeal fractures usually require a longer period of external immobilization. Postoperative stall confinement is recommended for a total of 8–12 weeks with introduction of hand-walking exercise during this time. For the second 2–3 postoperative months, horses are either small paddock confined or

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the amount of in-hand exercise is increased, dependent on horse temperament and available facilities. Although some horses may be ready to return to performance by 6 months, up to a year of convalescence may be required for others. The implants do not typically need to be removed.

Prognosis

After arthrodesis of the PIP joint, approximately 89%–95% of horses with hindlimb and 70%–85% of horses with forelimb lameness should return to their intended use, and 85% will return to athletic soundness.[31,37,41,59] Complications that may prevent horses from becoming athletically sound include implant infection, excessive bony proliferation that impinges on the DIP joint, exostosis of the extensor process of the distal phalanx, and soft tissue "irritation" associated with the implants (screws protruding from the palmar/plantar aspect of P1 and P2).

OSTEOCHONDROSIS (OC) OF THE PIP JOINT

OC of the PIP joint is identified less commonly than other joints in the horse. However, both osteochondral fragmentation and SCLs can be seen in the PIP joint.[19,20,52,60,70,71] Osteochondral fragments tend to occur dorsally (usually from the distal aspect of P1) or palmarly/plantarly (midline from the eminences of P2; Figure 4.93). SCLs due to OC are usually seen on the

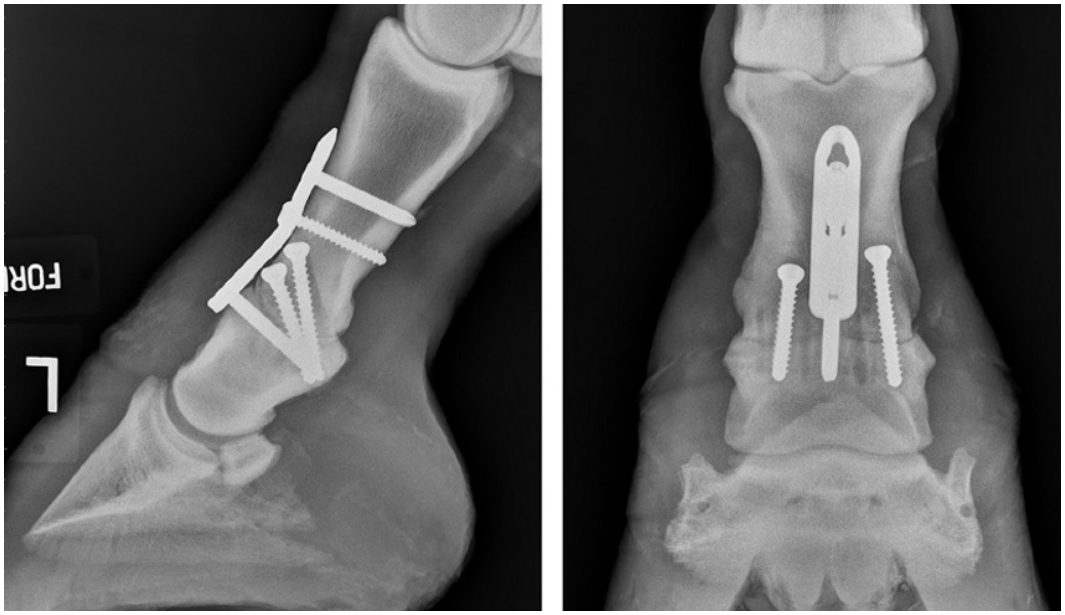


Figure 4.92. Lateral radiograph of the pastern following placement of a 3-hole LCP with two transarticular 5.5-mm screws for arthrodesis of the PIP joint.

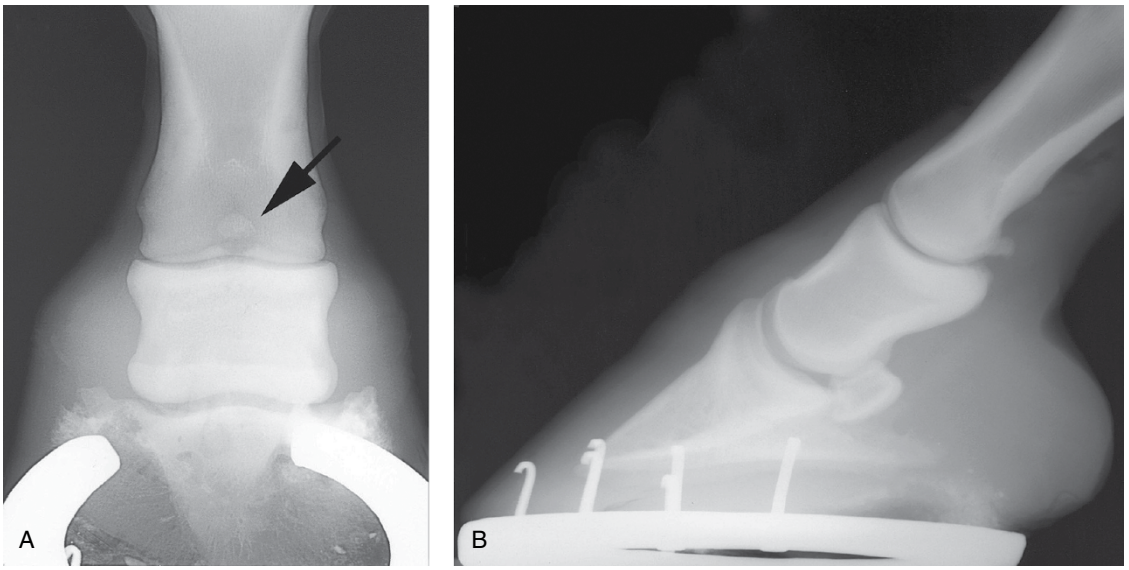


Figure 4.93. Dorsopalmar (A) and lateral (B) radiographs of a horse with palmar fragmentation of the PIP joint (arrow). The fragment was removed with arthroscopy.

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distal aspect of P1 but do occur rarely in the proximal aspect of P2 (Figure 4.94). SCLs due to OA occur with equal frequency in distal P1 and proximal P2. Malformation of the condyles of the distal aspect of P1 without fragmentation has also been recognized by the author and may represent another form of OC that leads to early OA within the PIP joint.

Etiology

The cause of OC within the PIP joint is assumed to be due to similar factors that cause the condition in other locations within the horse. However, traumatic fragmentation and articular cartilage or subchondral bone damage leading to SCLs can also occur within the PIP joint, and it may be difficult to differentiate between developmental and traumatic causes. Developmental lesions tend to occur in younger horses, whereas trauma can occur in any age horse. In addition, more severe clinical signs related to the PIP joint may be identified associated with trauma vs. OC.

Clinical Signs

As stated above, developmental lesions tend to occur in young horses and may cause variable signs of lameness, even within the same horse on a different day. Physical examination findings are similar to other problems within the PIP joint and include no abnormalities, enlargement of the pastern, pain with flexion and manipulation of the pastern region, and positive response to flexion tests. Horses with SCLs of the distal aspect of P1 tend to be more lame than those with osteochondral fragmentation. SCLs are also more common in the hindlimb than in the forelimb.[71] Due to the variable lameness that may be associated with OC in the PIP joint, it is important to identify the true source of lameness using diagnostic anesthesia in many cases.

Diagnosis

The diagnosis is confirmed with radiographs of the pastern region. Osteochondral fragmentation can usually be seen on both lateral and dorsopalmar/plantar views (Figure 4.93), whereas SCLs are often only visible on the dorsopalmar/plantar radiographic projection (Figure 4.94) or only with cross-sectional imaging such as CT or MRI (Figure 4.95). Some lesions, particularly osteochondral fragmentations, may be incidental findings on radiographs. Most SCLs that involve the distal condyle of P1 are clinically significant and often lead to lameness and OA. Radiography of the opposite PIP joint should be performed because OC lesions can be bilateral, similar to other locations.

Treatment

The treatment of choice for osteochondral fragments within the PIP joint that cause clinical problems is arthroscopic removal. Both the dorsal and palmar/plantar pouches of the PIP joint are accessible with the arthroscope, but the surgery can be difficult.[44,52] An arthrotomy can be performed but is associated with more soft tissue damage and increased likelihood of OA developing within the joint. SCLs of distal P1 (Figure 4.95) are often clinically significant and can be managed conservatively or surgically, depending on the severity of lameness. Surgical management includes trans-cyst screw application, transcortical cyst debridement, corticosteroid injection, or pastern arthrodesis (Figure 4.96). Conservative management with NSAIDs and IA medication usually resolves the lameness temporarily, but recurrence is common. Therefore, most horses with SCLs and PIP joint OA are best treated surgically.

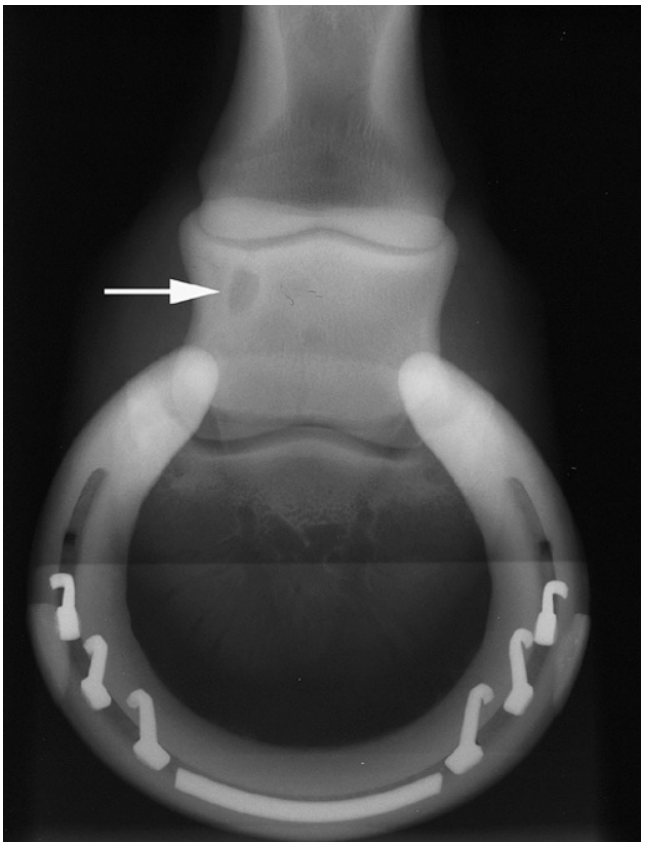


Figure 4.94. Dorsoplantar radiograph of a young horse demonstrating an SCL of the proximal aspect of P2 (arrow). This abnormality was not considered to be the cause of the lameness in this horse.

Prognosis

The prognosis following removal of OC fragmentation is usually very good, and many horses can perform athletically. The prognosis for SCLs of distal P1 after trans-cyst screw placement or transcortical debridement has not yet been reported in the literature, but if it is similar to SCLs in other locations, it will be good. SCLs of distal P1 typically do well following arthrodesis, and these horses can be used as athletes.

LUXATION/SUBLUXATION OF THE PROXIMAL INTERPHALANGEAL (PIP) JOINT

Luxation of the PIP joint is uncommon and can occur in the medial/lateral or palmar/plantar direction. Medial/lateral luxation is usually seen after severe injury to one

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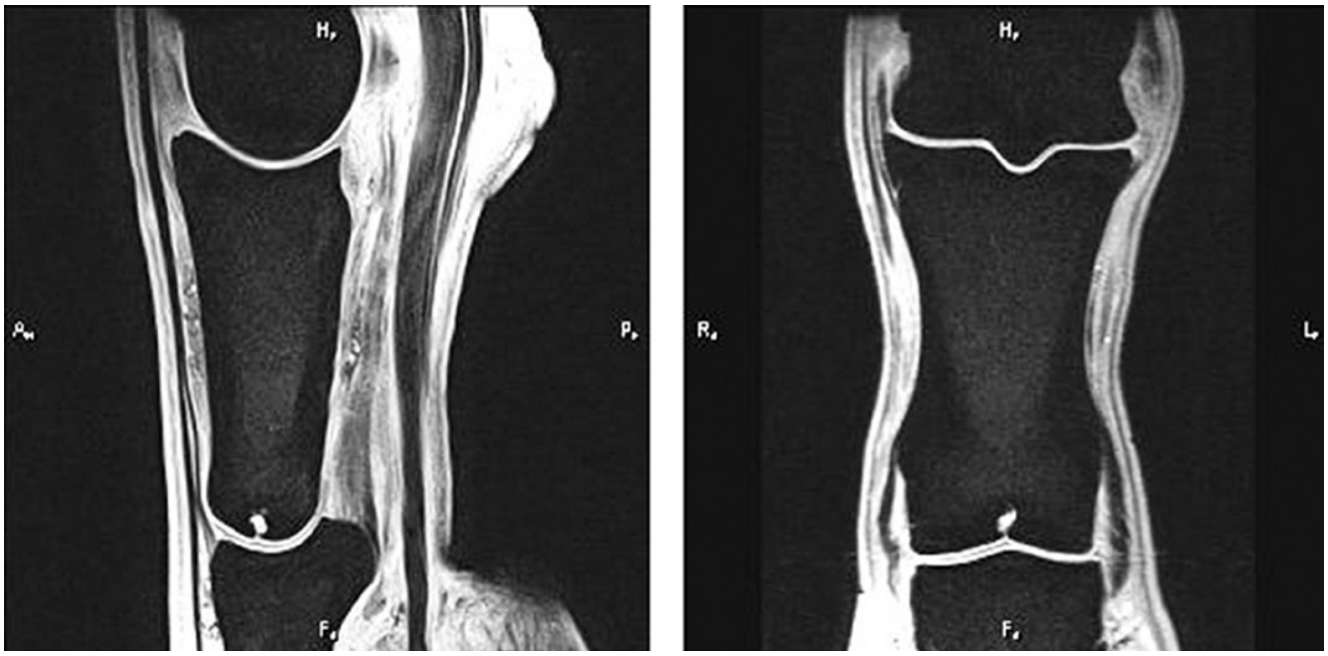


Figure 4.95. Sagittal (left) and dorsal (right) FLASH MRI images of the same horse in Figure 4.12. There is a subchondral cyst-like lesion of the distal aspect of P1 in the left hindlimb. Radiographically apparent lysis was not noted until after discovery of the cyst-like lesion on MRI.

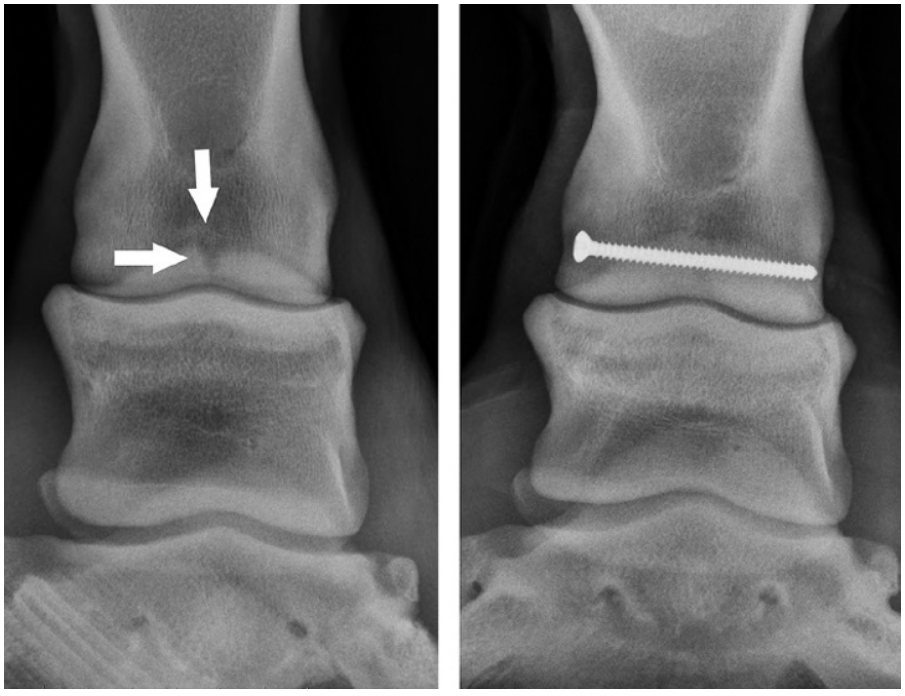


Figure 4.96. Preoperative (left) and postoperative radiographs (right) from a 4-year-old Warmblood mare treated with a transcortical screw for a subchondral cyst-like lesion in distal P1. This is the same horse shown in Figure 4.95.

of the collateral ligaments from external trauma and may be open or closed. Palmar/plantar luxation is usually seen following a severe, traumatic soft tissue injury, such as complete tearing of the straight DSL, branches of the SDFT, or a combination of these injuries, and usually only results in subluxation (Figure 4.97).[73] Luxation in the medial/lateral and palmar/plantar direction nearly always involves a single limb.

Subluxations of the PIP joint occur most commonly in a dorsal direction. The terms dorsal and palmar/plantar refer to the subluxation of the proximal phalanx relative to the position of the middle phalanx. Dorsal subluxations of the PIP joint are most common in young horses and may be secondary to flexural deformities and other developmental orthopedic diseases (Figure 4.98) as well as secondary to severe suspensory ligament injury and

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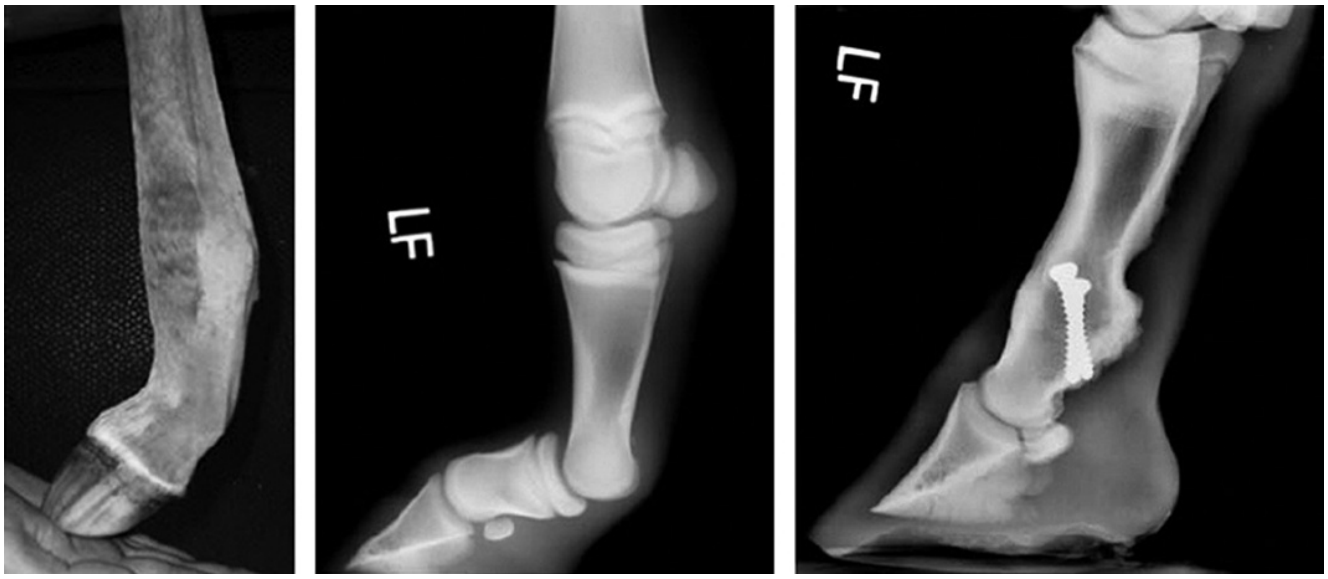


Figure 4.97. Subluxation of the pastern due to avulsion of the SDFT insertions and the straight distal sesamoidean ligament insertion that occurred at 1 month of age and was treated by pastern arthrodesis. *Source*: With permission EVE 2007.

hyperextension of the metacarpo(tarso)phalangeal joint. Although considered to be uncommon, subluxation/luxation of the PIP joint represented 21% of horses that underwent a PIP joint arthrodesis in a one report.[31]

Etiology

Lateral/medial luxations or subluxations are often caused by severe trauma resulting in joint capsule and ligamentous tearing (e.g. distal limb caught in something and the horse struggles and/or falls) or lacerations that transect the collateral ligament. Palmar/plantar subluxation/luxation generally occurs from acute trauma resulting in overextension of the PIP joint and tearing of the soft tissue support structures (joint capsule, straight DSL, and the insertion of the SDFT). Bilateral palmar/plantar eminence fractures of P2 may cause the same effect due to complete loss of the palmar/plantar supporting soft tissues. Palmar/plantar subluxation can also be seen in foals and weanlings that have jumped from heights and in foals with flexor tendon laxity that overexert themselves during free exercise.[26,73] Loss of the ligamentous support of the palmar/plantar aspect of the PIP joint results in subluxation/luxation and severe lameness.[8]

Dorsal subluxations are thought to be secondary to flexural deformities or limb contracture and may be seen in foals/weanlings that are rapidly growing with an upright conformation (Figure 4.98). A form of DDFT contracture was responsible for dorsal subluxation of the pastern in the pelvic limbs of three horses.[63] Dorsal subluxation may also be seen in horses after traumatic disruption of the suspensory apparatus and arthrodesis of the fetlock joint, or progressive, severe suspensory desmitis.[8]



Figure 4.98. A young horse with bilateral dorsal subluxation of the PIP joints. Dorsal swellings over both rear pastern joints can be seen.

Clinical Signs

The clinical signs associated with complete medial/lateral luxation due to tearing of the collateral ligament are usually obvious. These horses are often non-weight bearing or lame at the walk and have swelling of the pastern associated with the ligament injury, and a limb deformity may be present. Instability and pain may be identified with rotation or medial/lateral movement of the phalanges. In acute cases, heat, pain, and swelling of the pastern region are usually evident.

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Horses with palmar/plantar subluxation/luxation are also often very lame in the acute stage. The lameness may subside over time, but most will remain lame at the walk and the dorsal surface of the pastern will appear concave (dished out) rather than straight or convex as would occur with dorsal subluxation. In chronic cases the heel bulbs may contact the ground, and excessive hyperextension of the pastern and sinking of the fetlock is noted when the horse is walked.

Dorsal subluxation can occur without any identifiable structural abnormalities of the pastern or metacarpal/metatarsal soft tissue structures. This occurs primarily in the hindlimbs in young horses and lameness is usually absent or mild. A dorsal swelling in the pastern region may be evident when the affected limb is unweighted. This type of subluxation is often dynamic in nature and usually resolves during full weight-bearing of the PIP joint. An audible clicking sound often accompanies the reduction of the joint. When the pelvic limbs are involved, the condition is often associated with an upright conformation (straighter than normal hocks and stifle angles).[63]

With persistent dorsal subluxation of the PIP joint, an obvious swelling over the dorsal aspect of the pastern region is often evident, and the fetlock may appear slightly more extended (dropped) as compared to the contralateral unaffected limb. The dorsal swelling may appear similar to that associated with high ringbone, but on closer observation an abnormal alignment between P1 and P2 is found. However, with chronicity, both clinical and radiographic abnormalities consistent with OA of the PIP joint may develop. Lameness is variable and inconsistent in these cases and often depends on the secondary changes that develop within the joint.

Diagnosis

A tentative diagnosis can usually be made from the history and physical examination of the horse. Radiographs should be taken to confirm the diagnosis and identify concurrent abnormalities such as fractures or OA. Stress films may be needed to confirm medial/lateral subluxation because the phalanges can often remain in correct anatomic alignment unless pulled medially or laterally (Figure 4.99). Dorsal and palmar/plantar subluxations/luxations are usually obvious on standing lateral to medial views of the pastern.

Treatment

The treatment of choice for medial/lateral and palmar/plantar subluxations/luxations of the PIP joint is arthrodesis of the joint. External coaptation with a cast or Kimzey splint (Kimzey Leg Saver Splint; Kimzey, Inc., Woodland, CA) may be successful in adult horses managed acutely, but instability of the PIP joint may preclude successful realignment. Unlike medial/lateral luxations of the fetlock joint, similar luxations of the PIP joint do not respond well to casting alone and often develop secondary OA and persistent lameness. Conservative treatment of palmar/plantar subluxations/luxations is usually unsuccessful, and surgical arthrodesis is often the only option to realign the phalanges. Chronic palmar/plantar subluxations/luxations can lead to fibrosis of the PIP joint in an abnormal position, making surgical realignment difficult. Dorsal subluxations may also be treated with arthrodesis if they fail to respond to other methods of treatment. In most cases the subluxation is best treated surgically before excessive scar tissue has developed to permit more accurate and easier alignment of the joint. Horses with dorsal luxation of the PIP joint with secondary OA are also best treated with arthrodesis. See the section on OA of the PIP joint for more information on arthrodesis.



Figure 4.99. Dorsoplantar stress radiograph demonstrating complete rupture of a collateral ligament of the PIP joint. Arthrodesis of the joint is the recommended treatment.

Horses with intermittent dorsal subluxation with no apparent lameness may be treated conservatively. Horses with bilateral upright hindlimb conformation and dorsal subluxation often respond to anti-inflammatory medication and a controlled exercise program. Horses with intermittent dorsal subluxation of the pelvic limb associated with excessive tension of DDFT have been treated successfully with transection of the medial head of the DDFT.[63] The approach was between the DDFT and the suspensory ligament at the level of the proximal third of the third metatarsal bone, and a 2.5-cm segment of the tendon was removed. Alternatively, surgical transection of the accessory ligament of the DDFT may be of benefit.

Prognosis

Although there are few reports on long-term follow-up, the prognosis is good for survival and fair to good

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for return to use in horses with luxations/subluxations treated early by arthrodesis in which good reduction and stabilization of the PIP joint was achieved.[1,26,31,41,67] Convalescence is similar to that of other conditions that require a PIP joint arthrodesis, and up to a year may be required before the horse may return to performance. Three cases of bilateral acquired pelvic limb intermittent dorsal subluxation treated by tendonectomy of the medial head of the DDFT responded favorably to the treatment and the subluxation resolved between 1 and 7 days postoperatively.[63] The authors have no experience with this particular surgical technique.

FRACTURES OF THE MIDDLE (SECOND) PHALANX (P2)

Fractures of P2 occur most commonly in the hindlimbs of middle-aged western performance horses used for cutting, roping, barrel racing, pole bending, and reining.[8,10,42] However, these fractures may occur in any horse during lunging, turnout, after kicks or falls, or any form of single-event trauma. Fractures of P2 can also be seen in foals and usually involve the proximal physis, resulting in subluxation of the PIP joint.[12] Although the fracture is common in Quarter horses, any breed can be affected. Based on retrospective studies, the Quarter horse represents approximately 50% of the breeds affected, Western stock is the most common activity contributing to the injury, and the hindlimbs are affected approximately three times more frequently than the forelimbs.[8,10,42]

A variety of fracture types involving P2 have been reported, including osteochondral (chip) fractures, palmar/plantar eminence fractures, axial fractures, and comminuted fractures.[5,8,10,29,31,35,41,46,52,60,70,72] Osteochondral fractures and axial fractures are rare, whereas eminence and comminuted fractures occur commonly. Thoroughbred and Standardbred racehorses and hunter/jumper horses appear to be at increased risk for osteochondral fractures involving the PIP joint.[70] The fracture can either be located dorsally (most common) or on the palmar/plantar aspect lateral or medial to the midline.[46,52,60] Palmar/plantar fractures do not typically involve the attachments of the DSL or the branches of SDFT. Some of these osteochondral fragments may be seen in young horses and may be developmental in origin.[52] Occasionally, multiple fragments may occur on the proximal palmar/plantar aspect of P2 that appear to be avulsion fractures. Despite the size of the fragments, the development of secondary OA appears slow, but removal is usually recommended.

Radiographic examination of the opposite PIP joint should be done because palmar/plantar fractures have been reported to occur bilaterally.[46] Osteochondral fractures of P2 involving the DIP joint are uncommon and may be caused by use-related trauma or direct trauma from a penetrating injury.[70,72] Palmar/plantar eminence fractures involving the PIP joint occur frequently. They can either be uniaxial (involving one eminence; Figure 4.100) or biaxial (involving both eminences; Figure 4.101). Uniaxial eminence fractures do not result in subluxation of the PIP joint, whereas biaxial fractures can contribute to subluxation or complete luxation of the joint (Figure 4.102).

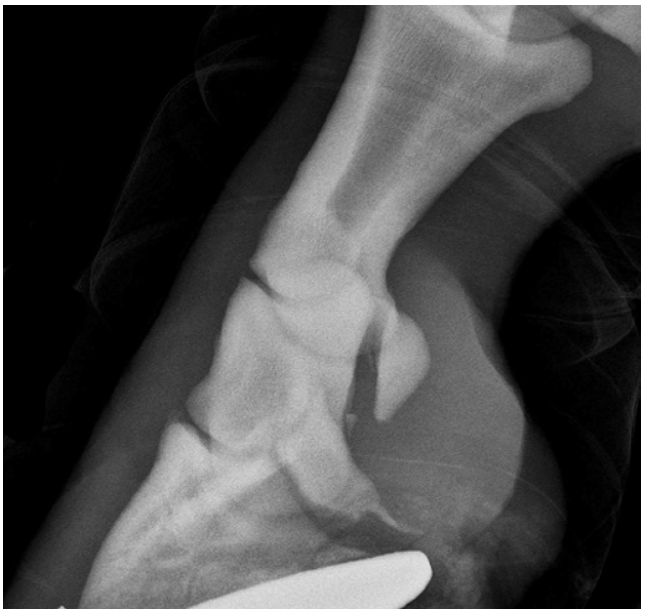


Figure 4.100. Oblique radiograph of the pastern region demonstrating a medial plantar eminence fracture of P2. This horse presented for a hindlimb lameness of 2 weeks' duration.



Figure 4.101. Lateral radiograph of the pastern demonstrating biaxial eminence fractures of P2. Internal fixation is required for these types of fractures to prevent palmar/plantar luxation of P1.

Simple axial fractures of P2 occur rarely.[63] A misdiagnosis of this fracture can easily be made if the central sulcus of the frog is prominent and it is filled inadequately with packing material prior to the radiographic exam. The fracture should be visible on at least two radiographic views to make a definitive diagnosis. This type of fracture may progress to a comminuted fracture under appropriate biomechanical factors.

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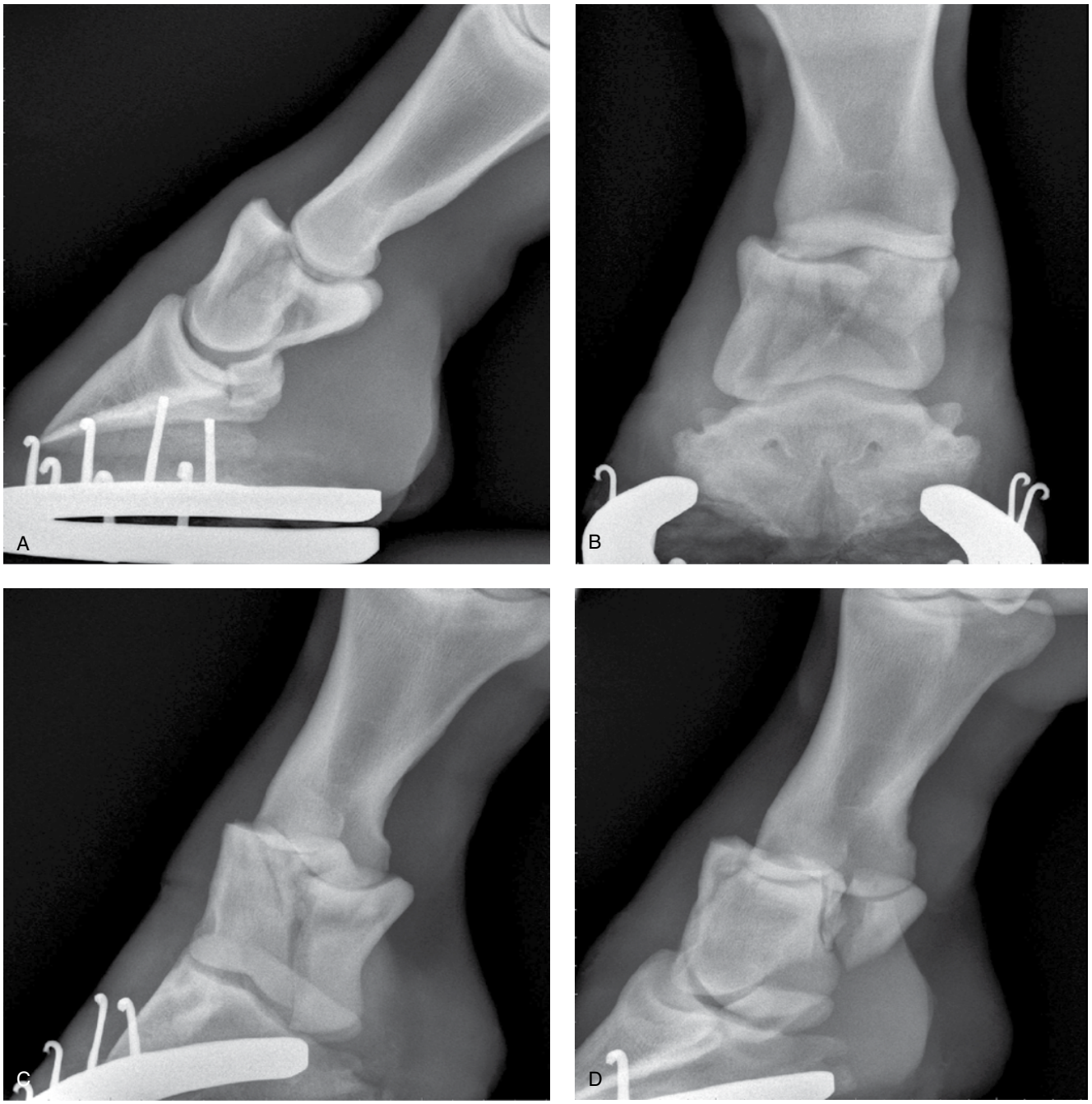


Figure 4.102. Lateral (A), dorsoplantar (B), and two oblique (C and D) radiographic projections of a horse with a comminuted P2 fracture. Multiple fracture lines are commonly seen, and most comminuted fractures involve both the PIP and the DIP joint surfaces.

Comminuted fractures are the most common fracture involving P2. They nearly always involve the PIP joint (uniarticular) but often extend distally into the DIP joint (biarticular). A variety of fracture configurations are possible, but typically multiple fracture lines oriented in several directions are visible on radiographs. In addition, it is not uncommon for these fractures to resemble a "bag of ice" due to the multiple fracture fragments. Although comminuted P2 fractures result in marked instability of the pastern region, they are rarely open. Proper immobilization of comminuted P2 fractures prior to transport to a surgical facility is highly recommended.

Etiology

Osteochondral fractures may occur from use, direct trauma to the bone (e.g. a penetrating wound), or avulsion of soft tissue attachments (palmar/plantar aspect), or they may be developmental associated with OC. Palmar/plantar eminence fractures may occur from compression and rotation that is associated with sudden stops and short turns. They may also occur during PIP joint overextension, which results in excessive tension of the SDFT and straight DSL, causing an avulsion of the eminence(s).

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The cause of simple axial fractures is unknown but may be associated with repetitive trauma. Comminuted fractures are thought to result from external trauma or a combination of compression and torsion (twisting) forces that occur with sudden stops, starts, and short turns. Most comminuted P2 fractures are thought to occur as a single-event injury, but a history of lameness in the affected limb may precede the fracture in some horses. Horses shod with heel calks are believed to be more prone to comminuted P2 fractures because the calks grip the ground, preventing the normal rotation of the foot and phalanges when the horse rapidly changes directions. These fractures may also occur in horses during light work or unrestrained paddock/pasture exercise due to sudden excessive forces (compression and torsion) placed on the limb ("bad step"). Horses turned out for exercise after long-term confinement have also been reported to be at risk for comminuted P2 fractures.

Clinical Signs

The clinical signs associated with P2 fractures that do not disrupt the weight-bearing capabilities of P2 (osteochondral fragments, single eminence, and simple axial fractures) can be variable. Some horses may have a history of an acute onset of lameness, while others may present for a chronic forelimb or hindlimb lameness. In most horses, exercise increases the severity of the lameness. Swelling of the pastern is not a reliable finding, but fetlock/phalangeal flexion and rotation of the pastern region often elicit a painful response. Crepitation or instability is generally not appreciated with uniaxial P2 eminence fractures. Circling at a trot usually exacerbates the lameness. Diagnostic anesthesia with either a basisesamoid nerve block or intrasynovial anesthesia is often required to localize the lameness to the PIP joint region. However, diagnostic anesthesia is contraindicated with other types of P2 fractures because of the risk of fracture displacement when the horse bears weight on the anesthetized digit.

Horses with comminuted or biaxial P2 eminence fractures often have a history of acute onset of severe lameness. Some owners may report that a loud "pop" was heard just prior to the onset of severe lameness. Horses are usually very lame and painful to manipulation of the pastern, and crepitus may be felt but is not a consistent finding. The pastern may also appear to be unstable during manipulation, and swelling may be present just above the coronary band in horses with comminuted fractures (due to effusion of the DIP joint). With biaxial eminence fractures the swelling is less evident and may not be apparent.

Diagnosis

A definitive diagnosis requires a complete radiographic examination. At least four views are recommended: dorsopalmar (DP), lateromedial (LM), dorsolateral to palmaromedial oblique (DLPMO), and dorsomedial to palmarolateral oblique (DMPLO). Osteochondral fractures are usually easily diagnosed with the routine radiographic views. Additional views may be necessary with comminuted fractures so that the fracture location and configuration can be accurately appreciated. Identification of whether the fracture lines extend into the DIP joint and whether there is an intact "strut" of bone that extends between the PIP and DIP is very important information for comminuted fractures. The fracture configuration has considerable bearing on the treatment method selected as well as the prognosis for future soundness.

Cross-sectional imaging like CT of comminuted P2 fractures is especially helpful in defining the fracture configuration[57] (Figure 4.103). In general, the degree of comminution and DIP joint involvement is underestimated on radiographs compared to CT. The authors routinely use CT for comminuted P2 fractures that aids in prognostication and in reconstruction of the DIP joint.

Treatment

Osteochondral Fractures

Fracture fragments associated with the PIP joint that contribute to lameness are best removed with arthroscopy.[52,70,72] Both dorsal and palmar/plantar fragments can be removed with arthroscopy, although the maneuverability of the instrumentation is somewhat limited in the dorsal joint pouch due to the extensor tendon attachment immediately distal to the joint.[60] The palmar/plantar recess

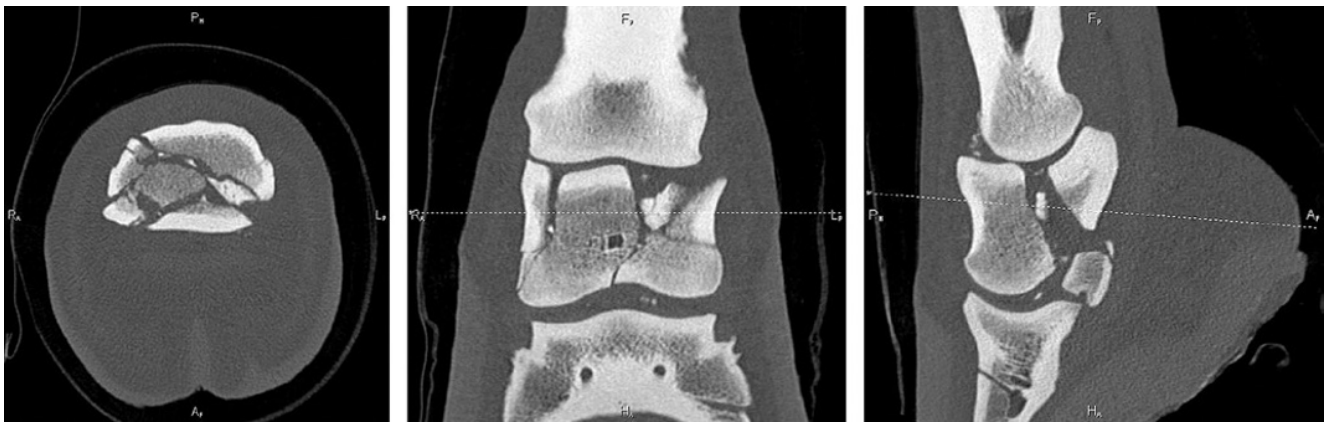


Figure 4.103. CT images of a comminuted P2 fracture that demonstrate the numerous fracture fragments that are present in different orientations. This fracture was repaired with two dorsally applied bone plates and pastern arthrodesis.

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of the PIP joint is larger, which makes surgical manipulation somewhat easier, but it is still difficult.[52] Osteochondral fractures involving the DIP joint are also best managed by arthroscopy if possible.

Eminence Fractures

Uniaxial or biaxial eminence fractures of P2 that involve the PIP joint are best treated by arthrodesis of the PIP joint followed by cast application.[42]

Although casting alone has been reported as an acceptable method of treatment in horses with uniaxial eminence fractures, it should be reserved for those cases in which pasture or breeding soundness is desired and economic constraints dictate this approach. The fractures rarely heal back to the parent bone with casting alone, and secondary OA of the PIP joint leading to chronic lameness is common.

Horses with biaxial eminence fractures will develop a palmar/plantar subluxation of the PIP joint when casted due to weight-bearing forces pushing P1 distally. If internal fixation of biaxial P2 fractures is not elected, then transfixation pin casts or another type of external fixator is recommended over casting alone to maintain phalangeal alignment. With either casting alone or transfixation pin casts, the initial cost may be less to the owner, but the costs of repeat casting, prolonged confinement, complications, etc. are often not that much less or may be more than what the cost of internal fixation would have been. Therefore, internal fixation of eminence fractures is the preferred method of treatment.

Comminuted P2 Fractures

There are varying degrees of comminution associated with nearly all P2 fractures, which can greatly affect treatment. In general, horses with comminuted P2 fractures should be reconstructed and repaired with some type of internal fixation (bone plating) if at all possible. Horses with an intact strut of bone spanning from the PIP to the DIP joints are ideal candidates for internal fixation. Horses without an intact bony strut yet have large enough bony fragments for screw fixation also often benefit from internal fixation. Horses with highly comminuted P2 fractures (so-called bag of ice) that do not have fracture fragments large enough to engage screws are best treated with transfixation pin casts or another type of external fixator.[29,35,47,48] Casting alone can also be used for these highly comminuted fractures, but fracture collapse would be an expected complication.

The use of casts, transfixation pin casts, or an external fixator is usually reserved for horses with severely comminuted P2 fractures that cannot be adequately reduced or stabilized by internal fixation and for which euthanasia is not an option. Economic constraints may also enter into the decision regarding when to use these options, although with the prolonged treatment period, the difference in cost is often negligible. Pasture soundness for breeding purposes or use as a pet is all that can be expected from horses treated with casts with or without transfixation pins. Traction to the phalanges using wires placed through the hoof wall, especially in the hindlimb, helps reduce the fracture fragments. A lower limb cast that incorporates the hoof is mandatory. Some comminuted P2 fractures may be treated successfully using this approach, but cast application for a minimum of 8 weeks is usually required.

Prognosis

The prognosis of horses with osteochondral fractures treated by arthrotomy or arthroscopy appears to be very good for return to full serviceability.[46,52,60,70,72] In one study 4 of 5 cases with osteochondral fragments (3 involved the PIP joint and 2 involved the DIP joint) treated by arthrotomy returned to their intended use.[70] There are only a few reports of using arthroscopy to remove these fragments, but it is the preferred approach with most horses returning to performance.

The prognosis for uniaxial or biaxial palmar/plantar eminence fractures treated by arthrodesis also appears to be very good for return to performance and should be considered similar to that expected for PIP joint arthrodesis used to treat OA of the joint.[42] The use of single or double bone plating is considered mandatory for biaxial eminence fractures and will most likely improve the prognosis of these horses returning to function.[12,42]

Horses with comminuted fractures that only involve the PIP joint usually have a good prognosis to return to athletic performance, provided they are treated with internal fixation. The prognosis for horses with biarticular comminuted P2 fractures is much reduced because the limiting factor in many cases is related to the health of the DIP joint. Older literature has stated that horses with comminuted biarticular P2 fractures have a 50% survival rate and slightly greater than a 10% chance of returning to athletic performance with casting alone.[8] This remains true today and emphasizes that horses with comminuted P2 fractures treated without internal fixation are very unlikely to be athletically sound.

Internal fixation with bone plates increases both survival and return to athletic function in horses with comminuted P2 fractures. In one report 7 of 8 horses with forelimb involvement were alive 2 years after injury, and 4 of 7 horses were being ridden. Of the two horses with hindlimb involvement, one was being ridden and one was lost to follow-up.[10] In another report, 2 of 3 mature horses with comminuted fractures that involved the forelimbs returned to athletic performance, and 2 of 3 foals with physeal fractures also performed.[12]

The prognosis of these horses is often dictated by the amount of fracture displacement at the DIP joint and the ability to reduce this displacement at the time of surgery. Horses with fractures of the hindlimb are also thought to have an improved prognosis over those affecting the forelimb. In general, horses with biarticular comminuted P2 fractures should be considered to have a 40–50% chance of returning to performance after internal fixation, provided complications do not occur.

FRACTURES OF THE PROXIMAL (FIRST) PHALANX (P1)

Fractures of the proximal phalanx (P1) occur frequently and can be broadly categorized into noncomminuted and comminuted fractures. Osteochondral fragmentations of the proximal dorsal or palmar/plantar

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aspects of P1 are types of noncomminuted fractures and are discussed in the fetlock section. Fracture configurations (excluding osteochondral fragmentation) range from small fissures that enter the metacarpophalangeal/metatarsophalangeal [MCP/MTP] joint to highly comminuted fractures ("bag of ice") that affect both cortices and the proximal and distal joint surfaces. Stress or fatigue-type fractures that may not be identified on routine radiographs may also occur in performance horses. Most P1 fractures are closed, although 8 of 64 comminuted fractures were open at the time of presentation and an additional 4 had extensive bruising with serous fluid oozing through intact skin in a retrospective review.[32]

Noncomminuted P1 fractures have been classified into several types:

1. Midsagittal or sagittal fractures: Exist primarily in the sagittal plane and begin at the proximal articular surface
   a. Short (extend less than 30 mm in length distally; Figure 4.104A)
   b. Long (extend more than 30 mm in length distally; Figure 4.104B)
   c. Complete (exit the lateral cortex or span both joint surfaces; Figure 4.105)
2. Dorsal frontal fractures: Begin at the MCP/MTP joint in the frontal plane and extend to the dorsal cortex or distally toward the PIP joint; can be incomplete or complete (Figure 4.106)
   a. Distal joint fractures: Involve the PIP joint
   b. Palmar/plantar eminence fractures: Involve the MTP/MCP joint (Figure 4.107)
   c. Physeal fractures: Usually Salter–Harris type II
   d. Oblique or transverse diaphyseal fractures

Comminuted P1 fractures can range from fairly simple three-piece fractures to the bag of ice type of injury (Figures 4.108 and 4.109). However, a variety of configurations of comminuted P1 fractures is possible. For treatment purposes, they are divided into fractures that have an intact cortex (strut) of P1 from the proximal to distal joint surfaces (moderately comminuted) and fractures that do not have an intact bone strut (severely comminuted).[32,40] Less common types of P1 fractures include proximal medial collateral ligament avulsion fractures, dorsal nonarticular fractures, and stress or fatigue fractures. Stress- or fatigue-type fractures may precede sagittal or comminuted P1 fractures and occur almost exclusively in performance horses. Medial collateral ligament avulsion fractures are discussed in the fetlock section.

Sagittal P1 fractures occur most commonly and may be present in the contralateral limb in a small percentage of the cases.[17,38,69] Sagittal and other types of noncomminuted P1 fractures are primarily seen in racing Thoroughbreds and Standardbreds but can also occur in other types of performance horses. They often affect the hindlimbs in Standardbreds and the forelimbs in

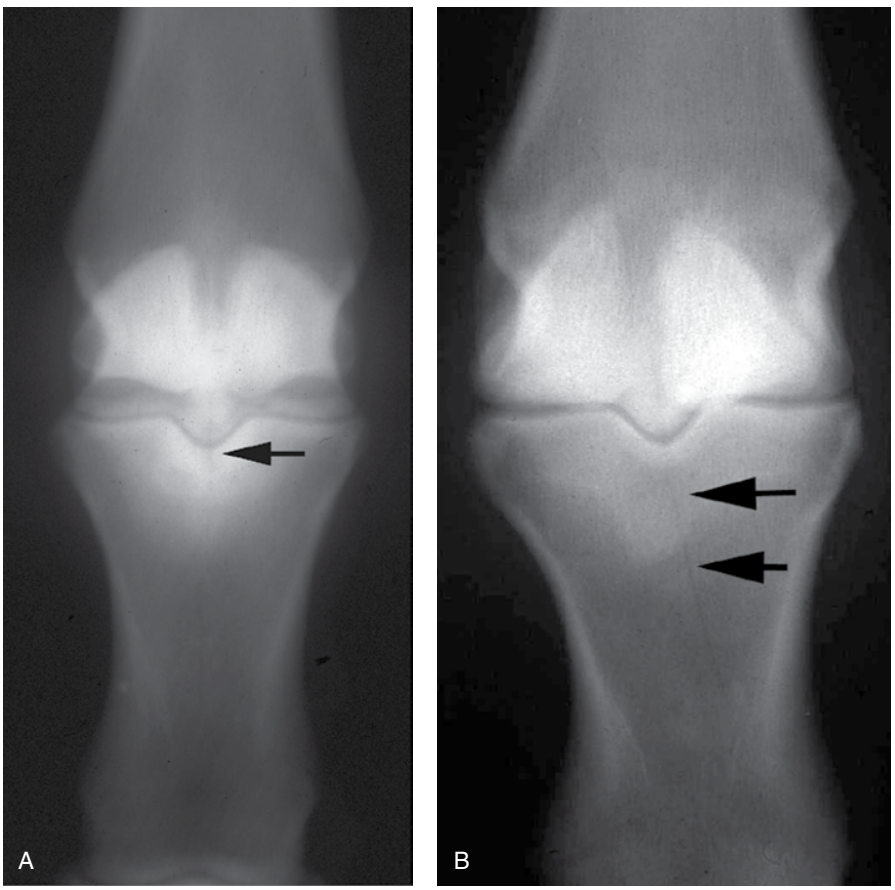


Figure 4.104. Dorsopalmar radiographs of the pastern region revealing short (A) and long (B) incomplete sagittal fractures of P1 (arrows). The fracture in (B) was repaired with two lag screws through stab incisions (Figure 4.110).

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Figure 4.105. Dorsopalmar radiograph of the pastern region demonstrating a complete sagittal fracture of P1 that exists in the lateral cortex. This fracture was repaired with three lag screws through an open approach to the fetlock joint. *Source*: Courtesy of Dr. Jeremy Hubert.

Thoroughbreds.[17,27,38,69] Dorsal frontal fractures are relatively rare but usually occur in the hindlimb.[3] Comminuted P1 fractures also occur commonly in racing Thoroughbreds and Standardbreds. In a recent report, 72% of horses with comminuted P1 fractures sustained the injury during racing or race training.[32] However, comminuted P1 fractures may occur in other types of performance horses (especially western performance or polo) and may occur in any horse at pasture or at exercise from a single traumatic event.[32,38] In one report 27% of the horses sustained a comminuted fracture while at pasture, and a smaller percentage developed the fracture while being used for showing or pleasure riding.[38] In a more recent report, 28% of all comminuted P1 fractures were from nonracing causes.[32]

Etiology

It appears that a combination of longitudinal compression in conjunction with asynchronous lateral to medial rotation of P1 or twisting of P1 in relation to the metacarpus/metatarsus may be the cause of some fractures. During normal weight-bearing, the convex sagittal ridge of the distal end of the third metacarpal or metatarsal bone fits into the concave groove in the proximal surface of P1. If this alignment is not perfect, the convex sagittal ridge may act as a wedge to create the fracture. Also, during limb flexion, there is a lateral to medial rotation of P1 around its long axis. If the rotary movement is accelerated, as would occur if the foot slips, a P1 fracture may result. In most cases, a combination of axial weight-bearing and torsional forces usually contributes to P1 fractures.

Nearly all midsagittal fractures begin at the concave groove of the proximal aspect of P1 and tend to be



Figure 4.106. (A) Oblique view of an incomplete dorsal fracture of P1. (B) Lateral view of a complete dorsal fracture of P1 that exits the dorsal cortex. *Source:* Courtesy of Dr. Julie Dechant.

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Figure 4.107. Lateral radiograph of the fetlock demonstrating a fracture of the palmar eminence of P1. This fracture was an acute injury and was repaired by lag screw fixation. Smaller, chronic fractures are more common in this location and are often removed if they are problematic.

oriented lateral to the midline in the forelimb. This may be due to a smaller lateral articular surface of P1 in the forelimb compared with the hindlimb.[17] The consequent position of the center of the sagittal groove lateral to the midline may dictate that fractures propagate down that side of the bone in the forelimb. In contrast, sagittal fractures that extend distal or medial to the midline are approximately twice as common in the hindlimbs as in the forelimbs.[17] The relationship between the slope of the proximal and distal articulations of P1 may also account for the different fracture configurations between the fore- and hindlimb.[17]

Parasagittal P1 fractures in the athletic horse may occur as part of a chronic pathological process characterized as bone maladaptation or fatigue-type fractures. In support of maladaptation etiopathogenesis, P1 fractures can occur during normal events without unusual trauma, at a consistent and site a specific location along dorsoproximal P1 with repeatable configurations and with prodromal signs such as periostitis. These so-called fatigue fractures are usually only seen in performance horses and often represent an area of bone weakness related to stress remodeling. As microdamage accumulates from repetitive loading of P1, the bone weakens or "fatigues," predisposing to fracture. This same scenario can occur in multiple locations in performance horses (humerus, metacarpus, tibia, pelvis, etc.). Radiographically silent short incomplete parasagittal P1 fractures have been seen on MRI and scintigraphy,[51,53] and these may precede the development of a radiographic apparent sagittal P1 fracture or may predispose to a comminuted fracture. Stress fractures tend to occur most commonly in the midsagittal groove at the proximal aspect of the bone, the same area where most P1 fractures originate.

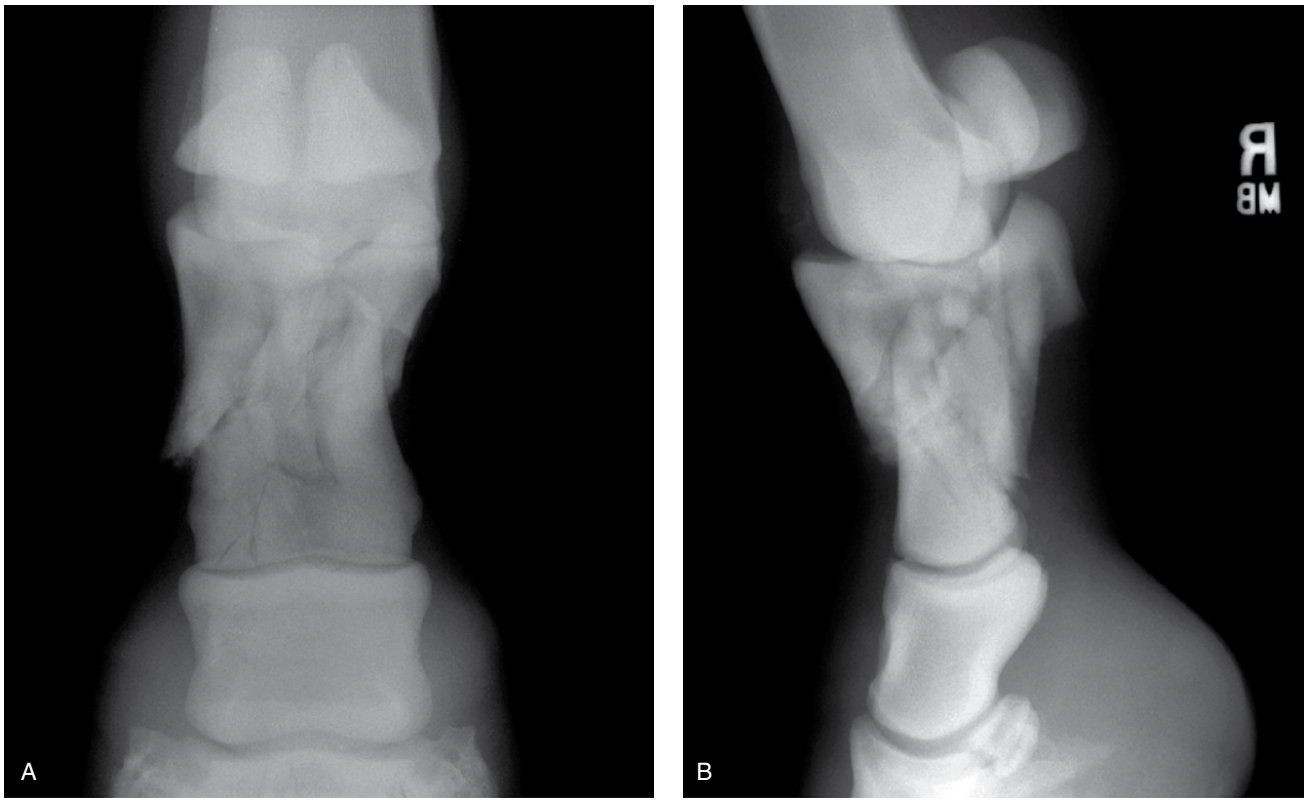


Figure 4.108. Lateral (A) and dorsopalmar (B) radiographs of a horse with a severely comminuted fracture of P1. Internal fixation is not possible with these types of fractures, and transfixation pin casts or external fixators are usually used to attempt salvage of these horses.

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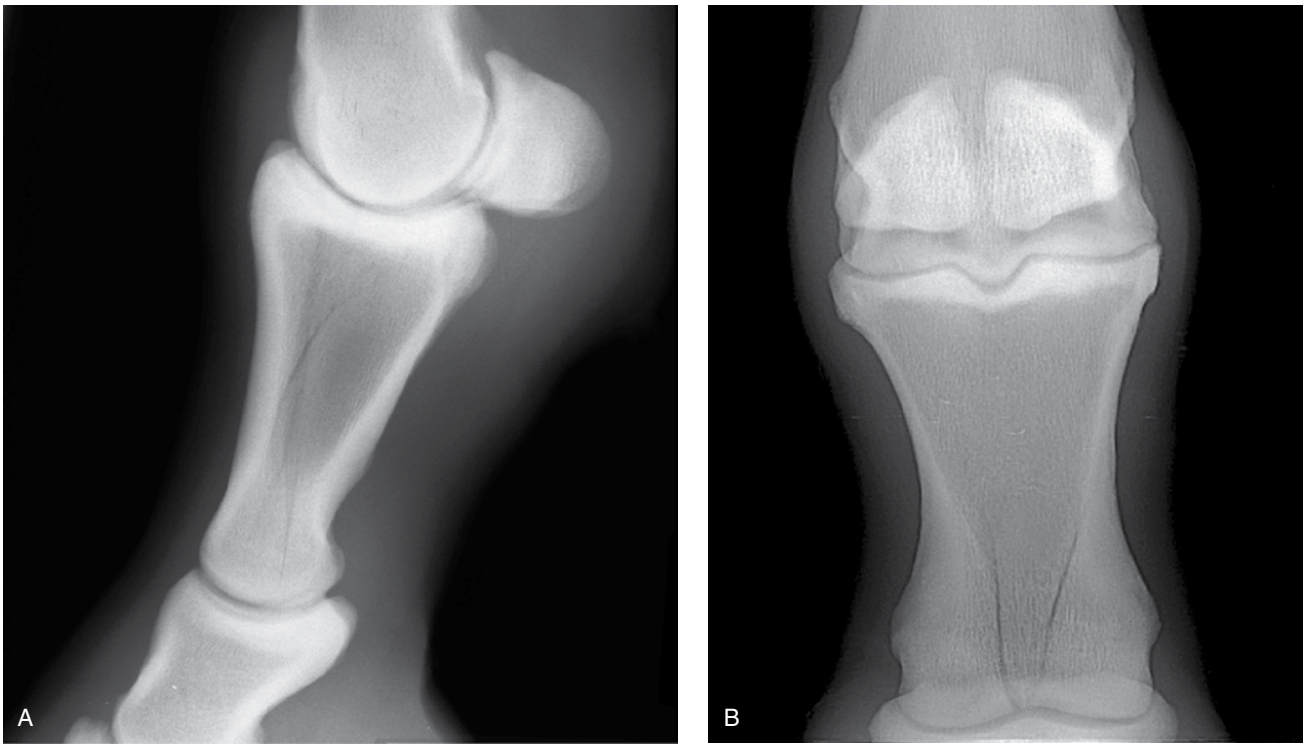


Figure 4.109. Lateral (A) and dorsoplantar radiographs (B) of a nondisplaced, moderately comminuted P1 fracture that was repaired with multiple lag screws placed through stab incisions.

Clinical Signs

The clinical signs associated with P1 fractures are variable and depend on the fracture type and degree of fracture propagation. In most cases there is usually a history of an acute onset of lameness. Horses with incomplete sagittal fractures may demonstrate moderate pain and lameness initially, but it may be of a short duration. However, fetlock effusion is usually present, and a painful response is often elicited with flexion and rotation of the phalanges.[69] Horses with complete sagittal fractures are usually quite lame (grade 3–4 of 5), and fetlock effusion and swelling of the pastern region is usually apparent. Horses with comminuted fractures are usually non-weight bearing and may show signs of physical distress such as sweating. The pastern region is often obviously swollen, and crepitus and instability is palpable.

Adequate external immobilization of the fracture in these horses is mandatory if they are being transported for surgical repair. Perineural anesthesia is usually unnecessary to make the diagnosis, but some horses with short sagittal P1 fractures may present for a routine lameness evaluation. Perineural anesthesia is contraindicated if any type of P1 fracture is suspected because it will increase the risk of fracture propagation.[32] Guidelines for transporting horses with comminuted P1 fractures can be found in Chapter 12.

Diagnosis

Radiographs are required to characterize the type of P1 fracture and dictate the appropriate treatment. The radiographic examination should include at least four views: DP, LM, DLPMO, and DMPLO. Additional views at varying angles may be necessary to accurately document the fracture configuration with comminuted fractures as well as the presence or absence of joint involvement.

Midsagittal fractures are often readily apparent on the DP view, but some short, incomplete fractures may be difficult to see radiographically. In addition, multiple fracture lines on a single radiographic view do not always indicate more than one fracture because of the spiral nature of these fractures. This occurs because the fracture lines are not completely superimposed on the radiographic view.

Some P1 fractures may also be misdiagnosed as the nutrient foramen, especially in Standardbreds.[36] In horses with comminuted P1 fractures, the presence or absence of an intact bony strut that spans from the MCP/MTP to the PIP joints is one of the most important radiographic features. In addition, the degree of fracture displacement, the presence of comminution at the joint surfaces, and whether the fracture involves the PIP joint are all important radiographic features that may affect treatment. Similar to comminuted P2 fractures, CT can be very beneficial to more accurately assess the degree of comminution of P1 fractures and to aid in preoperative planning for surgery.

Treatment

Noncomminuted P1 Fractures

The decision on how to treat horses with noncomminuted fractures usually depends on the fracture type, fracture location and length, degree of displacement, and intended use of the horse. Treatment options include confinement with bandaging, confinement with a distal limb cast, internal fixation with lag screws and/or bone plates, external skeletal fixation alone, or internal fixation combined with external skeletal fixation.

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In general, most noncomminuted P1 fractures involve an articular surface (MCP/MTP and/or PIP joint) and are best treated with internal fixation using lag screws placed through stab incisions. Horses with displaced fractures that are not treated with internal fixation have a reduced chance of returning to performance.

Short, incomplete sagittal fractures can be treated conservatively with pressure bandaging and stall rest,[17] but most are treated with lag screw fixation.[27,32,33] Fracture propagation is a risk of treating short incomplete sagittal fractures conservatively, and this occurred in 3 of 85 racehorses in one study.[17] These fractures heal with a periosteal callus over the dorsal aspect of the fracture site, which does not appear to limit function. If the fracture has not healed after 3 months, lag screw fixation is recommended. However, a recent study found an improved prognosis in nonracehorses with short, incomplete sagittal fractures that were treated with lag screws.[33] Horses treated conservatively remained lame, only 1 of 4 horses had radiographic evidence of fracture healing, and 2 of 4 horses had catastrophic propagation of the fracture. In addition, another study indicated that all incomplete sagittal fractures greater than 15 mm should be treated surgically.[69]

Horses with long (greater than 30 mm) sagittal incomplete fractures that are to be used for racing should be treated with lag screw fixation placed through stab incisions followed by external coaptation (Figures 4.104B and 4.110). Two to three screws placed 20 mm apart are generally used depending on the length of the fracture. The most proximal screw should be placed within 5 mm of the most distal point of the sagittal groove in P1. Radiographic or fluoroscopic monitoring is recommended to ensure that the MCP/MTP joint is not entered and to document fracture compression. If a cast is used postoperatively, it is generally removed after 10–14 days. Others prefer bandage support only for recovery from anesthesia.

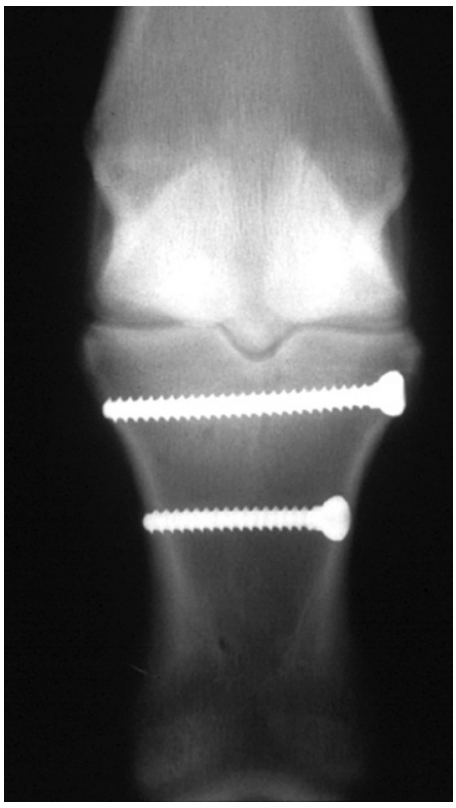


Figure 4.110. Dorsopalmar radiograph of the midsagittal incomplete P1 fracture illustrated in Figure 4.104B that was treated with two 4.5-mm lag screws placed through stab incisions.

Complete sagittal fractures that extend distally from the MCP/MTP joint to involve the PIP joint or that exit the lateral cortex are best treated by internal fixation and coaptation. These fractures are often displaced and can generally be better reduced with open approaches to P1 followed by lag screw stabilization. Plate fixation may also be used, depending on the fracture configuration. However, lag screw fixation through stab incisions similar to incomplete sagittal fractures may be sufficient, depending on the degree of displacement. A distal limb cast is usually recommended after surgery for 2–4 weeks, depending on the security of the fixation.

Bandaging and/or external coaptation has been used alone in cases in which breeding soundness is the objective or if there are economic constraints. In general, horses that are treated conservatively require about 4 months to become free of pain and lameness. They often develop considerable exostosis at the fracture site and secondary OA of the MCP/MTP joint, which may cause lameness when they resume work.[17,39]

Dorsal frontal incomplete or complete nondisplaced P1 fractures can be treated by rest and bandaging or by internal fixation using lag screws, depending on the fracture size. Needles placed into the MCP/MTP joint during the repair may help determine the proper placement of the screws. Arthroscopic examination of the fetlock joint should be considered to visualize the dorsal articular margin and debride damaged cartilage if needed. A cast or bandage may be used postoperatively. Complete dorsal fractures that extend into the PIP joint are best treated by lag screw fixation using an open approach or through stab incisions and external coaptation.[11] Needles placed in the MCP/MTP and PIP joint help guide the placement of 3–4 screws. A half-limb cast is recommended after surgery and is generally removed in 3 weeks. Fracture healing and return to training can be expected earlier following surgical treatment than with nonsurgical treatment of these fractures.

Distal articular fractures occur almost exclusively in the hindlimbs and appear to be more common in foals.[39] The acute fracture is generally best treated by lag screw fixation and external coaptation. If the fracture is chronic, secondary OA of the PIP joint is likely and arthrodesis of the PIP joint is recommended.

Physeal fractures are usually Salter–Harris type II fractures and are most common in weanlings.[11,39] Minimally displaced fractures are generally best treated conservatively with stall confinement and bandage support. In one report (four cases), all of these fractures healed with a moderate degree of malunion, but P1 remodeled so that a normal hoof-pastern axis was maintained.[39] If the fracture causes limb deformity and cannot be reduced, internal fixation may be required. If the PIP joint becomes subluxated as a result of the injury, arthrodesis of the PIP joint is recommended to realign the phalanges.

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Oblique or transverse fractures are uncommon and do not affect the physis or articular margins. Stabilization of the fracture with lag screws placed through stab incisions has been described.[39] Minimally displaced fractures may heal with external coaptation. Displaced fractures usually require open reduction and internal fixation with bone plates.

Comminuted Fractures

The objective for treatment of most horses with comminuted P1 fractures is usually to preserve the horse for breeding purposes or pasture soundness.[32] Even horses with only moderately comminuted P1 fractures that are repaired surgically rarely return to racing. In one report, zero of 33 Thoroughbred racehorses and 4 of 28 Standardbred racehorses returned to racing after surgery.[32] The goals of surgery are usually to restore the articular congruity of the joint/joints involved and to stabilize the fracture to maintain bone length. Methods for treatment of comminuted P1 fractures include:

a. External coaptation alone
b. External skeletal fixation alone (transfixation pin casts or Nunamaker skeletal fixator)
c. Lag screw fixation through stab incisions ± external skeletal transfixation (Figure 4.111)
d. Open reduction with lag screws and external coaptation
e. Open reduction with plates and screws and external coaptation
f. Reduction combined with transfixation pin casts (Figure 4.112)

Selection of the specific treatment method often depends on the fracture configuration, intended use of the horse, economic constraints, and preference of the surgeon. If internal fixation is considered, the implants should permit reapposition of the joint surfaces and provide longitudinal stability of P1. Internal fixation is usually recommended in horses with moderately comminuted P1 fractures that permit fracture realignment (Figure 4.111). With severely comminuted fractures, accurate fracture repair and longitudinal stability is sometimes impossible.

Horses with an intact cortex (strut of intact fractured P1 that extends from the proximal to distal joint surfaces) should be repaired with internal fixation. These horses have a much greater chance of surviving than horses without an intact strut of bone. In one study, 92% of horses with moderately comminuted P1 fractures treated with internal fixation had a successful outcome, whereas horses with severely comminuted P1 fractures had a much lower survival rate.[32] The intact strut of bone provides longitudinal stability to the fracture site as well as a solid piece of bone to which fracture fragments can be lagged.

External skeletal fixation is usually the treatment of choice to repair severely comminuted P1 fractures that lack an intact bony strut (Figure 4.112) and for those fractures that are open or have a severely compromised blood supply.[29,35,47,48] External fixation techniques reduce the risk of fracture collapse by using transcortical pins placed in the mid to distal portion of the third metacarpus/metatarsus, bypassing weight-bearing load through the fracture. Most horses should walk comfortably after surgery. Pin loosening, bone sequestration,

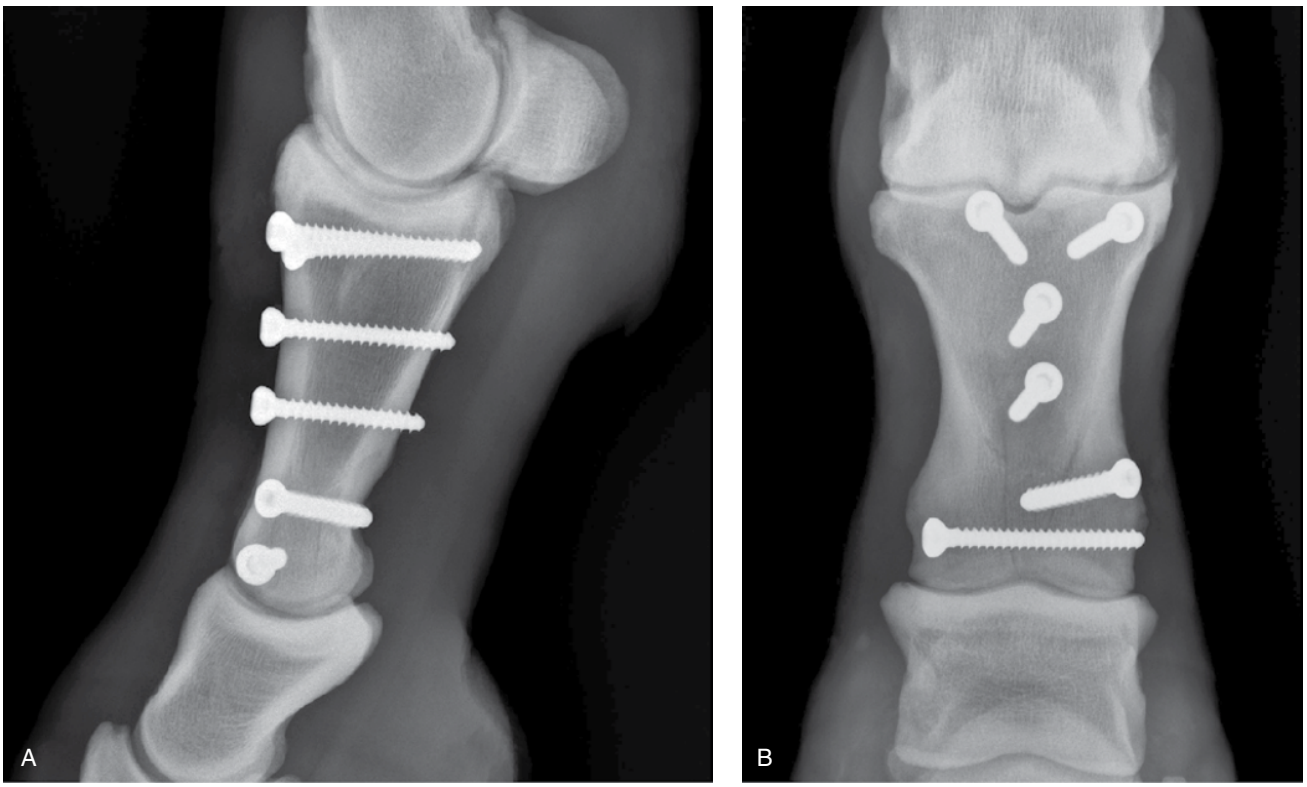


Figure 4.111. Lateral (A) and dorsoplantar (B) radiographs that were repaired with multiple lag screws placed through stab incisions.

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Figure 4.112. Dorsopalmar and lateral radiographs post-op and 3 months later at the time of hospital discharge of a severely comminuted P1 fracture treated with limited internal fixation and a transfixation pin cast.

and iatrogenic metacarpal/metatarsal fractures through the pin tracts are complications of using transfixation pin casts.[29,35] The addition of internal fixation is sometimes also used with external fixation to improve joint alignment and reduce longitudinal fracture collapse.

Casting alone can be used to treat some horses with comminuted P1 fractures, but it is less than optimal. Case selection is important and the fracture should be minimally comminuted and relatively stable to prevent axial collapse of the fracture.[39] Complications associated with using a cast alone to treat severely comminuted and unstable P1 fractures include (1) axial collapse of the fracture, potentially leading to an open fracture, (2) supporting limb laminitis, (3) excessive callus formation and OA of the MCP/MTP and PIP joints, (4) shortening of the pastern region, and (5) partial ankylosis of the MCP/MTP joint.[17,32] Because of these numerous complications, external coaptation alone is not recommended for treatment of horses with severely comminuted P1 fractures. In addition, casting or external skeletal fixation is not recommended for horses with moderately comminuted P1 fractures because of the very good success with internal fixation of these fractures.[32]

Prognosis

Noncomminuted Fractures

The prognosis for performance with noncomminuted fractures of P1 often depends on the configuration of the fracture, duration of the fracture until treatment, method of treatment, and breed and intended use of the horse.[11,17,27,69] In one study in racehorses, a significantly lower percentage of horses returned to racing following repair of complete sagittal fractures that extended into the PIP joint (46%) than following repair of short incomplete sagittal fractures (71%), long incomplete sagittal (66%), or complete sagittal fractures that extended to the lateral cortex (71%). The time from fracture to repair did not affect the outcome. Additionally the median number of races and the median fastest race times before and after surgery were not significantly different.[27] In another study with Standardbred racehorses, 89% of the horses returned to racing, but at significantly decreased performance levels.[69] In another study in young Thoroughbred racehorses, 70% of the horses treated conservatively with short, incomplete sagittal fractures raced, and 65% of the horses with long, incomplete sagittal fractures treated conservatively raced.[17] Horses with dorsal frontal fractures treated by lag screw fixation also appear to have favorable prognosis to return to performance.[11,39]

Comminuted Fractures

Horses with open or closed severely comminuted fractures that do not permit reconstruction of the fragments remain difficult to treat and have only a fair prognosis for survival, regardless of the treatment approach used. Moderately comminuted P1 fractures (those with an intact bony strut) can usually be repaired with internal fixation, and a 92% successful outcome has been reported.[32] Reasons for euthanasia of horses with comminuted P1 fractures include economic constraints, fracture collapse, iatrogenic metacarpal/metatarsal fractures, contralateral limb laminitis, and infection of the fracture site.[29,32,35]

DESMITIS OF THE DISTAL SESAMOIDEAN LIGAMENTS (DSLS)

There are three DSLs: the straight (superficial), paired oblique (middle), and paired cruciate (deep) (Figure 1.11). All of the ligaments originate from the base of the proximal sesamoid bones and intersesamoidean ligament. The straight ligament attaches distally to the proximopalmar/plantar aspect of P2, and the paired oblique ligaments attach to a triangular region on the middle and distal third of P1. The paired cruciate ligaments attach distally to the contralateral eminence of the proximal extremity of P1. There are also paired short ligaments that attach at the proximal articular margins of the MCP/MTP joint but are not considered to be part of the DSL complex. See Chapter 1 for further information regarding the anatomy of the DSLs.

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Desmitis of the oblique, straight, and cruciate DSLs occurs in all types of performance horses with injury to the oblique DSL being most common.[58] Horses that jump (e.g. event horses, show jumpers, field and show hunters, steeplechasers, and timber racehorses) and race and Quarter horses used for western performance such as reining, cutting, and barrel racing appear to be particularly prone to these injuries. However, injuries to these ligaments may not be the sole cause of lameness in many horses.[30,65]

The medial branch of the oblique DSL is more commonly injured than the lateral branch in some studies although others report equal medial and lateral frequency, and the hindlimbs are more often affected than the forelimbs.[30,58,65] When the straight DSL is injured, it is most often in the distal one-third of the ligament.[65] Injuries to the DSLs can be the primary injury or can be part of a complex of lesions, all of which contribute to some part of the current lameness. Horses with a valgus or varus limb conformation or long, sloping pasterns may be at increased risk for DSL injury.

Etiology

The DSLs are a functional continuation of the more proximally located suspensory ligament and are an important part of the suspensory apparatus that provides resistance to extension of the MCP/MTP joint during the stance phase. Hyperextension of the MCP/MCT joint can result in supraphysiologic strains in the suspensory apparatus, which may lead to injury to the DSLs. Although the DSLs in total provide this functional counter-resistance to extension, each ligament has a separate function that may account for the specific injuries that occur to these structures.

The straight DSL is the only unpaired ligament and is thought to contribute to sagittal stabilization of the MCP/MTP and PIP joints. The straight DSL would most likely be injured during hyperextension, but surprisingly, injury to this ligament is less common than to the oblique DSL.[58,61] The paired oblique DSLs are thought to play a prominent role in the limitation of rotation and abaxial movements of the MCP/MTP joint. Injuries to the oblique DSLs usually occur unilaterally, probably as a result of asymmetric loading caused by abnormal conformation, lateral/medial foot imbalances, a misstep, or poor footing. Injuries to the oblique DSL are more common than those to the straight or cruciate DSLs, although concurrent injuries to the straight and oblique DSLs have been reported.[58]

Clinical Signs

Horses with acute desmitis often present with a sudden onset of lameness. Mild swelling of the palmar/plantar surface of the pastern region may be present as a result of digital sheath effusion. The effusion is most commonly seen in acute cases (less than 3 weeks' duration), but soft tissue swelling is usually not apparent in most cases. Heat and pain with digital pressure may also be palpable in acute injuries.

Horses with chronic injuries may have a more insidious onset of lameness. Obvious heat, pain, and swelling are rarely present, and the location of the lameness often must be localized with perineural anesthesia. Sometimes there will be pain on deep palpation of the ligament. The lameness is usually mild to moderate in severity, positive to fetlock or phalangeal flexion, and worsened when the affected limb is in the inside of the circle. Palpation of the DSLs is best performed with the foot held off the ground and the MCP/MTP joint flexed so the flexor tendons are relaxed.

Swelling of a DSL must be differentiated from swelling of the medial or lateral branch of the SDFT, which is also located in the midpastern region. Passive fetlock and phalangeal flexion are commonly painful, and direct digital pressure over the swollen region of the DSL for 30 seconds may increase the signs of lameness.

Although the clinical findings may indicate a problem in the palmar/plantar pastern region, perineural anesthesia should be performed to rule out concurrent involvement of the foot. Perineural anesthesia of the PD nerve at the base of the sesamoid bone (basisesamoid block) should improve the lameness in most cases as will intrathecal block of the DFTS.[30] However, an abaxial sesamoid or low four-point block may be necessary if the ligament injury is located proximally in the pastern.[4] In addition, other concurrent problems should be closely evaluated because a recent report suggested that lesions of the DSLs found on MRI were the sole cause of lameness in only 2 of 58 horses.[65] It should be kept in mind that most horses with obvious DSL injury on ultrasound exam would not have received an MRI by the authors. See Chapter 2 for more information on perineural anesthesia.

Diagnosis

Diagnostics that may help document an abnormality to one of the DSLs include radiography, ultrasound, and MRI. Radiographic abnormalities that may suggest a previous or concurrent injury of a DSL include enthesophyte formation, avulsion fractures/fragments, and dystrophic mineralization within one of the DSLs. Enthesophyte formation at the attachment of the oblique DSL at the palmar/plantar aspect of P1 is a relatively common finding and may be incidental (Figure 4.113). Enthesophyte formation at the proximal aspect of P1 and at the base of the proximal sesamoid bone is also believed to be evidence of injury to the cruciate or short DSL.

Fractures/fragments of the base of the proximal sesamoid bone can involve the DSLs.[4] Fragments from either the dorsal aspect of the base of the proximal sesamoid bone or proximal palmar/plantar articular margin of P1 typically involve the short DSL.[55] Bone fragments have also been observed on the nonarticular proximal extremity of P1 and at the base of the sesamoid bones.[4] These fracture fragments may involve the oblique, cruciate, or short DSLs.[4,55] Dystrophic mineralization associated with the DSLs may also be present radiographically, usually at the base of the sesamoid bones. It is important to differentiate dystrophic mineralization from avulsion fractures at the base of the sesamoid bones because fracture fragments can and should be removed, whereas there is usually no treatment for mineralization.[4]

Sonographic evidence of acute desmitis of the DSLs is manifested by a diffuse increase in ligament size, fiber

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Figure 4.113. Enthesophytes on the palmar lateral cortex of P1 can be seen on this DLPMO radiograph of the pastern. The clinical significance of these lesions is questionable but may suggest a previous injury at the insertion site of one of the DSLs or far less commonly the insertion of the distal digital annular ligament (DDAL). This horse was sound and radiographs were taken as part of a prepurchase examination.

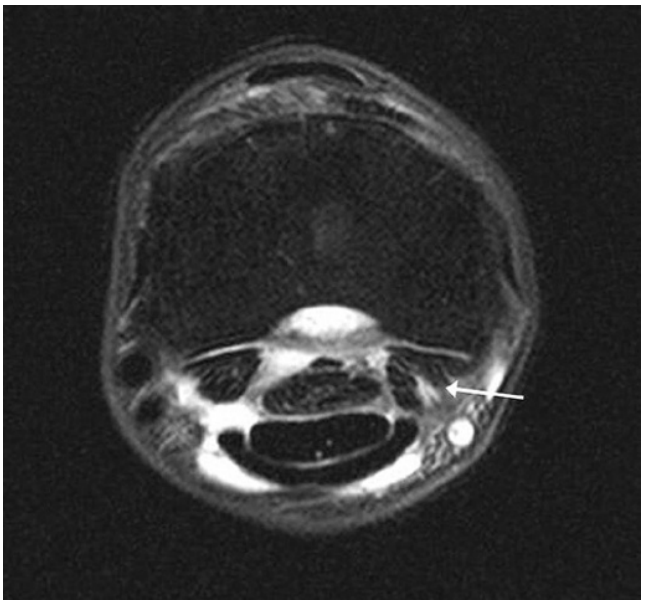


Figure 4.114. Transverse proton density MRI image showing high signal intensity (arrow) in the lateral branch of the oblique DSL in the pastern region.

disruption, discrete core lesions, and periligamentous fluid surrounding the affected ligament.[13,55] The anechoic space between the ligament and the SDFT is often reduced in size with desmitis of the oblique DSL. Chronic sonographic changes may include varying degrees of the acute abnormalities within the ligament, hyperechoic areas consistent with dense scar tissue formation, and dystrophic mineralization.[13,55] Periosteal proliferation in areas of ligament attachments may appear as irregular contours on the bone surface. Basilar sesamoid avulsion fragments, desmitis of the ipsilateral branch of the suspensory ligament in the same limb, and fragments off the proximopalmar/plantar aspect of P1 may occur concurrently in more chronic cases. Ultrasonographic identification of cruciate DSL injuries is difficult due to the location of these ligaments and therefore may be underdiagnosed.

Ultrasonography of the palmar/plantar pastern region is difficult to perform and requires experience to become proficient. There are numerous soft tissue structures that must be ruled out as potential problems. Therefore, only obvious abnormalities within the DSLs may be recognized. In a recent study to describe the normal appearance of the DSLs using MRI, 80% of the lesions found with MRI were not detected with ultrasonography.[65] Although ultrasound can be useful to diagnose problems in the DSLs, lack of ultrasound abnormalities does not rule out a problem within the DSLs. Currently, MRI is the best diagnostic tool to make a definitive diagnosis (Figure 4.114).[58,61] See Chapter 3 for additional information on MRI.

Treatment

In general, injuries to the DSLs are treated very similarly to other soft tissue problems such as tendinitis. In acute cases, confinement, cold therapy, pressure/support wraps, and administration of NSAIDs are recommended. Cold therapy in the form of an ice/water slurry applied for 30 minutes twice a day during the acute inflammatory phase is beneficial. Pressure/support bandages can be applied in between the cold treatments and maintained for 2–3 weeks or as needed. NSAIDs can be administered for up to 2–3 weeks' post-injury. Feet and hoof-pastern axis imbalances should be corrected if possible.

Once a definitive diagnosis has been made, a 6-month rest and rehabilitation program is currently recommended.[58,61] This usually involves a short period of stall confinement depending on the severity of the injury (3–6 weeks), followed by increasing periods of hand-walking and controlled exercise. Hand-walking exercise usually begins with 5 minutes once or twice a day, 3–5 days a week, and then increases 4–5 minutes/week. Clinical evaluation should be performed at 4–6 weeks, and if the horse has improved, controlled exercise can be increased.

If abnormalities were apparent on ultrasound, reevaluation is recommended 2–3 months post-injury. Further controlled or free exercise recommendations are made depending on the ultrasound findings. In one study of 27 horses with DSL injuries, 76% of the horses successfully resumed performance following a 6-month controlled exercise program.[58]

Adjunctive treatments that may be used in addition to the rehabilitation protocol include extracorporeal shockwave, ligament splitting, injection of the DFTS

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with corticosteroids and HA,[58] and intralesional treatment of the damaged ligament with stem cells or platelet-rich plasma (PRP). Nonarticular base sesamoid fragments that may be associated with DSL avulsion injuries should be removed using a "keyhole" surgical approach through the DFTS.[4] With this approach, fragments are localized with needles and removed using arthroscopic rongeurs. Nine of 10 surgically treated horses returned to their intended use.[4]

Prognosis

The prognosis for horses with DSL injuries to return to performance has historically been considered to be guarded because of the high probability of re-injury. However, more recent studies have indicated a much better prognosis. Seventy-six percent, 66%, and 90% of horses with DSL injuries or avulsion fractures of the proximal sesamoid bones returned to performance following treatment.[4,58,61] However, recurrence of DSL desmitis is always a possibility, similar to other soft tissue injuries. In addition, concurrent musculoskeletal problems such as PIP joint OA, navicular syndrome, and suspensory desmitis often reduce the prognosis for full recovery.

DESMITIS OF DIGITAL ANNULAR LIGAMENTS

Within the pastern region there are two superficial ligaments that form a retinaculum for the flexor tendons: the proximal digital annular ligament (PDAL), which inserts medially and laterally on the proximal and distal tubercles of P1, and the distal digital annular ligament (DDAL), which runs from the medial and lateral borders of distal P1 to the palmar/plantar aspect of P3 (Figure 1.11). Primary injury to both the PDAL and the DDAL can be a cause of lameness in the horse.[7,22]

Etiology

Similar to injury to other ligaments, there is often an initial small injury that leads to inflammation, fibrosis, and loss of elasticity, which leads to functional shortening of the ligament and constriction over the flexor tendons. Continued athletic use then causes additional injury to the previously damaged and functionally shortened ligament leading to recurrent inflammation, fibrosis and loss of elasticity, and further functional shortening. The permanent fibrosis and resultant constriction over the flexor tendons is likely why horses generally do not respond to rest and medical therapy.

Clinical Signs

Affected horses have a mild to moderate and consistent lameness and minimal to no external signs. Horses may be positive to flexion of the distal limb.

Diagnosis

A palmar (plantar) digital nerve block or intrathecal DFTS block will greatly improve the lameness associated with injuries to the PDAL and DDAL.

Ultrasound of the PDAL is easily performed and the normal dorsal to palmar/plantar thickness of the ligament is 2 mm.[22] Desmitis of the PDAL is seen as a thickened ligament with heterogeneous echogenicity. Due to location within the foot, distal to the DFTS, it is difficult to thoroughly assess the DDAL with ultrasound. High-field MRI is the preferred technique for diagnosis of injury to the DDAL (Figure 4.115). The thickness of the normal DDAL is also 2 mm. When injured, the thickness can increase up to 10 times the normal thickness.[7]



Figure 4.115. Transverse and sagittal PD-weighted images showing thickening and mixed signal intensity DDAL. The dotted line on the sagittal image represents transverse slice location.

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Treatment

Surgical transection of the PDAL and DDAL is the treatment of choice. In one report, five of seven horses with DDAL injury that had surgical transection of the DDAL were able to return to performance, two of which had failed to respond to rest and medical therapy.[7]

Prognosis

The prognosis for return to work after surgical transection of the DDAL appears to be good, although only one report is currently available.[7]

SDFT AND DDFT ABNORMALITIES

In general, injuries in the pastern region to the SDFT occur most frequently in the forelimbs. Chronic DFTS effusion seems to be more common in the hindlimbs, and injuries to the DDFT within the DFTS occur most frequently in the hindlimbs; however, in horses that jump, DDFT injury within the DFTS was more common in the forelimbs.[2,64] Injuries to the DDFT that are associated with navicular syndrome are covered under the foot section. Injuries involving the SDFT most commonly involve the branches of the SDFT located outside the DFTS.[13] The SDFT branches at the level of the MCP/MTP joint, giving rise to medial and lateral branches that insert on the palmar/plantar eminences of P2.[75] Abnormal conformation such as a long pastern or an underrun heel may predispose the horse to injury of the SDFT branch. Injuries to the DDFT within the pastern are nearly always within the DFTS, often cause effusion of the sheath, and may contribute to chronic tenosynovitis of the DFTS (Figure 4.116). In two different studies of horses with DFTS tenosynovitis, injuries to the DDFT were found in 44 of 76 horses[64] and 101/130 horses.[2]

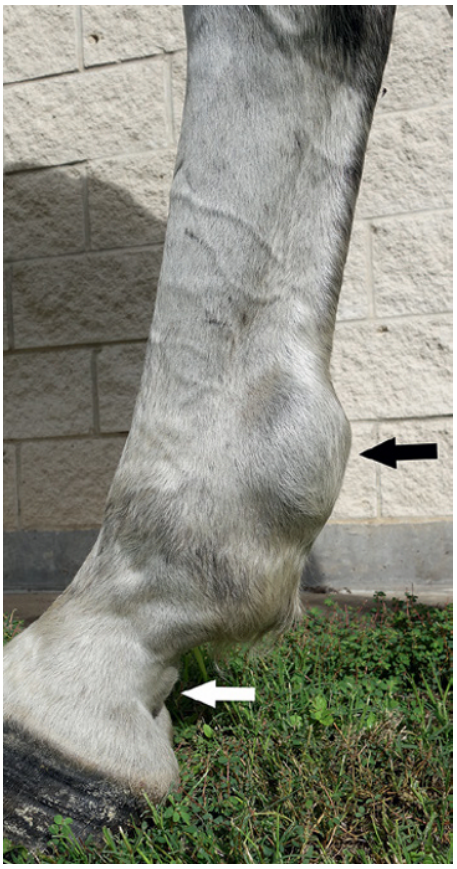


Figure 4.116. This horse was 4/5 lame in the left hindlimb and had severe effusion of the digital flexor tendon sheath and chronic DDFT injury. Arrows demonstrate the largest outpouchings of synovial effusion proximolateral and medial (black) and plantarodistal (white). Lesions of the DDFT within the tendon sheath appear to be more common in the hindlimbs than the forelimbs.

Etiology

Injuries to the SDFT in the forelimbs are usually associated with hyperextension of the MCP joint, resulting in nonphysiologic stretching and overload of the SDFT.[54] These injuries occur commonly in racehorses, but why some horses get SDFT injuries in the pastern compared to the metacarpal region is unknown. The cause of DDFT injuries within the tendon sheath is unknown, but hyperextension of the MCP/MTP joint and overstretching of the tendon are also likely.[2] It seems as though both SDFT and DDFT injuries in the pastern region occurs more frequently as a result from a single traumatic event as compared to flexor tendon injuries at the level of the metacarpus (tarsus).

Clinical Signs

SDFT Branch Injuries

Lameness usually occurs at the onset of injury with focal heat, swelling, and sensitivity noted on palpation. However, careful palpation and comparison of the medial to lateral branches are important to detect differences in size, heat, and pain because these injuries can be easily missed. Generally, swelling develops within 3–4 days and is usually uniaxial on the limb. The medial SDFT branch appears to be more frequently injured than the lateral branch, and avulsion fractures of P1 at the insertion of the SDFT branch occur infrequently. Some SDFT injuries and damage to the manica flexoria may occur within the DFTS and result in tendon sheath effusion.[14,64]

DDFT

Deep digital flexor tendinitis occurs in a variety of horses and typically presents as an acute-onset, unilateral moderate to severe lameness that is persistent.[54] Heat, pain, and swelling of the DDFT itself are usually not palpable because the damage is often located within the DFTS, which can be quite effusive (Figure 4.116). Lameness is often worse on a soft surface and generally improves with perineural anesthesia of the palmar/plantar nerves at the level of the proximal sesamoid bones. Distension of the DFTS often occurs in conjunction with the injury, and many horses present with chronic tenosynovitis of the DFTS of undetermined cause. The use of MRI and/or diagnostic tenoscopy in addition to ultrasound has increased the frequency of a definitive diagnosis of DDFT injury[2,64,78] (Figure 4.117). If the DFTS is distended, intrasynovial anesthesia of the sheath is the preferred method to confirm the location of the lameness.

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Figure 4.117. Ultrasound images (left) and contrast tenogram (right) demonstrating a lateral marginal lesion of the DDFT within the tendon sheath at the level of the proximal sesamoid bones (left; arrows).

Diagnosis

Ultrasonography is currently the most commonly used method to diagnose tendon injury and can be used in the pastern for branch lesions of the SDFT, core lesions of the SDFT, and some abnormalities of the DDFT. However, ultrasonographic examination in the pastern region may result in false-negative results.[2,14,64] This is especially true for the DDFT, where surface and longitudinal lesions are more common than core lesions and more difficult to visualize with ultrasound.[14,77,78] It is important to note that the DDFT becomes bilobed at the level of the pastern. Each lobe should be similar in size and shape. A lesion may involve one or both lobes and is typically characterized by enlargement and alteration of the tendon with or without a hypoechoic region (Figure 4.118), making it more difficult to visualize with ultrasound. Dystrophic mineralization may be seen with chronic injuries (Figure 4.119).[76]

In cases that have been blocked to the DFTS or with nonspecific signs of chronic tenosynovitis of the DFTS, diagnostics beyond ultrasound should be considered.[77,78] An MRI examination is superior to an ultrasound examination to characterize the location, type, and severity of damage to both the SDFT and DDFT within the pastern (Figure 4.120). When MRI is not available, a contrast tenogram may be useful to define marginal tears of the DDFT as well as tears of the manica flexoria with improved positive and negative predictive values over

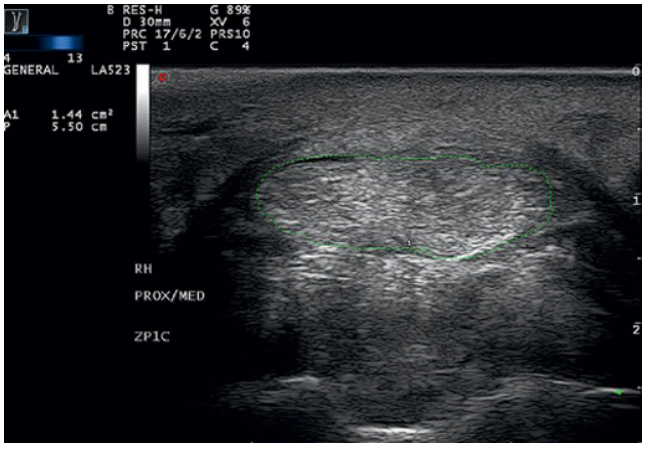


Figure 4.118. There is enlargement and mixed echogenicity of the lateral lobe of the DDFT.

ultrasound.[18,24,25] Five- to 7-mL contrast is injected with or without 10-mL local anesthetic, the horse is walked 4–5 strides to distribute contrast, and then a lateral to medial radiograph is made.

Tenoscopy of the DFTS is a definitive diagnostic tool to document intrathecal lesions of both SDFT and DDFT that may not be visible with ultrasound. An added benefit of tenoscopy is the ability to debride

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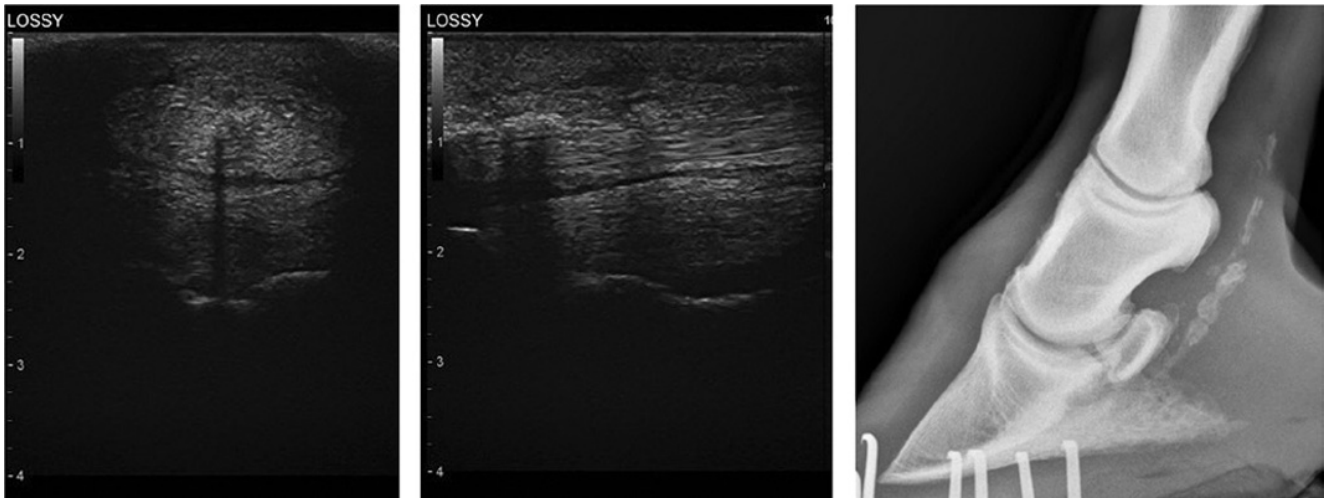


Figure 4.119. There is dystrophic mineralization of the DDFT visible on both the ultrasound and radiographic images of this horse.

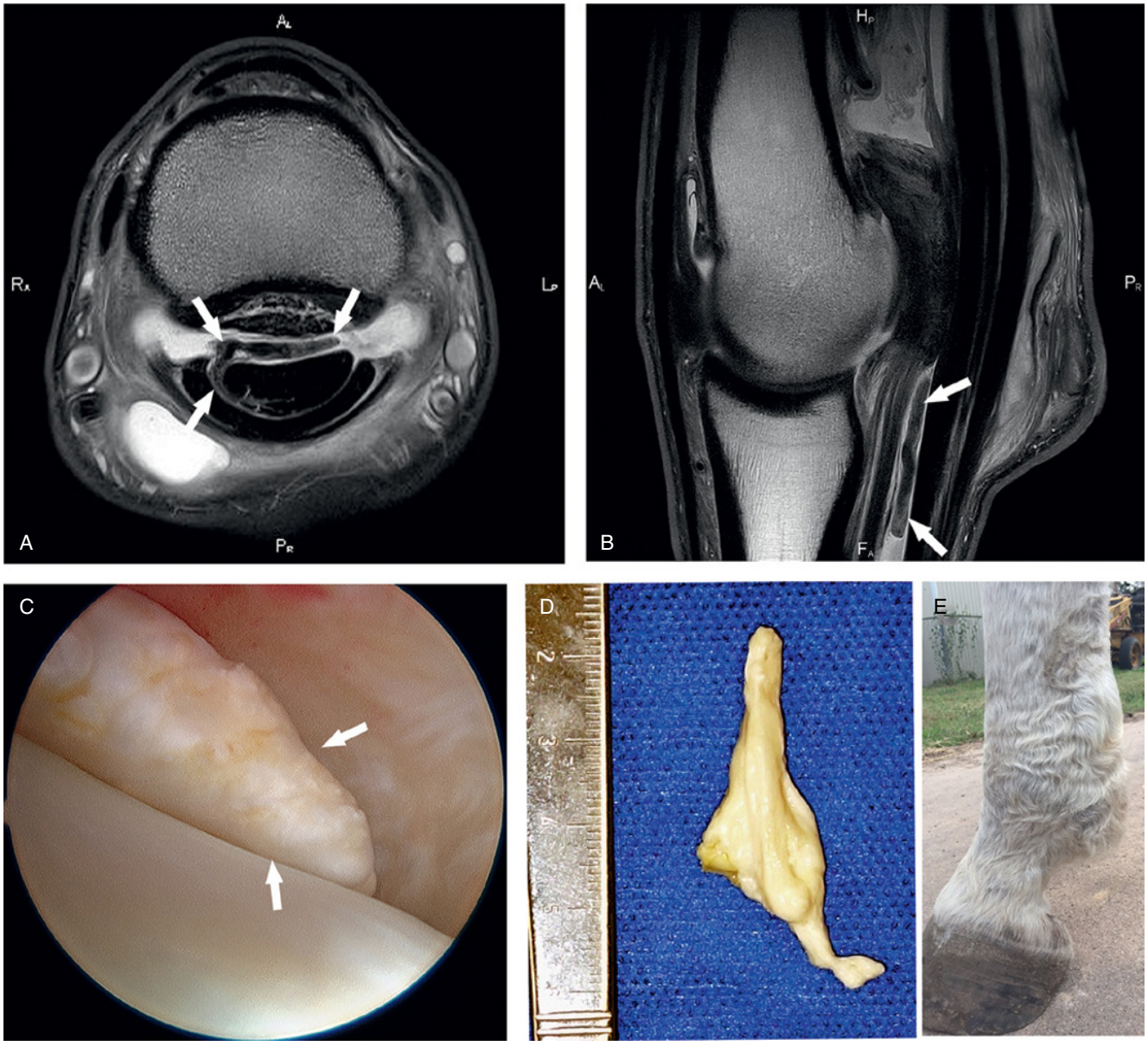


Figure 4.120. Images of the same horse in Figure 4.116. (A) transverse and (B) sagittal PD-weighted images of the extruded fibers from the plantar margin of the DDFT wrapping around the dorsal edge of the DDFT (white arrows) in the proximal pastern. (C) The chronic DDFT extruded fibers at (C) tenoscopy after (D) tenoscopic removal. The plantar annular ligament was also transected. (E) Photograph of the limb 8 months later with marked improvement of DFTS effusion and minimal lameness. The DDFT injury was not recognized as a surface split with extruded fibers by ultrasound.

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marginal tears, especially those with extruded fibers or adhesions, and tears in the manica flexoria.[14,64,77,78] If the lameness is confirmed to the DFTS and effusion is present, tenoscopy is strongly recommended, especially if the ultrasound findings are equivocal.[14,77] This is especially true if the DFTS has been treated by intrathecal administration of anti-inflammatory drugs with lack of resolution or rapid recurrence of lameness.

Treatment

Treatment depends on the location, type, and severity of the lesion. Branch lesions of the SDFT are usually treated with a controlled rehabilitation program similar to any bowed tendon injury; however, 6–12 months is typically needed. If the SDFT lesion is within the DFTS or if DFTS effusion is present, surgical exploration via tenoscopy is often helpful to further diagnose the specific problem and debride the damaged tendon.[77,78] Lesions of the DDFT at the level of the pastern or fetlock canal are often within the DFTS, and tenoscopy is often beneficial because these lesions are often difficult to diagnose and treat.[2,64,77,78] An annular ligament desmotomy may be performed at the time of surgery if considered appropriate.[2,77] When there is distension of the DFTS and ultrasound does not reveal tendon injury, medication of the DFTS with corticosteroids, HA, or interlukin-1 receptor antagonist protein (IRAP) can be performed. If there is lack of lameness resolution or recurrence of lameness, additional diagnostics such as tenoscopy should be pursued. Intralesional injection of regenerative products such as PRP or stem cells directly into the damaged tendon can also be performed. See Chapter 8 for more details on these treatments.

Prognosis

In general, soft tissue injures of the SDFT and DDFT in the pastern region can be difficult to diagnose and tend to have a worse prognosis than SDFT and DDFT injuries in the metacarpal or metatarsal region. Horses with SDFT branch injuries in the pastern region are thought to have a poorer prognosis to return to racing than SDFT injuries in the metacarpal region, with more frequent recurrence of injury. Following tenoscopic debridement, surface lesions of the DDFT within the DFTS result in only 38%–42% of horses returning to previous level of performance.[2,64] Without tenoscopic debridement of surface DDFT lesions, especially when there are extruded fibers, the prognosis appears to be poor. In one study, 10 of 17 horses with longitudinal tears of the DDFT returned to their previous level of work.[77] In another study of 76 horses with chronic DFTS tenosynovitis, sheath distension was eliminated in 33% and improved in 69% of the horses.[64] In the same study, 68% of the horses were sound, and 54% returned to their preoperative level of performance.[64]

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THE FETLOCK

Matt Brokken and Alicia Bertone

OSTEOCHONDRAL FRACTURES AND FRAGMENTATION OF THE PROXIMAL PHALANX

Osteochondral fractures of the proximal aspect of the first phalanx (P1) can occur in any horse used for performance but are particularly common in horses that exercise at speed. Most fractures of this type involve the dorsal articular surface of proximal P1 in the forelimb, medial or lateral to sagittal midline. The left forelimb and medial aspect of the joint are affected more often. Chip fractures from the dorsodistal aspect of the third metacarpal/metatarsal (MC/MT) bone also occur but are less common.

Other less frequently occurring fractures of P1 include fractures of the lateral and medial eminences of the proximopalmar (or proximoplantar) surfaces and avulsion fractures of the midproximal palmar articular margins just below the sesamoid bone. In addition, there are fragments associated with the palmar or plantar proximal aspect of P1 that may be traumatic or developmental in origin.

Etiology

Dorsoproximal P1 "chip" fractures are caused by concussion and overextension of the fetlock joint. Typically, similar to osteochondral fractures of the dorsal aspect of the carpus, these fractures occur toward the end of the race in racehorses. From the appearance of the fractures, it seems that excessive overextension of the fetlock joint is probably involved. Overextension places stress on the dorsoproximal aspect of P1 as it is pressed against the distal third metacarpal bone. Limb fatigue is a factor in overextension of the fetlock joint, noted at the end of races when the back of the fetlock may contact the ground (running down). The fracture most frequently occurs medial to midline most likely due to the fact that the medial aspect of proximal P1 is more prominent and extends slightly more proximal than its lateral counterpart.

Fractures of the proximopalmar (or proximoplantar) eminence are also a result of trauma (most likely torsional) and may be associated with complete or partial tearing of the collateral ligament of the fetlock joint with possible subluxation/luxation.[43]

The cause of proximal palmar/plantar P1 fragments is debatable. In most horses, the belief is that they are developmental.[36] Axial fragments are classified as type I and are articular. Type I fragments can be associated with lameness at speed with clinical signs similar to dorsoproximal P1 fractures. Type II fragments are located abaxially and typically have minimal articular component; however, they can be a source of performance-limiting lameness.

Clinical Signs

Synovitis of the fetlock joint indicated by distension of the palmar/plantar pouch (between the suspensory ligament [SL] and the palmar or plantar surface of the cannon bone) is commonly found. Horses in race training often present with a history of lameness, which increases after exercise, and a workout or a race may cause the horse to be markedly lame. After prolonged rest, the horse may seem to be sound, only to go lame again when returned to training. Horses with fractures of the palmar/plantar eminence of proximal P1 will typically have an easily observable lameness (normally lame at the walk) along with varying degrees of swelling/edema associated with the fetlock region.

Some horses, particularly with chronic chip fractures, have only a small amount of swelling or lameness to indicate that there is a chip fracture. There may be fibrous enlargement on the dorsal surface of the fetlock joint that is easily palpated. It is difficult to produce pain in the affected region by digital pressure, but some heat may be detected over the dorsal surface of the joint. Flexion of the affected fetlock often elicits pain, and a fetlock flexion test usually exacerbates the lameness. If the examiner is unsure of this response, it should be compared with the opposite fetlock.

In most cases it is not necessary to use local anesthesia to identify chip fractures within the fetlock. If the examiner is suspicious that the fetlock is involved, radiographs should be taken. However, if confusion exists regarding the contribution of the fetlock to the lameness, either intrasynovial anesthesia of the fetlock (preferred) or a low four-point nerve block (proximal to the fetlock) can be performed. Diagnostic analgesia is recommended for some fragments of the palmar/plantar aspect of proximal P1 to determine their clinical significance.

Diagnosis

A definitive diagnosis is best made with radiographic examination. A minimum of four views (five views including the flexed lateral) should be obtained. Oblique radiographs should be taken to determine whether the chip is on the medial or lateral side of the midline (Figure 4.121). For palmar/plantar fragments, oblique radiographs raised ~20° from horizontal can be helpful in limiting the superimposition of the sesamoids on proximal P1 (Figure 4.122). However, it is important to note that the downward oblique projections may make detection of dorsal P1 fractures more difficult. It is important to radiograph the contralateral fetlock, because bilateral fractures are not uncommon, and clinical signs may not appear until the horse is back in training.[1,24,28,49,108] Ultrasonography also may be used to diagnose chip fractures of the dorsal aspect of P1 and concurrent proliferative synovitis of the fetlock synovial pad if present. Acute proximal eminence fractures can be difficult to appreciate on the lateral and dorsopalmar/plantar radiographic views and are best detected on oblique projections (Figure 4.123).

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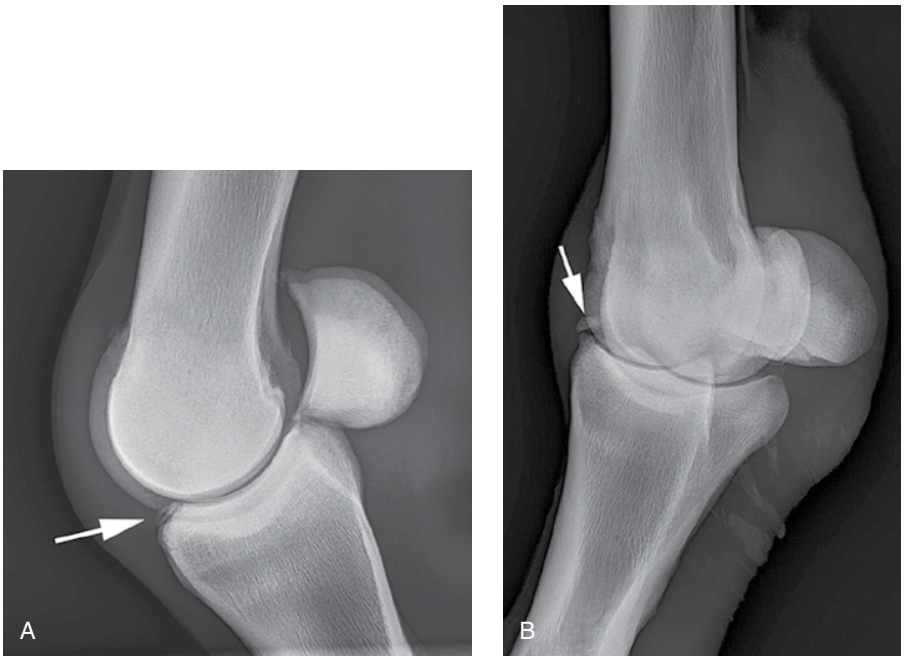


Figure 4.121. (A) Flexed lateral radiograph of the fetlock demonstrating a small fragment off the dorsoproximal aspect of P1 (arrow). (B) DLPMO radiograph of a horse with a rounded fragment off of the dorsomedial aspect of proximal P1 (arrow).



Figure 4.122. DMPLO radiograph demonstrating a rounded fragment off of the plantaromedial aspect of proximal P1 (arrow).

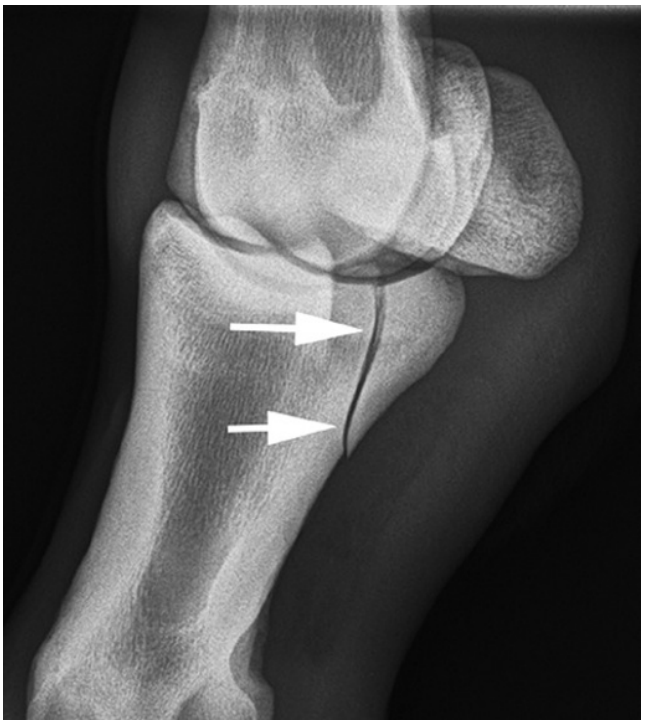


Figure 4.123. Oblique radiograph of the fetlock in a horse with a palmar eminence fracture of proximal P1 (arrows).

Treatment

Arthroscopic removal of small chip fractures in the dorsal or palmar/plantar aspect of the joint is the treatment of choice for the greatest chance of a quick return to full performance.[24,49,56,108] Arthroscopic evaluation permits removal of multiple fragments, visualization of the entire joint, and surgical treatment of lesions that may not have been identified on radiographs (Figure 4.124). Lesions commonly seen in association with P1 chip fracture include proliferative synovitis of the dorsal metacarpal synovial pad (32% of which had chip fractures) and cartilage erosion of the metacarpal

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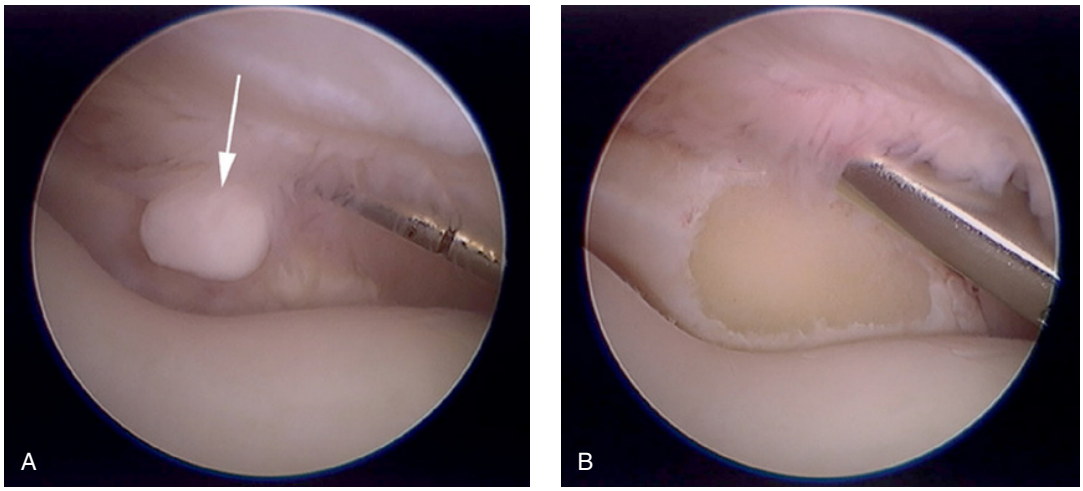


Figure 4.124. Arthroscopic images of a small dorsoproximal P1 fragment before (A; arrow) and after (B) fragment removal.

condyle.[28,49] It is recognized that some small nondisplaced fractures can be treated successfully with adequate rest for 120 days. If training continues, these fragments often displace and cause adjacent articular cartilage erosion. Two to four months of rest and controlled exercise is recommended before the horse is returned to a regular exercise/training program. This somewhat depends on the degree of joint damage and cartilage debridement. In some horses, small frontal fractures of the dorsal margin of proximal P1 are identified. These fractures can heal with conservative management but if lameness persists, some have advocated compression of the fracture with small screws (2.7 or 3.5 mm).[106]

Fractures of the palmar/plantar eminence can be treated conservatively, but due to the soft tissue attachments, there typically will be a large degree of osseous remodeling around the fracture due to movement. Osteoarthritis (OA) of the fetlock joint typically results in varying degrees. If the fragment is large enough, internal fixation with lag screw(s) may allow for a faster return to function and may lessen the development of OA. Removal of smaller fragments is also an option. Typically, these horses are in a rest and rehabilitation program at least 4–6 months to allow time for the bone and adjacent soft tissues to heal. The benefit of removing palmar/plantar fragments to improve performance has been questioned based on a study that indicated there was no difference in racing speed, career earnings, or lifetime starts between Standardbred racehorses with the fracture and those without.[19]

Prognosis

The prognosis is usually good to excellent for treatment of proximal dorsal chip fractures, but it is somewhat dependent on the size and number of chip fractures, their duration, whether or not corticosteroids have been injected, amount of concomitant articular cartilage damage, and degree of OA. Several reports indicate that the prognosis for return to athletic performance, including racing performance, is good to excellent (approximately 80%) with arthroscopic surgery to remove the fragment.[19,20] The presence of other fetlock lesions noted at arthroscopy decreased the prognosis in racing Thoroughbreds, but success was still good (greater than 70% return to racing).[24,49,56,108]

Small acute, nondisplaced fetlock chip fractures usually have a good prognosis with conservative treatment. Arthroscopic surgical removal may still be elected in these cases because the convalescence is shorter after surgical removal (often under 30 days) than for the bone to heal (90–120 days) and the risk of fracture displacement or refracture is eliminated. Factors that lower the prognosis include extreme large size of the fragment, chronicity, degree of synovitis/capsulitis, and amount of OA present. Standardbred racehorses often have chronic joint changes associated with dorsal P1 fractures. In general, racehorses that develop dorsal P1 fractures have reduced lifetime earnings compared with racehorses that do not develop these fractures.[59]

The prognosis for proximal palmar/plantar fractures that require compression screw fixation depends on the degree of initial trauma at the time of fracture. Often this is not fully appreciated until the radiographs are taken 3–4 months after surgery. The prognosis after removal of smaller proximal palmar fractures is considered to be very good, although the benefit of removal has been questioned in Standardbred racehorses.[19,20]

FRACTURES OF THE PROXIMAL SESAMOID BONES

Fractures of the proximal sesamoid bones are common injuries in racing Thoroughbreds, Standardbreds, and Quarter horses.[6,15,18,54,67,78,79,83,84,86,92] These fractures take various forms including apical, abaxial (articular and nonarticular), midbody, basilar (articular and nonarticular), sagittal, and comminuted. Combinations of fracture types such as apical/abaxial fractures may also occur. The forelimbs are most frequently affected in the Thoroughbred (right forelimb) and Quarter horse, whereas the hindlimbs are more frequently affected in the Standardbred (left hindlimb). Most of these fractures distract as a result of the pull of the SL proximally and the distal sesamoidean ligaments (DSLs) distally.

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Severe fractures involving the sesamoid bones and metacarpophalangeal/metatarsophalangeal joints are one of the most common types of fatal musculoskeletal injuries in racing Thoroughbreds and Quarter horses.[7,46,68,77] In a recent study in Thoroughbred racehorses, fractures of the proximal sesamoid bones represented 55% of all catastrophic fractures, while the most common noncatastrophic fractures involved the carpus and first phalanx.[91]

Fractures of the apical portion of the sesamoid bone are the most common, comprising more than 88% of sesamoid fractures. In Thoroughbred racehorses 2 years or older, 64% involve hindlimbs and 36% forelimbs.[78] Sesamoid fractures are most common (53.4%) in 2-year-olds, and then 3-year-olds (23%).[78,79] Apical fractures are frequently articular, singular, rarely comminuted and usually involve less than one-third of the bone (Figure 4.125). Apical fractures occur most frequently in the lateral sesamoid bone of the left hindlimb (42.8% of fractures) compared with the right hindlimb (36.6%) in Standardbreds, whereas a more equal distribution is observed in Thoroughbreds.

Basilar fractures are less common (6% of sesamoid fractures in Standardbreds). They represent an avulsion fracture associated with the DSLs and may be comminuted. These fractures are more common in the Thoroughbred than in the Standardbred and can be small articular, transverse articular, or nonarticular (Figure 4.126).[15,84,92]

Abaxial fractures are uncommon in Standardbreds (3%), and more common in Thoroughbreds and Quarter horses.[84,92] These fractures can either be articular or nonarticular and can be difficult to diagnose and may require an additional tangential projection on the radiographic examination to identify their exact location, or they can be identified on the dorsopalmar/plantar projection.[8,66]

Midbody transverse sesamoid fractures are seen most frequently in Thoroughbreds, older Standardbreds (mean age 6.5 years), and young foals under 2 months



Figure 4.126. DP radiograph of a Thoroughbred racehorse with a thin fracture of the base of the proximal sesamoid (arrow). Also note the other degenerative changes present in the fetlock (marked sclerosis of the palmar condyles of the distal third metacarpal bone, remodeling of the fetlock joint space; primarily on the side of the base sesamoid fragment).

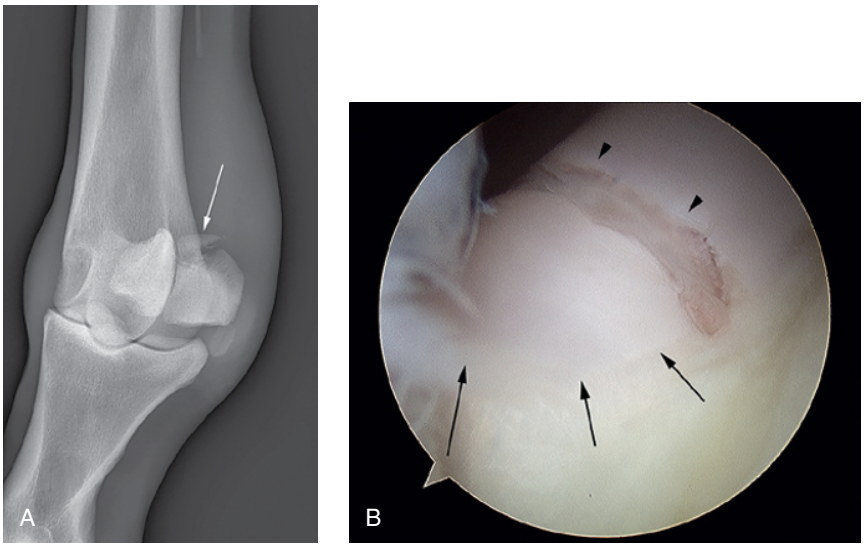


Figure 4.125. (A) Oblique radiograph of a Standardbred racehorse with a large, displaced apical sesamoid fracture (arrow). (B) Arthroscopic image of a horse with a large apical sesamoid fracture (arrows are fragment; arrowheads are fracture line).

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of age.[18,32,44,74] Fractures roughly separate the bone into equal portions and invariably enter the fetlock joint (Figure 4.127). Due to the distractive forces of the SL proximally and the DSLs distally, most of these tend to separate. Infrequently, they remain in apposition, but may separate at a later time. If both sesamoid bones are fractured, they usually become distracted and the suspensory support apparatus is lost.

Etiology

The cause of most proximal sesamoid bone fractures is excessive tensile forces related to fetlock hyperextension that maximally loads the sesamoid bones. Fetlock extension is greatest at the end of a race due to fatigue of the digital flexor muscles that support the fetlock. The bone fails when the sesamoid bone can no longer withstand the distraction forces applied to it by the SL and DSLs. The muscle fatigue factor is illustrated when young foals that are placed on pasture fracture their sesamoid bones while running to keep up with the dam.[32,44] It has also been reported that 70% of Quarter horse fatal musculoskeletal injuries occur within 10 yards of the finish line regardless of the race distance.[7]

Other factors such as poor conditioning, improper trimming and shoeing, and poor conformation create additional stresses on the sesamoids, as do training and racing schedules.[2,68] Although uncommon, direct blunt trauma such as a kick or interference or a misstep leading to unequal tension on the bone may also contribute to sesamoid fractures. The sesamoid bones also undergo marked bone resorption when the fetlock is immobilized, and pathologic fracture after cast removal has been reported in adult horses.[53]

During training these bones rapidly remodel, which initially decreases bone porosity and increases bone trabecular width and mineralizing surface, thereby enhancing the bone's ability to withstand stress.[107] The SL also increases in strength with training until it exceeds the strength of the bone, making bone failure the method of suspensory breakdown in racing or heavily training racehorses.[17] The vascular pattern of sesamoid bones may be implicated in site selection of fractures as the orientation and distribution of vessels parallel the radiographic lucencies seen in horses with sesamoiditis and correspond to the configuration of apical fracture patterns.[94]

Clinical Signs

The medial or lateral sesamoid bones, or both, may be fractured. Lameness is very pronounced in acute stages. The horse is reluctant to bear weight on the limb and will not permit the fetlock to descend to a normal position during weight-bearing. Swelling, heat, and pain are often severe in the fetlock region. Tenosynovitis or suspensory desmitis, which also may be present, may confuse the diagnosis if radiographs are not taken. The horse evidences pain when pressure is applied to the affected bone or bones. Extension of the fetlock during weight-bearing causes pain. Observation of the gait reveals that the fetlock is held rigid so that it cannot extend as much as the opposite normal fetlock. Lameness at the walk and trot may not be obvious after 1–2 weeks' rest, but joint effusion persists.

The fracture in the bone may occur in any area of the sesamoids, but proximal fractures are more common than distal fractures; proximal fractures also are more

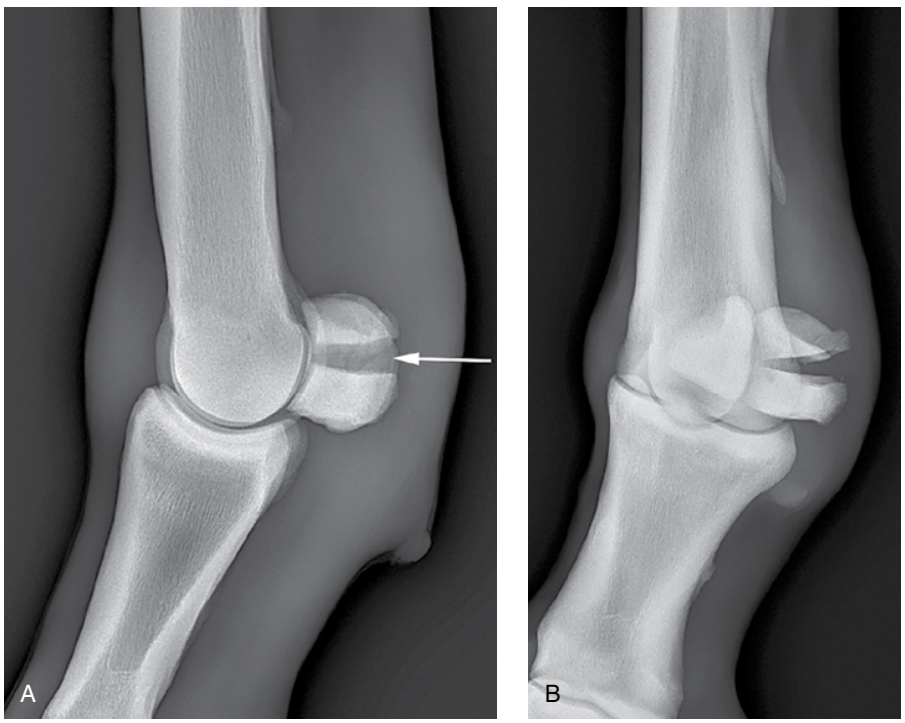


Figure 4.127. (A) Lateral and (B) oblique radiographs of a horse with a midbody sesamoid fracture (arrow).

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amenable to treatment. Desmitis of the SL and DSLs may occur concurrently with fractured sesamoids.

A history of galloping to exhaustion in an attempt to keep up with the dam is common for young foals under 2 months of age that have sustained fractures of their sesamoid bones.[32,44,74] These fractures often occur in foals that have been confined to a box stall for several days and then turned out for free exercise with the dam.

Diagnosis

Diagnosis is based on the physical examination findings described and confirmed with radiologic examination of the affected fetlock. If a fetlock joint is severely swollen and the horse shows pain when pressure is applied over the sesamoid bone(s), radiographs should be taken to rule out the possibility of fracture. Cases of tenosynovitis also should be radiographed to eliminate the possibility of fractured sesamoid bones accompanying this condition. Sesamoiditis may cause similar signs, but radiographs do not demonstrate a fracture, and joint effusion is not a prominent feature of sesamoiditis.

Standard radiographic views should include the dorsopalmar (DP), lateromedial (LM), and oblique projections. Occasionally the flexed LM may demonstrate lesions that are not detectable on the other standard projections, and estimation of reduction potential of midbody fractures can be made. The addition of the skyline projection of the abaxial surface of the sesamoid bone is helpful in some cases to identify the exact location of fractures on the abaxial surface (Figure 4.128).[66] The radiographs should be closely examined for any signs of OA associated with the fetlock and incomplete fractures that may be confused with enlarged vascular channels.

If an incomplete sesamoid bone fracture is suspected, repeat radiographs should be taken after a period of 2–4 weeks of stall rest, or nuclear scintigraphy can be performed. This permits better evaluation of the fracture line because it allows sufficient time for lysis/remodeling of the bone to occur. It should be emphasized that if these horses are allowed to exercise, they run a great risk of distracting the fracture and worsening the prognosis. Ultrasonography should be performed on all apical and abaxial fractures to identify the degree of concurrent injury to the SL.

Treatment

The treatment of sesamoid bone fractures is based on the location of the fracture and the intended use of the animal. Treatments include stall rest, cast application, surgical removal, lag screw fixation, circumferential wiring, and bone grafting.

Most fractures that are treated conservatively heal by a weak fibrous union, and the fracture line can be observed on radiographic examination for prolonged periods of time. A portion of these fractures that have apparently healed will separate at a later date and result in pain again. Generally, the conservative approach should be used for those horses that are not going to be used for performance in the future and in young foals without distraction of fracture fragments. Surgical treatment of sesamoid bone fractures in horses intended for performance is preferred for the most rapid return to performance with the least risk of future OA or sesamoid re-injury. Surgical removal or stabilization to promote earlier bony union reduces the risk of secondary OA and provides the best prognosis.

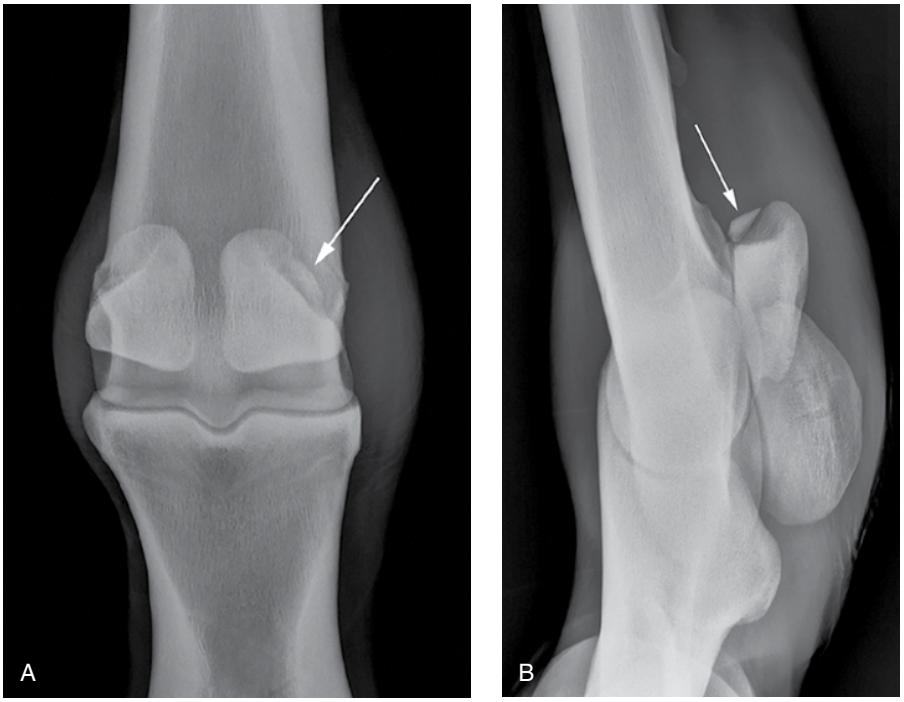


Figure 4.128. (A) DP radiograph of a horse with an abaxial sesamoid fracture (arrow). (B) Skyline projection to highlight the abaxial fragment of the sesamoid (arrow).

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Apical, articular abaxial, and basilar fractures involving less than one-third of the sesamoid bone are best treated by surgical removal of the fragment.[8,13,67,78,79,83,84,86,92] Nonarticular abaxial fractures may require removal, but horses often perform successfully without surgery. Midbody transverse fractures affecting the middle third and basilar transverse fractures of the proximal sesamoid bones have been treated successfully with lag screw fixation or circumferential wiring to provide postoperative bone compression and immobilization.[8,39,54,76,102,104,105] Lag screw fixation has been shown to have superior results in comparison with other surgical options.[18] Ideally, the fragment should be in one piece and should involve at least 30% of the bone for use of screw fixation repair. Casting is recommended after surgery because the forces to bend or cyclically fail the screw at the fracture site are high, particularly if anatomic reduction was not complete. Bone graft can be inserted into the fracture site just prior to tightening the screw. Bony union is expected within 6 months and return to training at about 9 months. The screw does not need to be removed, even if it breaks, as long as it is not infected. Approximately 44% of horses repaired with a 4.5-mm lag screw returned to race in one study.[18]

Surgery is easily performed with the horse in lateral or dorsal recumbency under general anesthesia. Surgical removal of apical sesamoid fractures smaller than one-third of the length of the bone can be performed by arthroscopy or arthrotomy through the palmar/plantar recess of the fetlock joint. Arthroscopy offers the advantage of more precise dissection and a faster return to performance for small articular fractures without suspensory involvement (3 weeks) and the ability to remove more than one fracture from a joint with minimal incisional morbidity. Lag screws are best directed from a distal to proximal direction.

For basilar fractures, the arthroscope is similarly placed in the fetlock recess, but the instrument portal must be made into the distal fetlock recess such that instruments can be placed parallel to the base of the sesamoid. Abaxial fragments that lie entirely on the basilar aspect (nonarticular) of the sesamoid bone cannot be reached by any arthroscopic approach and must be removed by incision directly over the fragment. Most of these fractures do not need to be removed and heal by fibrous union. Removal of nonarticular basilar fractures can be performed between the DSLs for smaller fragments or through the tendon sheath,[15] and using intraoperative ultrasound to help locate these fragments has been reported to be beneficial.[5]

Biaxial midbody sesamoid fractures are a common cause of breakdown in the Thoroughbred and Quarter horse racehorse.[7,46,77] Due to the fact that the suspensory apparatus is lost and distal limb vascular supply may be disrupted, management is directed toward immobilizing the fetlock joint for a sufficient time period to stabilize soft tissue damage. Treatment options include surgical arthrodesis or conservative ankylosis, although many of these horses may be humanely euthanized. A major complication to this "breakdown" injury is supporting limb laminitis in the contralateral weight-bearing limb. See the section on traumatic suspensory rupture for more information.

Fractures of the sesamoid (typically axial) that occur in conjunction with fracture(s) of the metacarpal or metatarsal condyle are serious injuries that should be identified with radiography because the prognosis for returning to racing, even with repair of the condylar fracture, is poor.[6] These fractures are usually sagittal and axial and occur due to extreme pulling on the intersesamoidean ligament when the condylar fracture displaces. These fractures suggest significant soft tissue injury to the fetlock joint and that OA is likely to ensue regardless of treatment (Figure 4.129).

Prognosis

The reported prognosis for apical sesamoid fractures is good to excellent (88% of Standardbreds[104] and 77% of Thoroughbreds[78] return to racing), for abaxial fractures is good (71% of Thoroughbreds or Quarter horse racehorses returned to racing),[86] for basilar fractures is fair (50%–60% of Thoroughbreds return to racing),[83] and for midbody fractures repaired by either lag screw fixation or circumferential wiring is fair (44%–60% return to performance).[39,54] However, repair of midbody fractures using lag screws is thought to provide a more favorable prognosis.[18] In addition, a recent study indicated that the size of apical fractures does not appear to influence the prognosis in racehorses.[47] Reports of conservative treatment options are not available for comparison, but generally it is presumed that the prognosis is guarded to poor for either basilar or midbody



Figure 4.129. Axial sesamoid fracture (arrows) in a horse with a lateral condylar fracture. *Source:* Courtesy of Dr. Wes Sutter.

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fractures that are not treated surgically. Most of these joints develop significant OA and restricted range of joint motion. If both sesamoids are fractured, the prognosis is poor due to the loss of suspensory support and only should be considered for salvage of valuable breeding stock or in horses of great sentimental value.

SESAMOIDITIS

Sesamoiditis is observed frequently in racing horses and hunters and jumpers between 2 and 5 years of age.[60–62,89] The condition is characterized by pain associated with the proximal sesamoid bones and insertions of the SL that result in lameness. The pain is thought to result from inflammation at the interface of the SL with the proximal sesamoid bones.[62,89] Concurrent SL branch disease should be suspected in these horses. A significant relationship between the presence of sesamoiditis and the subsequent development of SL injuries has been documented.[71] Evidence of sesamoiditis suggests a 5 times greater risk for developing injuries to the SL branch with the onset of training.[58] Pain, heat, and inflammation can usually be clinically detected at the insertion of the SL during the active stages of the disease process, but marked lameness and limitations on performance can also occur without any clinically detectable signs.[22,37]

Radiographs can reveal a range of changes from accelerated early remodeling response in the bones (increased size and number of vascular canals) to marked proliferation of bone along the abaxial margin of the sesamoid and increased bone density of the sesamoid. The SL and the DSLs may also be affected and show calcified areas. The increased bone production is thought to result from inflammation from tearing of the attachments of either the SL or the DSLs. Young Thoroughbreds with more severe radiographic signs of sesamoiditis are thought to be at increased risk to develop future suspensory branch injuries.[58] However, it is also possible to see radiographic changes indicating chronic sesamoiditis in sound performing horses, suggesting that some horses heal the injury and regain suspensory strength.[37]

Etiology

Any unusual strain to the fetlock region may produce sesamoiditis. Most commonly in racehorses, hunters, and jumpers, it can affect any type of horse. It is caused by increased stress to the attachment of the SL to the sesamoid bones. Injury to the DSLs may also occur at their attachment to the basilar portion of the sesamoid bones.

The sesamoid bones have a substantial intraosseous blood supply that enters the midportion of the bone through multiple abaxial channels that correspond to the channels that enlarge in sesamoiditis, indicating bone remodeling. This may represent the initiation of the remodeling response to bone stress of training, or it may reflect an increase in blood flow due to inflammation and injury to the SL, or both.[94] The sesamoid bones undergo intense remodeling in response to training,[107] and the progression of radiographic changes correlates to bone response to remodeling and injury.

Interestingly, several studies have categorized the radiographic changes in sesamoid bones of young (2- and 3-year-old) racehorses and discovered that many horses have increased size and number of vascular channels without lameness.[37,38] Yearling Thoroughbreds with more than two irregular vascular channels had a decrease in number of race starts and earnings at 2 and 3 years of age.[85] This supports the idea that remodeling is a normal response to training and that only if the stresses exceed, the bones' capability to strengthen would microfracture and bone damage occur. Although radiographic vascular changes of bone remodeling were not associated with sesamoid fracture, the vascular structures course along known lines of fracture in adult racehorses. The sesamoid bones have an extensive sensory nerve supply that may explain bone pain associated with trabecular bone injury.[26,27] Concurrent suspensory branch injury may also contribute to the palpable pain and lameness in affected horses.[34,58,71]

Clinical Signs

Symptoms of this condition are similar to those caused by fracture of the sesamoid bone. In the early stage, minimal swelling is observed, but increased heat may be felt over the abaxial surface of the sesamoid bone(s). As the disease progresses, visible enlargement of the soft tissues overlying the palmar/plantar surface of the fetlock can be seen as fibrosis of/around the injured SL becomes apparent. On palpation, pain withdrawal can usually be elicited by placing pressure over the abaxial surface of the sesamoid bones. In more advanced cases, pain may be elicited by applying pressure over the branches of the SL. The horse will also likely be positive to distal limb flexion.

At exercise, the lameness varies considerably and depends on the acuteness of the injury and its degree. In general, the lameness is most evident during the first part of exercise and is more exaggerated when the horse is exercised on hard surfaces. Perineural and/or intrasynovial anesthesia is used infrequently to diagnose this condition. Horses respond to a low four-point nerve block, but not to an intra-articular fetlock block, locating the lameness to the periarticular structures.

Diagnosis

The radiologic changes of true sesamoiditis have been described as bony changes on the abaxial surface or basilar region, increased number and irregularity of the vascular channels, and increased coarseness and mottling of the bone trabeculation (Figure 4.130). In acute cases, radiographs may have to be taken approximately 3 weeks after onset of the condition to determine whether bony changes will occur on the sesamoid bones. The condition may also occur with tenosynovitis, fracture of the sesamoid bones, and injury to the SL from which it must be differentiated. Careful radiographic interpretations of the mottled trabecular pattern seen in the proximal sesamoid bone are necessary to differentiate a fracture. Incomplete fracture can be differentiated from these coarse vascular canals because the fracture usually extends to the abaxial surface and the vascular canals do not. In addition, fracture lines frequently run at different angles than the vascular channels.

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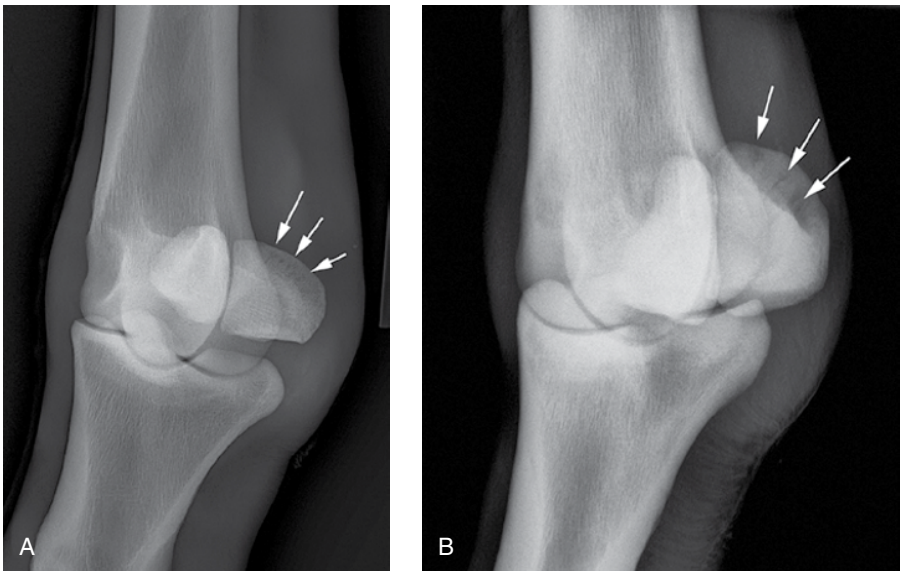


Figure 4.130. Radiographs of the fetlock in two different horses demonstrating mild sesamoiditis (A) and moderate sesamoiditis (B) with increased number of the vascular channels in the lateral proximal sesamoid bone (arrows).

Nuclear scintigraphy typically indicates increased radiopharmaceutical uptake (IRU) in the region of the sesamoid bones, but the uptake is usually less than what would occur with a fracture. Ultrasound of the SL and DSLs may be useful, but many horses may have no abnormalities in the ligaments in the acute phase. However, subclinical SL branch change has been reported in Thoroughbred yearlings with evidence of sesamoiditis suggesting that ultrasound examination should be performed in all affected horses.[71]

Treatment

If heat, pain, and swelling are detected at the bone or suspensory insertion, then efforts should be made to reduce the inflammation. Both topical and systemic anti-inflammatory medications may be used. Rest from performance until soundness at the trot is achieved followed by slow increasing duration and intensity of exercise allows the bone to continue to remodel and strengthen. Importantly, the exercise must be kept below the level that would reinjure the bone. Similar to other SL injuries, convalescence is long (6–8 months), and injury often recurs when horses return to full work. In chronic stages, blistering and ESWT have been used but with equivocal success. Young Thoroughbred horses with suspensory branch injuries and concurrent sesamoid inflammation treated with PRP at the junction between the bone and the ligament were more likely to start at least one race during their 2-year-old racing year than horses treated with saline; but this benefit did not extend into their 3- and 4-year-old racing years.[34]

Prognosis

The prognosis for return to full athletic performance free of lameness is guarded to unfavorable, depending upon the amount of periosteal reaction and new bone growth that occurs on the sesamoid bones and the extent of concurrent injury to the SL and the DSLs. In yearling racehorses, enlarged vascular canals identified on radiographs have been associated with fewer race starts and reduced earnings compared with horses with normal vascular canals.[85] Young racehorses with abnormal sesamoid bones should also be monitored closely for future adjacent SL branch injuries that may occur.[34,71]

AXIAL OSTEITIS/OSTEOMYELITIS OF THE PROXIMAL SESAMOID BONES

Etiology

Osteitis/osteomyelitis of the axial border of the proximal sesamoid bones is an uncommon cause of lameness in horses. The cause remains unknown and speculative with vascular, infectious, and traumatic etiologies implicated.[51] The condition is specifically associated with the intersesamoidean ligament within the apical to midbody axial margins of the sesamoid.[51] The condition has occurred in horses with septic tenosynovitis of the digital sheath, septic bacterial arthritis of the fetlock, and secondary to an *Aspergillus* fungal fetlock infection.[80,103] In another report, the clinical signs and radiographic lesions were suggestive of sepsis, although histology of lesions revealed infarction and necrosis as well as osteoporosis and chronic inflammation in the intersesamoidean ligament.[16]

Clinical Signs and Diagnosis

Horses typically present with a consistent lameness that may be severe (often lame at the walk). In one report, lameness ranged from 2 to 5 out of 5 (mean 4 out of 5).[103] In another report of 12 Friesian horses, all lesions were in the hindlimb, and lameness was acute and severe.[16] Radiographs usually reveal bone lysis at the attachment of the intersesamoidean ligament primarily at the midbody and apical regions with varying degrees of joint effusion (Figure 4.131A).[51] A single or

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both sesamoids may be affected. Some lesions appear cystic, whereas others appear to erode the axial border more diffusely.

Treatment and Prognosis

Ultrasonography along with advanced imaging (CT and/or MRI) of the fetlock can assist with determining the surface location of the lytic areas and assist with the decision for surgery (Figure 4.131B).[51] Small lesions that are internal or only open toward the intersesamoidean ligament may be best treated conservatively. Most lytic lesions have surgical access to the area from the palmar/plantar fetlock joint and/or digital sheath using arthroscopy. Abnormal bone and ligament should be debrided if present, and samples are submitted for culture and sensitivity. Debridement can make horses more lame due to the disruption of the thick, fibrous intersesamoidean ligament. Cast or splint application may help improve the horses' comfort level while healing. Regional limb perfusion with an antimicrobial offers the advantage of perfusion of multiple affected sites including the fetlock joint, tendon sheath, and sesamoid bones.[80] Prolonged administration of antimicrobials should be based on accurate culture and sensitivity results.

The prognosis for return to performance is considered guarded to poor, although horses may become pasture sound or return to less strenuous activities.[51,103]

OSTEOARTHRITIS OF THE METACARPOPHALANGEAL/METATARSOPHALANGEAL JOINT

Traumatic arthritis includes a diverse collection of pathologic and clinical states that develop after single or repetitive episodes of trauma and may include synovitis (inflammation of the synovial membrane), capsulitis (inflammation of the fibrous joint capsule), cartilage injury/damage, subchondral bone injury, or intra-articular fractures.[9–11,57] Any type of traumatic joint injury can progress to OA within the fetlock.

Etiology

Soft tissue injury to the joint commonly occurs in horses in full work and represents an overuse of the joint or a single-event injury. Joint injury can also occur as a sporadic event due to trauma. In young horses just put into training, fetlock joint soreness and joint effusion can develop and can usually be managed with a modified exercise plan and medication. Abnormal limb conformation may also predispose horses to joint soreness and eventually OA of the fetlock. Bone fatigue and subchondral damage are also known to occur in racing Thoroughbreds and predispose to the development of OA.[3,4] See section on palmar/plantar osteochondral disease (POD) later in this chapter.

Clinical Signs

In mild cases, joint inflammation occurs without lameness and is noted as joint soreness on flexion and joint effusion in a young, recently worked or maximally performing horse. Swelling of the palmar/plantar joint pouches is usually noted first, followed by dorsal pouch effusion with more chronic effusion. Cases that involve a capsulitis of the fetlock, particularly the dorsal fetlock, often have palpable heat and a more severe response to flexion. If exercise is sustained, lameness may ensue and is frequently bilateral. In moderate cases, joint soreness and effusion persist and lameness worsens with exercise. In severe cases of injury or advanced joint degeneration, lameness can be severe, and obvious joint enlargement and decreased range of motion can ensue.

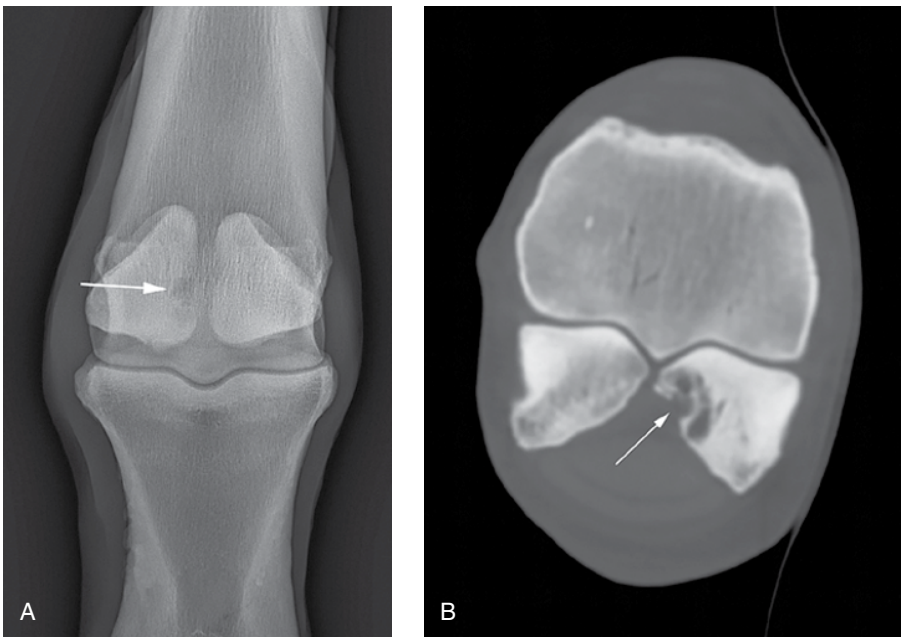


Figure 4.131. DP radiograph (A) and transverse CT image (B) of two different horses demonstrating focal lysis in the axial aspect of the lateral proximal sesamoid (arrows).

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Diagnosis

Clinical examination provides a high degree of suspicion that OA of the fetlock is the cause of lameness. Both radiographs and nerve blocks or a fetlock joint block can help localize and define the fetlock OA. Radiographs can help diagnose an underlying cause in many cases, such as intra-articular osteochondral fragments (either as part of osteochondrosis or as an intra-articular fracture), subchondral cystic lesions (SCLs), subchondral bone erosion, osteophytosis, or joint space narrowing (Figure 4.132). Typically, digital nerve blocks do not block fetlock OA, but a low four-point block significantly improves the lameness.[9–11] However, there have been reports of horses with marked cartilage injury in the fetlock joint improving with a palmar digital nerve block.[25] Direct intra-articular anesthesia of the fetlock offers additional diagnostic benefits of achieving complete resolution of lameness, access to a joint fluid sample for analysis, and a direct portal for simultaneous treatment. If the joint fluid appears hemorrhagic or cloudy, an immediate synovial fluid analysis and culture should be performed to rule out a septic process.

In the past, horses that responded to a direct block to the joint but did not respond to rest or medical therapy were candidates for arthroscopic surgery. Now, the use of advanced imaging (CT or MRI) provides a more accurate identification of the pathology and determination of whether a horse is a candidate for arthroscopic surgery or medical therapy (Figure 4.133). This is especially true for horses with subchondral bone and articular cartilage damage. Both CT and MRI are considered to be superior to digital radiography to detect subchondral bone pathology in the metacarpophalangeal joint.[63,64]



Figure 4.133. Sagittal proton density MR image depicting marked cartilage damage of the distal third metacarpal bone (arrows) along with marked sclerosis of the adjacent subchondral and medullary bone (arrowheads).

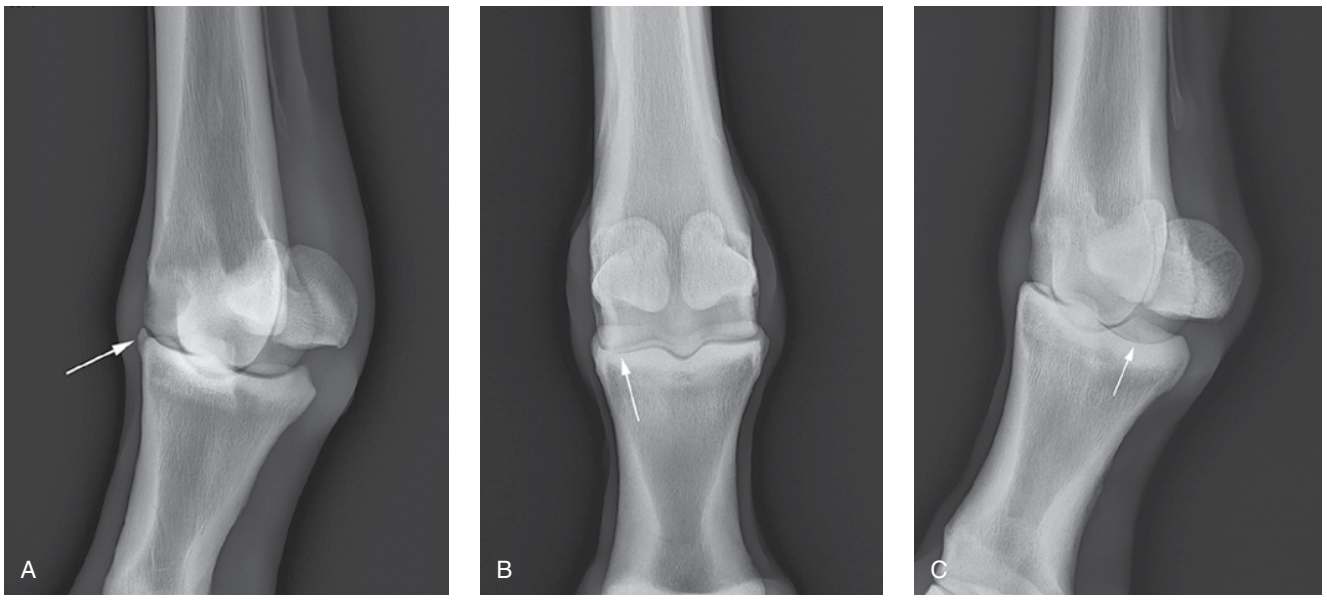


Figure 4.132. DLPMO radiograph of the fetlock (A) of a horse with a large periarticular osteophyte on the dorsomedial aspect of proximal P1 (arrow). DP (B) and oblique (C) radiographs of another horse with severe narrowing of the medial aspect of the fetlock joint (arrow) due to marked cartilage loss. The joint narrowing can also be seen on the oblique projection (C; arrow). Note that there are minimal other radiographic signs of OA in this horse other than subchondral bone sclerosis in proximal P1 (B and C).

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Treatment

Management of fetlock OA should address any primary problem and potentially include the following: rest, physical therapy, bandages, shoeing changes, and systemic and intra-articular joint medications. Arthroscopic surgery is recommended in horses that have concurrent predisposing conditions such as osteochondrosis or osteochondral damage/fragmentation. Performance horses that are lame from fetlock joint soreness should be rested until lameness resolves, frequently a minimum of 30 days. Return to work should be gradual and can be supplemented with systemic joint medications and shoeing alterations to promote breakover of the foot and an easy landing (rim pads, remove caulks and toe grabs, etc.). Use of athletic bandages to provide support to the fetlock joint during work can be very helpful and pressure bandages after workouts prevent swelling. Many jumping and racehorses are routinely treated with icing and wrapping of the fetlocks after workouts. If the fetlock effusion becomes chronic in nature with no active bone injury, many of these horses benefit from a low level of controlled exercise.

Joint medications can be effective for horses with OA of the fetlock. These include systemic nonsteroidal medications to reduce pain and inflammation, systemic polysulfated glycosaminoglycans (PSGAGs) to manage cartilage health, systemic hyaluronan to reduce synovitis and promote cartilage health, and intra-articular use of these and similar products and/or steroids for maximal relief of pain and inflammation.[9–11] Biologic therapies, such as autologous conditioned serum and stem cells, have also been promoted for intra-articular treatment of OA. These medications are frequently used after surgery or in OA without a surgical indication. Many show horses, particularly jumping and racehorses, have chronic soreness and early degenerative changes in the fetlock joints. Many of these horses are maintained on intramuscular injection of PSGAGs and intravenous (IV) hyaluronan throughout the show or race season.

The importance of regular and regulated exercise is critical to managing horses with fetlock OA. Confining horses with primary OA often worsens their stiffness and discomfort. Regular exercise and turnout provides the greatest longevity with this condition.

Prognosis

Early recognition and management of traumatic OA of the fetlock are key to keeping the joint in good health and continuing training in racehorses. Once degenerative changes in the joint are visible on radiographs, the prognosis is still fair with management if the horse is sound enough to perform with treatment. Many horses are maintained in full work while their fetlock OA is being managed. Once horses are no longer sound with the medical management listed, an extended period of rest may permit them to return to training, usually at a reduced expectation. Many top equine athletes can continue to perform at some level of activity with fetlock OA.

PALMAR/PLANTAR OSTEOCHONDRAL DISEASE

The palmar/plantar MC/MT condyle of the racehorse is thought to receive greater than twice the amount of stress compared with the dorsal surface of the bone at racing speeds. These excessive forces make this area vulnerable to bone necrosis and cartilage injury.[75] With these lesions, the subchondral bone of the palmar condyle is unable to carry out one of its important functions to support the overlying cartilage, which has been implicated as a major component in the development of OA (Figures 7.22–7.24).[72] A well-documented sequela of these repetitive high impact injuries is palmar/plantar osteochondral disease (POD), particularly in Thoroughbred racehorses. POD is thought to be due to repetitive overloading and impact of the distal MC/MT III bones, resulting in performance-limiting lameness with progression to permanent OA in severe cases.

Etiology

Repetitive overloading to the palmar/plantar aspect of the third MC/MT condyles at training and racing speeds contributes to focal areas of fatigue to the subchondral bone at the articulation of the palmar/plantar MC/MT III condyles with the proximal sesamoid bones. As a result, the bone remodels in an attempt to enhance its ability to handle the stress and load.[10,41] With continued stress, microtrauma occurs due to the decreased compliance of the sclerotic bone, resulting in edema and hemorrhage in these regions usually accompanied with microfractures.[3,50,52] The subchondral bone damage is thought to cause lameness and loss of training days primarily in Thoroughbred racehorses. Periods of rest are useful to allow for bone healing between months of training but have also been shown to result in subchondral bone resorption and eventual collapse of the overlying articular surface adjacent to the subchondral bone lesions with eventual articular cartilage degeneration.[3] The pathology is reportedly worse in the medial condyle of the forelimbs and lateral condyle in the hindlimbs.[70] Parasagittal microcracks and condylar fractures have a similar etiology to POD and therefore have been reported to occur simultaneously within the same limb, but are thought of as separate clinical entities.[31,45] Severe cases of POD often result in career-ending lameness and significant OA.

Clinical Signs

The primary clinical sign of POD is performance-limiting lameness localized to the fetlock region in racehorses. Metacarpophalangeal/metatarsophalangeal joint effusion is an uncommon finding with POD. The most common baseline lameness grade is 2/5 with approximately 60% of cases of diagnosed POD being positive to flexion of the fetlock joint.[95] Perineural anesthesia of the palmar/plantar metacarpal nerves is more useful in these cases as intra-articular anesthesia yields inconsistent results due to the pathology originating within the subchondral bone.[95] The lameness can present as bilateral or as an abnormal action in the limbs suggestive of quadrilateral disease.[4]

Diagnosis

Diagnosis of POD can be difficult with radiographs alone. However, due to the cost of nuclear scintigraphy and MRI, Davis et al. in 2017 described the common

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radiographic features of POD as focal radiopacities and/or radiolucencies in the palmar/plantar MC/MT III condyles along with flattening of the condyle and osteophytosis (Figure 4.134).[29] They reported a significant improvement in sensitivity and specificity of diagnosis of POD with plain radiographs following education of reviewers on the common radiographic features of POD. The authors concluded that radiography is useful in later stages of disease but other imaging modalities are required for early diagnosis of POD.[29] Nuclear scintigraphy has been reported as a useful diagnostic tool for POD. In 2011, Trope et al. demonstrated that increased IRU of the palmar/plantar MC/MT III condyles was the most common finding in the fetlock of Thoroughbred racehorses and was associated with fewer starts and less money earned than controls.[93] The patterns of IRU with nuclear scintigraphy are consistent with POD lesions diagnosed on postmortem examination; however, there has been no correlation with the degree of uptake and prognosis for return to racing.[4] The use of MRI has allowed for improved understanding of the relationship between pathology and prognosis in cases of POD. In 2012, Powell described the appearance of POD lesions with standing low-field MRI as regions of focal hyperintensity along the margin of the joint surrounded by hypointense regions on STIR/T2* sequences. Abnormalities were not detected radiographically in these horses.[73]

On *postmortem* evaluation, lesions can range grossly from discoloration of the subchondral bone with minimal to no cartilage erosion to severe subchondral bone discoloration and associated cartilage damage (Figure 7.47).[4] Histologically, common findings include microfractures of the trabecular bone with thickening and sclerosis of the adjacent bone, areas of osteocyte necrosis and eventual collapse, and degeneration of the overlying articular cartilage.[3]

Treatment

Treatment options are limited and range from rest, which allows for bone remodeling in the early stages of the POD, to systemic and intra-articular anti-inflammatory therapies. A period of 60- to 120-day small paddock turnout to allow for appropriate subchondral bone remodeling was recommended by Tull et al., with 95% of horses diagnosed with POD returning to racing.[95] If progression of the disease continues, pain control is difficult and typically unrewarding in the majority of cases.

Prognosis

Lameness associated with the early stages of POD can resolve with appropriate rest from training allowing the bone time to remodel and heal. As lesions progress and cartilage damage occurs, progression to severe OA occurs, resulting in career-ending lameness.

FETLOCK SUBCHONDRAL CYSTIC LESIONS (SCLS)

Fetlock subchondral bone cysts (SCLs) occur most commonly on the weight-bearing surface of the MC/MT condyle and less commonly on the weight-bearing surface of proximal P1. Cystic lesions of the distal metacarpus/tarsus that open into the fetlock joint can occur in young horses and are possibly considered part of the developmental osteochondrosis syndrome.[12]

Etiology

Some SCLs in the fetlock and other articular locations are considered part of the developmental orthopedic disease complex that occur during growth and the conversion of cartilage to bone. However, some are thought to

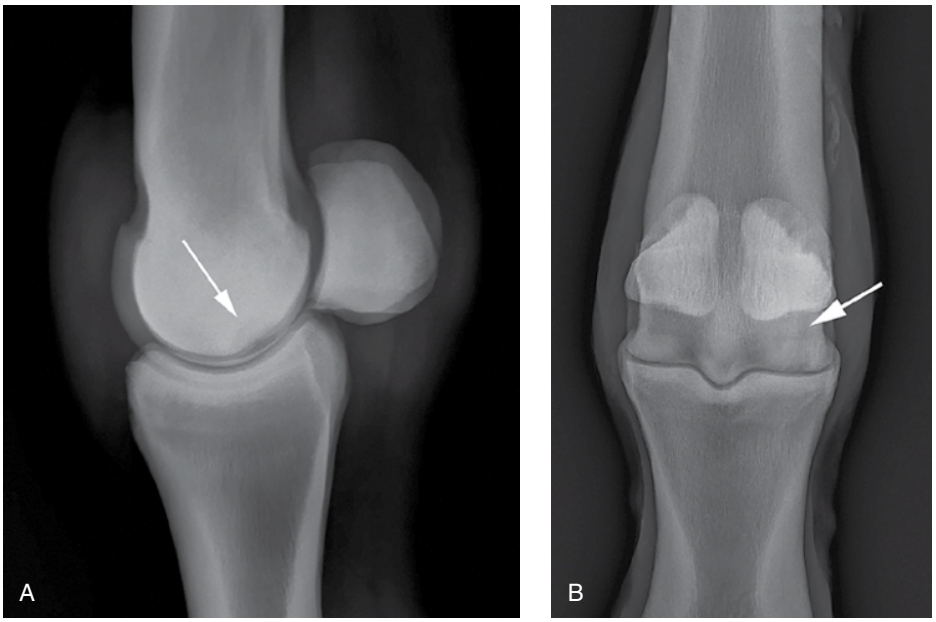


Figure 4.134. Flexed lateral (A) and dorsopalmar (B) radiographs of two different fetlock joints demonstrating radiolucent defects and severe sclerosis of the palmar condyles of the distal third metacarpal bone (arrows).

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be associated with subchondral bone trauma and are more common in adult horses. Injury to the articular cartilage and underlying subchondral bone from high impact may allow synovial fluid to enter the fissure and increase intraosseous pressure, contributing to cyst development. Typically, the lameness and joint inflammation in these horses improve as the cyst matures.

Clinical Signs

The average age of horses with clinical signs in one study was 18 months, although the SCL may develop prior to the onset of clinical signs.[42] Proximal P1 or distal MC/MT SCLs can develop in horses of any age from trauma. Moderate lameness is often present together with pain on fetlock flexion in most cases and fetlock joint effusion in approximately 50% of the cases. However, the degree of lameness can vary considerably depending on the location and age of the SCL.

Diagnosis

The diagnosis is usually made by radiographic evaluation of the joint (Figure 4.135A). In the early development of the SCL, it may not be apparent on radiographs, and follow-up radiographic examination is recommended if clinical signs that refer to the joint persist. Nuclear scintigraphy can identify a focal area of increased uptake in the bone as the SCL is developing and for several months after it has formed. Chronic SCLs may not have significant bone turnover above the surrounding condyle and may not be detectable with scintigraphy. Advanced imaging (CT or MRI) is often helpful to document the extent of the SCL and any secondary articular pathology that may be present in horses with trauma-induced lesions.

Treatment

Surgical debridement of metacarpal SCLs by an arthroscopic approach has historically been the preferable treatment if the diagnosis is made before significant signs of OA have developed (Figure 4.135B).[42] Medical therapy with newer regenerative therapies or intra-articular PSGAGs may also be helpful depending on the location and characteristics of the SCLs. For SCLs that do not respond to conservative treatment or debridement, osteostixis by extra-articular approach or mosaic autologous osteochondral grafting can be performed.[12]

Prognosis

The prognosis for return to performance appears to be good for most horses with SCLs of the distal metacarpus/tarsus. In one report, surgical treatment of third metacarpal SCLs resulted in 80% of horses (12 of 15) returning to their intended use.[42] Follow-up radiographs to determine the degree of OA/degeneration of the joint are helpful in determining the long-term prognosis. Horses with trauma-induced SCLs typically have a more guarded prognosis for performance than those with developmental lesions regardless of their location.

TRAUMATIC RUPTURE OF THE SUSPENSORY APPARATUS

Traumatic rupture of the suspensory apparatus with or without fractures of both proximal sesamoid bones is a common cause of acute breakdown in the racing Thoroughbred and often results in humane destruction of the animal.[14,40,46,77] Proximal luxations of the sesamoid bone without fracture can also occur with trau-

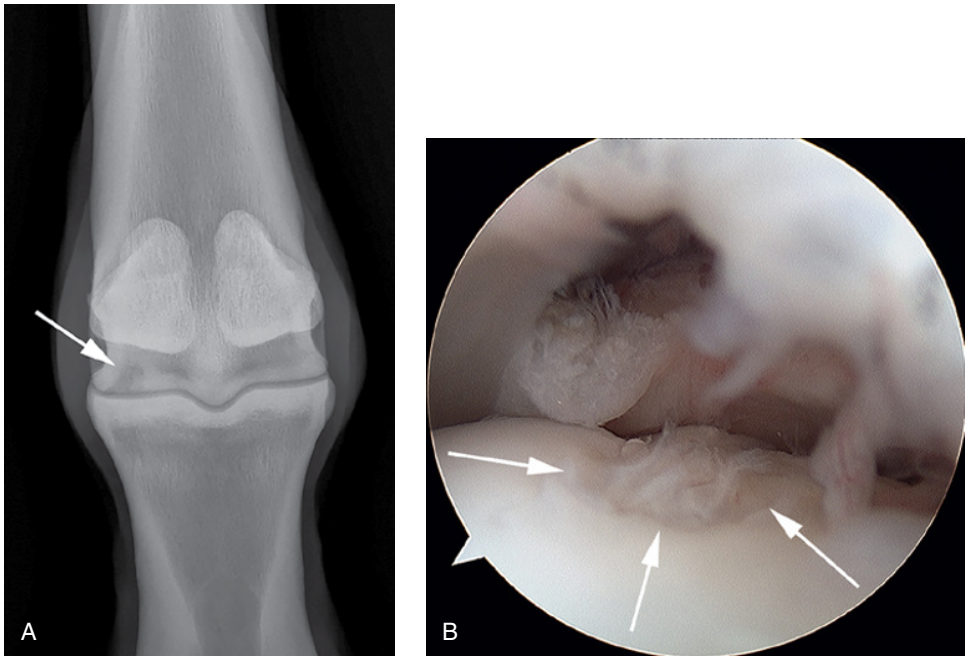


Figure 4.135. (A) DP radiograph of a horse with a subchondral cystic lesion in the medial aspect of the distal third metacarpal bone (arrow). (B) Arthroscopic image of a subchondral cystic lesion on the distal condyle of the third metacarpal bone. Note the well-defined margins of the cyst (arrows).

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matic rupture of the DSLs. Transverse or comminuted fractures of both proximal sesamoid bones allow the apical portions to be drawn proximally by the pull of the SL while the basilar fragments remain attached to the DSLs. Occasionally, open luxation of the fetlock joint can occur. Besides the severe trauma sustained by the supporting soft tissues and bone, the adjacent digital arteries are frequently damaged sufficiently to result in ischemic necrosis of the hoof. Treatment of traumatic rupture of suspensory apparatus should only be considered as a salvage procedure.

Etiology

Extreme overextension of the fetlock is the likely cause for disruption of the suspensory apparatus. Preexisting pathology of the bones or SL need not be present for this catastrophic failure to occur, but the presence of an abnormal finding in the SL on prerace inspection by a regulatory veterinarian increased the risk of SL injury 3.4-fold in future races as compared with a control population.[23] In a recent postmortem study of Thoroughbred racehorses, moderate lesions identified in the SL branches or DSLs were more likely to occur in horses that died from suspensory apparatus failure or metacarpal condylar fractures than in horses that died from nonmusculoskeletal causes.[40] Factors that increase the strain on the flexor surface of the limb would be expected to increase the risk of suspensory apparatus failure. Thoroughbred horses racing with low toe grabs or full toe grabs were 6.5 and 15.6 times more likely to incur a fatal suspensory apparatus injury than horses without toe grabs.[48] A study of Thoroughbred horses in New Zealand suggested that injuries to the suspensory apparatus were more likely in males, older horses, and horses in training without any starts, and there was no association between injury and cumulative high-speed exercise.[69]

Clinical Signs

On gross observation the affected fetlock is usually very swollen, and the horse usually bears its entire weight on the unaffected limb. The lameness is obvious, and if the animal transfers its weight to the affected limb, the fetlock will sink to the ground. Palpation often reveals the proximal displacement of either the intact sesamoid bone or the apical fractured fragments. Immediate stabilization of the limb is critical to prevent rupture of the neurovascular bundles. The blood supply to the distal limb can be estimated by palpation of a pulse, presence of distal bleeding and/or use of Doppler devices, or the IV injection of 5 g of sodium fluorescein.[90] The vascular supply is best evaluated after the horse has been treated for shock and the limb stabilized. Intense vasoconstriction initially can be interpreted as lack of blood supply.

Diagnosis

Radiographic examination usually reveals either the proximal displacement of the intact sesamoid bone or proximal displacement of the apical portions of the fractured sesamoid bones (Figure 4.136A). Associated swelling of the soft tissues is also quite evident, and preexisting degenerative lesions within the sesamoid bones and fetlock joint may also be present.[100]

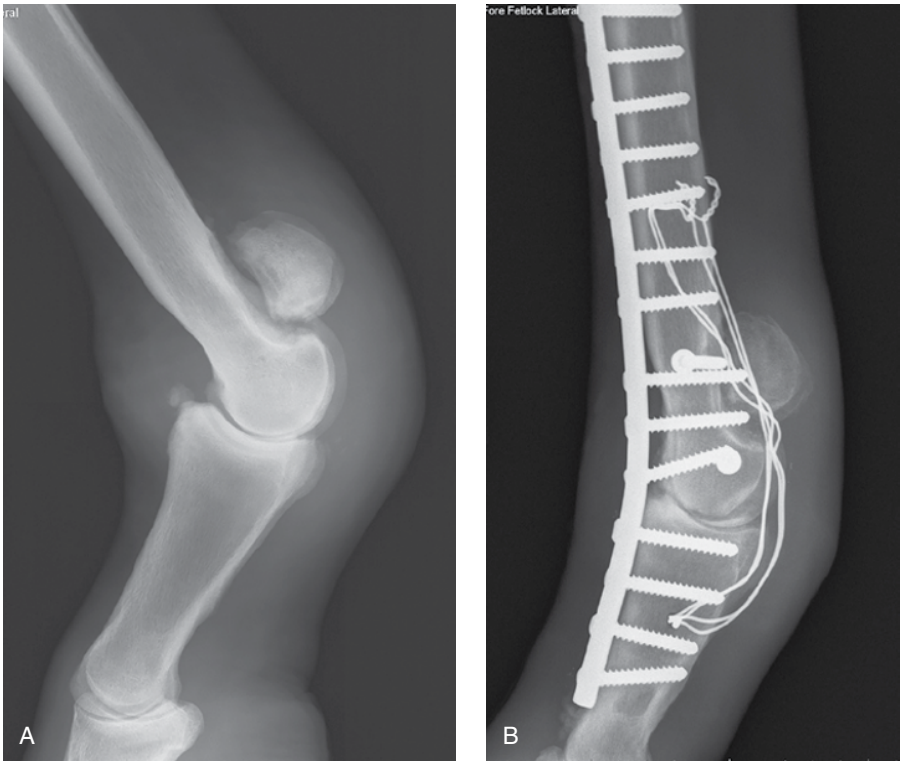


Figure 4.136. (A) Rupture of the distal sesamoidean ligaments causing proximal displacement of the proximal sesamoid bones. (B) Postoperative appearance of the same horse in (A) after fetlock arthrodesis.

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Treatment

Treatment should be considered for horses that are to be used for breeding or when there is sentimental value. Management of the horse on the racetrack is critical for success in treatment. Emergency crews must immediately restrain the horse and apply secure splints, such as a cast or the Kimzey Leg Saver® to permit transport of the horse to a facility for radiographs. Immediate immobilization of the affected limb is required to decrease the chances of further injury to the soft tissue as well as the vascular supply. Maintenance of the splint for a 4- to 5-day period prior to the selection of the final treatment permits the horse to acclimate to the immobilization and recover from the trauma and allows definition of the extent of skin necrosis and loss of vascular supply accompanying the injury. The fetlock arthrodesis procedure does not need to be performed immediately, and the soft tissue injury and permanent deficit and risk of infection must be evaluated to properly predict the outcome with surgery.

Treatments include casting, splinting, and fetlock arthrodesis (Figure 4.136B).[14,21,100] Casting and splinting are aimed at supporting and immobilizing the fetlock until soft tissues have healed sufficiently to support body weight. The use of a fetlock sling shoe can work in partial breakdown injuries. The Kimzey Leg Saver splint is not as rigid as a cast, but allows cleaning and treatment of soft tissue injuries and is designed to assist with fetlock joint support in suspensory apparatus injuries. Arthrodesis with implants and bone graft can be used to achieve a pain-free stable fusion of the fetlock joint if the soft tissues are intact and risk of infection is minimal. Supporting limb laminitis of the unaffected foot is a common sequela to this injury; therefore, the contralateral foot should be supported to distribute loading along the sole.

Casting the limb in flexion can immobilize the fetlock until healing has occurred, but may be complicated with severe pressure sores over the sesamoids that may result in septic osteomyelitis requiring euthanasia.[100] Casting is also expensive and labor intensive due to the multiple cast changes required and the prolonged duration for fusion. After 8 weeks in a cast, the limb is supported in a bulky bandage, and a special shoe is applied to elevate the heel. Other methods of supporting the limb with weight-bearing on the toe (splints) and with sling support shoes can be successful, but continual monitoring and aftercare is required for an extended duration. Of 25 cases managed conservatively, 15 survived.[82] Complications were similar to those from surgical techniques and included pressure sores, osteomyelitis, avascular necrosis, and supporting limb laminitis.[82]

Surgical arthrodesis of the fetlock should be considered in acute cases with intact skin that have not developed wounds during the initial management of the soft tissues. It is also recommended in chronic cases that fail to ankylose or in those horses that have chronic joint pain. The most popular method to achieve fetlock arthrodesis is use of a single 14-hole broad plate secured with 5.5- and 4.5-mm cortical screws spanning the dorsal surface of the metacarpus, fetlock joint, and proximal phalanx.[14,21] If the suspensory apparatus is disrupted, the palmar tension band must be reestablished or the plate will bend when loaded. The palmar tension band can be reestablished by securing dorsal 4.5-mm cortical screws into the sesamoid bones from adjacent to the plate for avulsions proximal to the sesamoid bones or of the apex of the sesamoid bones. For injuries without intact sesamoid bones or with disruption of the DSLs, a tension band wire must be placed around the palmar surface of the joint.[14]

Prognosis

The prognosis appears to be good for pasture and breeding soundness with preselection of cases. With arthrodesis, 32 of 54 horses with arthrodesis of the fetlock survived and were eventually allowed unrestricted activity.[14] The prognosis is better for horses in which fetlock arthrodesis was elected as the primary treatment rather than as a last resort and was better for horses in which fusion was elected for OA rather than rupture of the suspensory apparatus. In one report, 4 of 6 Thoroughbreds with breakdown injuries treated with fetlock arthrodesis with the locking compression plate survived.[21]

LUXATION OF THE METACARPOPHALANGEAL/METATARSOPHALANGEAL JOINT (FETLOCK LUXATION)

Lateral and medial luxation of the fetlock joint occurs uncommonly, but is a recognized syndrome that can affect all ages and breeds of horses. Usually, either the lateral or medial collateral ligament is ruptured, creating an obvious varus or valgus deformity of the fetlock region.[88,109] Occasionally, avulsion fractures associated with the insertion of these ligaments or joint capsule may occur and be noted on the radiograph proximal to the joint space. Articular fractures of the palmar/plantar eminence may also accompany the luxation.[43] Both forelimbs and hindlimbs can be affected, and the joint was open in half of 10 reported cases.[109] The diagnosis is usually quite obvious because an angular deviation of the fetlock joint is present. Occasionally the luxation will reduce spontaneously, and only a lateral or medial swelling will be noticed. Immediately after the injury, if the luxation is reduced, some horses will be minimally lame and appear sound at the walk. Re-luxation can occur if the joint is not adequately stabilized. Physical manipulation of the fetlock in these cases clarifies the suspicion of luxation. Typically, the joint re-luxates when it is flexed and abducted away from the side of injury.

Etiology

This injury frequently occurs when the horse steps in a hole or gets a foot caught between two immovable objects. The luxation results while the horse attempts to break free. Owners frequently relate the history of finding the horse caught in this situation. Occasionally horses spontaneously luxate their fetlock during high-speed activities (e.g. racing, rodeo eventing) or after running into an object.[88]

Clinical Signs

The clinical signs are usually obvious and are helpful in differentiating this injury from a fracture. However,

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fractures of the proximal phalanx can accompany the luxation. A varus (outward deviation of the cannon bone and inward deviation of the digit) or valgus (inward deviation of the cannon bone, outward deviation of the digit) angular deformity is usually present (Figure 4.137). Occasionally, the luxation will reduce spontaneously, and the remaining evidence of its occurrence will be lameness, joint instability, and asymmetrical fetlock swelling over the torn collateral ligament.

On palpation, the fetlock can usually be reduced and re-luxated without the degree of pain or evidence of crepitation associated with fracture. More frequently, the swelling that is present is less than that observed with fracture, and the swelling is located selectively over the lateral or medial surface. Although the digital vascular supply is rarely compromised, it should be carefully evaluated, particularly in open luxations.

Diagnosis

Generally, the diagnosis can be made by physical examination alone. However, radiographs should be taken to identify concurrent fractures or damage to the articular surface and subchondral bone (Figure 4.138). Radiographs are particularly important in young foals to rule out the possibility of growth plate fractures as a primary cause or a secondary contributing cause to the angular deformity.



Figure 4.137. Image of closed subluxation of the left hind fetlock prior to reduction and cast application. Note the lateral aspect of the distal third metatarsal bone (arrow).

Treatment

Treatment of simple luxation of the fetlock can be rewarding. In most cases the injury is limited to the supporting soft tissues, and after the luxation is reduced under anesthesia, good axial alignment can be maintained by casting or splinting the limb until healing occurs. Prior to applying the cast, needle drainage of the hematoma (if present) that overlies the ruptured collateral ligament provides a better fit for the cast. Although centesis of the hematoma and cast application can be performed in the standing horse, general anesthesia and lateral recumbency are preferred. Reduction of the luxation is usually not difficult. A cast is applied that incorporates the foot and extends to just below the carpus or tarsus in the adult. Casts or splints are maintained for 6 weeks with stall rest. After cast or splint removal, bandage support and limited exercise are recommended. Swelling (thickening) will be noticed over the area of collateral ligament rupture, and a cosmetic blemish will remain. Additionally, some horses may have trouble placing their heel on the ground when coming out of the cast and may need a wedge shoe for a period of time. A gradual return to exercise is recommended because re-luxation of the fetlock can occur if collateral ligament healing is incomplete.

Success can be achieved without suture of the collateral ligament, although there are several reports in the literature on open repair. To repair the ligament, the end is located after surgical incision, debrided, and sutured.[109] Alternatively, a polypropylene mesh has been substituted for the ruptured ligament.[97] Arthroscopic removal of the articular fractures should be elected if full athletic performance is a goal. This is not necessary for light riding soundness if the fragments are small and from the palmar/plantar eminence. Occasionally large avulsion

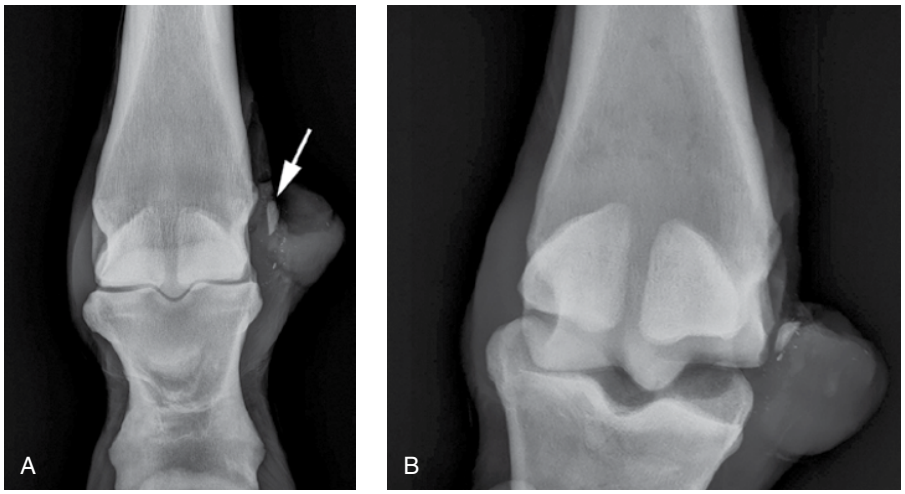


Figure 4.138. (A) Standing and (B) stressed DP radiographs of a horse with subluxation of the fetlock with fragmentation of the collateral ligament origin (arrow).

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fractures associated with the collateral ligament may warrant internal fixation to reconstruct the joint surface and improve the prognosis.

Acute open luxations can also be managed successfully.[88] However, prior to immobilization, thorough debridement of all devitalized soft tissues, bone, and cartilage should be performed. The joint should be thoroughly lavaged, and antimicrobials placed within the synovial cavity. IV regional perfusion also is warranted. If the joint is minimally contaminated and was treated immediately, a primary suture apposition of the soft tissues is recommended. A distal limb cast is then applied and removed when deemed appropriate based on the horse's progress. However, cast immobilization is usually required for 6 weeks minimum.

Prognosis

The prognosis for simple luxation of the fetlock is good for breeding soundness but guarded for athletic performance. In one report, 7 of 10 horses, some with open luxations, could be used for breeding, and 1 horse was riding sound.[109] Often the final prognosis cannot be accurately assessed until follow-up radiographs are taken at 2–3 months after injury. Immediate stabilization of the joint usually permits healing without the development of significant OA. Lack of stabilization or unrecognized luxation often results in OA in horses allowed exercise.[81]

Acute open luxations exposing the fetlock joint also respond well to early therapy as described. However, one must always be reserved in giving a good prognosis until joint infection and OA from direct trauma have been ruled out as complications. The long-term outlook for return to serviceability is often dependent on the degree of initial trauma sustained by the bone and the severity of secondary OA.

CONSTRICTION OF OR BY THE FETLOCK PALMAR/PLANTAR ANNULAR LIGAMENT

The palmar/plantar annular ligament (PAL) is a tough, fibrous, thickened, relatively inelastic part of the digital flexor tendon sheath (DFTS) strategically located to support the tendons as they course around the palmar/plantar aspect of the fetlock joint. The PAL together with the sesamoid bones provides a canal for the tendons to glide through. The PAL is lined with synovia and acts to prevent displacement of the tendon, which reduces its mechanical efficiency. Tendons within any tendon sheath and supported by an annular ligament can become enlarged from injury or infection. The chronic tendon enlargement/inflammation within the sheath can also create adjacent PAL thickening. Primary injury to the PAL can also occur, resulting in thickening (desmitis) and a subsequent reduction of the diameter of the canal.[65] Compression of the tendon and pressure within the canal subsequently restricts the free gliding function of the tendon, inducing pain and possibly ischemia to the tissues within the canal, analogous to carpal tunnel disease in people.

Constriction of or by the fetlock PAL can be a cause of lameness in performance and general-purpose riding horses. Although all breeds and uses of horses can be affected, sport horses, racehorses, Paso Fino horses, and Warmblood horses appear to be at a greater risk. In one report desmitis of the fetlock annular ligament was diagnosed mainly in sport horses and less frequently in racehorses.[98] In an unpublished report on 49 horses, Paso Fino horses and Warmblood types were overrepresented compared with the normal hospital population.[96] In another study, middle-aged to older general-purpose riding horses predominated, and PAL desmopathy occurred more frequently in the hindlimb than in the forelimbs.[65]

Etiology

Constriction of or by the fetlock annular ligament usually occurs as a result of trauma and/or infection. The injury may involve the annular ligament, primarily resulting in inflammation and thickening (desmitis) of the structure and thus producing constriction without abnormalities in other structures within the fetlock canal. Alternatively, the condition may be associated with distal superficial digital flexor tendon (SDFT) tendinitis or longitudinal tears in the deep digital flexor tendon (DDFT).[101] Due to the fact that the PAL is minimally elastic, swelling of the digital flexor tendons caused by fibrosis and associated tenosynovitis can cause the signs of constriction of the annular ligament. Inflammation and subsequent scar tissue enlargement often associated with a low flexor tendinitis ("low bowed tendon") may extend to involve the PAL, also resulting in desmitis. Adhesions may form between the SDFT and the annular ligament, further restricting motion. Tendinitis of the DDFT within the fetlock canal also enlarges the tendon within the canal and induces a tenosynovitis and produces the signs of annular ligament constriction. Wounds such as wire cuts or nail punctures can occur in the region of the palmar/plantar aspect of the fetlock, producing thickening, tenosynovitis, and constriction. One report revealed that primary desmitis of the annular ligament was the cause in 55% of the cases, tendinitis was the cause in 31%, and sepsis was the cause in 14% of the cases.[96] Synovial masses within the DFTS may also produce a constriction of the annular ligament and signs of tenosynovitis.[33]

Clinical Signs

Horses often present with a history of lameness that increases with exercise, resolves with rest, and returns when put back into performance. The most notable feature of annular ligament constriction is swelling of the palmar/plantar soft tissues of the distal limb around the fetlock and a characteristic observable proximal border of the annular ligament ("notching"). In nearly all cases, there is distension of the DFTS proximal and distal to the PAL.

The lameness is characterized by its persistence, pain on fetlock flexion, and worsening with exercise because of inflammation and increased constriction. In the most severe cases the horse is reluctant to place the heel on the ground and will accentuate the cranial phase of the stride to compensate. The continual external pressure on the tendons may produce abnormalities in the digital flexor tendons.[55]

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Perineural anesthesia, first of the foot and subsequently of the distal metacarpus or metatarsus, eliminates the lameness and locates the source of the lameness to the region of constriction. Direct anesthesia of the tendon sheath is usually not necessary, but may be performed if a fluid sample is being obtained for cytology or if the sheath is injected as a conservative treatment. However, anesthesia of the DFTS is fairly reliable for documenting painful conditions within the sheath. In horses that respond dramatically to tendon sheath anesthesia, tenoscopy of the tendon sheath is often indicated.

Diagnosis

The physical findings and the results of diagnostic anesthesia provide a presumptive diagnosis. Ultrasonography is indicated to identify the structures involved and assist with the selection of treatment. Normally the annular ligament is thin (less than 2 mm). One study found that measurement from the external skin surface to the internal annular ligament surface was the most reproducible index of annular ligament thickening.[65] Lesions identified at ultrasound vary and can include core tendon lesions or longitudinal tearing of the DDFT and SDFT, fibrosis, tenosynovitis and synovial proliferation, adhesions, and thickening of the annular ligament.[30,65,96] Subcutaneous fibrosis associated with PAL is common and should be differentiated from true thickening of the annular ligament itself.[65]

Radiographic evaluation of the fetlock region should always be performed to evaluate possible osseous involvement, particularly of the sesamoids. In lesions associated with wounds or of traumatic origin, osteomyelitis or sequestrum of the sesamoid bones can be present concurrently with PAL constriction. This finding necessitates additional treatment and decreases the prognosis for soundness or elimination of infection. In one study of 38 cases of annular ligament constriction, 6 horses had proximal sesamoid bone abnormality, and 12 horses had bone enthesophytes at the attachment of the annular ligament (insertional desmopathy).[87]

Treatment

Treatment of affected horses is often dependent on the stage of the disease, concurrent abnormalities, and intended use of the horse. Options usually include conservative medical therapy or desmopathy of the PAL with or without tenoscopy.[65] Adjunctive surgical procedures to simultaneously treat tendinitis, tendon core lesions, adhesions, and synovial proliferation are indicated if the underlying conditions exist. Conservative therapy is usually reserved for horses in the early stage of the disease or those with primary PAL desmitis and usually consists of oral and topical anti-inflammatories, intrasynovial treatment of the DFTS, controlled exercise, and bandaging the limb. The most commonly used surgical options for transection of the PAL is either a percutaneous approach or transection of the PAL concurrently at the time of tenoscopy. Tenoscopy of the DFTS is currently the preferred method to address synovial masses and tendon lesions and perform the annular ligament transection (Figure 4.139). Tenoscopy offers the advantage of visualization of the sheath and synovium and the tendons partially. Resection of adhesions, proliferative synovium, and tendon splitting can be performed as desired.[33,101] If tenoscopy is not an option, then a percutaneous approach either above or in the middle of the PAL can be performed. The PAL is transected either with curved scissors or a curved blunt-tipped bistoury. Although many feel that surgical transection is the ideal treatment, in one study of 71 horses with PAL injuries, the type of treatment did not appear to influence the outcome.[65]

Prognosis

Horses with PAL desmopathy alone that was not accompanied by abnormalities in the tendon (bowed tendon) had a good prognosis following surgical transection (84% returned to performance).[35,99] Other studies have reported a range of 50%–87% of horses with desmitis of the PAL returning to athletic function.[55,65] If SDF tendinitis is present, the tendinitis rather than the constriction appears to limit the performance. In one study, of the 13 horses with SDF tendinitis together with

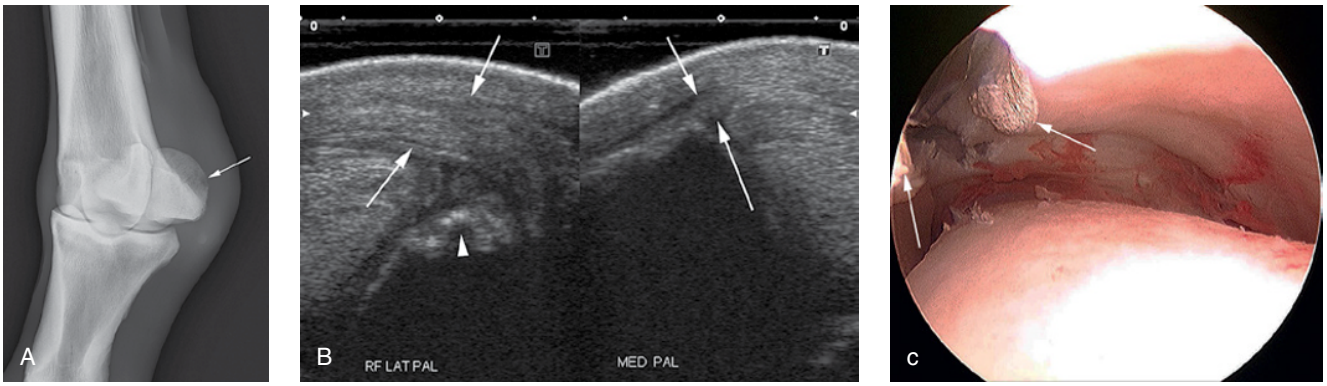


Figure 4.139. This horse sustained a wound previously over the lateral sesamoid bone of the fetlock. (A) DLPMO radiograph depicting the focal lucency in the lateral proximal sesamoid (arrow) with osseous reaction distally in the sesamoid. (B) Ultrasound examination of the fetlock demonstrating marked thickening of the lateral aspect of the PAL compared with the medial aspect (arrows). There is also irregularity of the lateral aspect of the proximal sesamoid (arrowhead). (C) Tenoscopic image after transection of the markedly thickened palmar annular ligament (arrows).

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annular ligament desmitis treated by annular ligament resection, only 38% became sound.[96] Of horses with longitudinal tears of the DDFT, 58% became sound.[101] Of 25 horses with synovial masses and adhesions, 72% became sound.[33] Some horses with primary desmitis can become sound with rest alone.[55] Bilateral PAL thickening, concurrent limb injuries, subcutaneous fibrosis, and lesions within the DFTS all have a negative effect on prognosis.[65]

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THE METACARPUS AND METATARSUS

Kyla F. Ortved and Alicia L. Bertone

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BUCKED SHIN COMPLEX AND STRESS FRACTURES OF THE DORSAL THIRD METACARPUS

Bucked shins and stress fractures of the dorsal surface of the third metacarpal bone constitute a spectrum of diseases that are observed in young (2–3 years of age) fast gaited horses. Although the greatest incidence is observed in young racing Thoroughbreds, it also affects young Quarter horses and occasionally racing Standardbreds (Figure 4.140). The hindlimbs are much less commonly involved. Overall, the incidence of bucked shins and stress fractures has decreased significantly in the last 10 years, most likely due to changes in training methods.



Figure 4.140. A racing Quarter horse with the classical metacarpal profile of a dorsal cortical stress fracture.

Etiology

Different subdivisions of this disease are described later; however, it appears that all clinical syndromes have a common pathogenesis.

In young horses put in training, the metacarpal bone is exposed to new stresses. In younger horses (2-year-olds), the metacarpal bone is less stiff, and therefore, greater strains are measured on the dorsal cortex during high-speed exercise than in older horses. This high-strain environment may lead to cyclic fatigue of the bone and subsequent bone pain (bucked shins, sore shins).[94] Dorsal metacarpal strain is even greater in the lead limb (the left limb in racing in the United States), correlating with the most common location of dorsal cortical stress fractures.[28]

During normal maturation of the third metacarpus, horses aged 1–2 years resorb primary osteons and have a greater amount of resorption cavities and incompletely filled secondary osteons than older horses. This bone structure is more susceptible to fatigue microdamage resulting from training because of higher bone porosity, fewer completed secondary osteons, and a lower proportion of circumferentially oriented collagen fibers.[133] In racing Quarter horses put in training, the bone density of the medial and lateral dorsal metacarpus significantly decreased during the first 62 days of training and then significantly increased from day 104 to day 244 of training.[52,90] Horses experienced fewer bone-related injuries when they had greater cortical mass in the lateral aspect of the third metacarpus at the commencement of training.[90] The changing shape of the metacarpal bone during maturation[93] and training[29,122] is consistent with lower strains during high-speed exercise in older horses.

The thickening of the dorsal cortex, including diffuse dorsal periosteal new bone seen particularly in horses in training, is likely a natural response to these demands on the bone to withstand stress without developing microfractures or dorsal cortical fractures (Figure 4.141). This has been further confirmed by experimental exercise conditions that induced marked modeling and remodeling of the dorsal cortex, particularly subperiosteal bone formation at the midshaft of the third metacarpal bone. Additionally, horses completing the full training program had greater mineral bone content despite a lighter body weight.[83] Foals significantly increase metacarpal bone mineral density with age, and lack of exercise retards the normal bone development.[25]

In summary, in horses in race training, high strains can induce cyclic fatigue of bone, resulting in microdamage or ultimate bone failure. The body responds with bone modeling and remodeling, but microfracture damage may develop and cause pain. Historically, the majority (more than 80%) of 2-year-old racing Thoroughbreds[6] and many racing Quarter horses[52] demonstrated dorsal cortical pain with an estimated 12% that went on to develop acute failure or dorsal cortical fracture, usually within 6 months to 1 year of showing dorsal cortical pain.[93] Transformational clinical and experimental studies performed during the 1990s evaluating bone modeling and remodeling in Thoroughbred racehorses demonstrated that galloping long distances increased the likelihood of bucked shins by 36.4%, while breezing short distances reduced the likelihood of bucked shins

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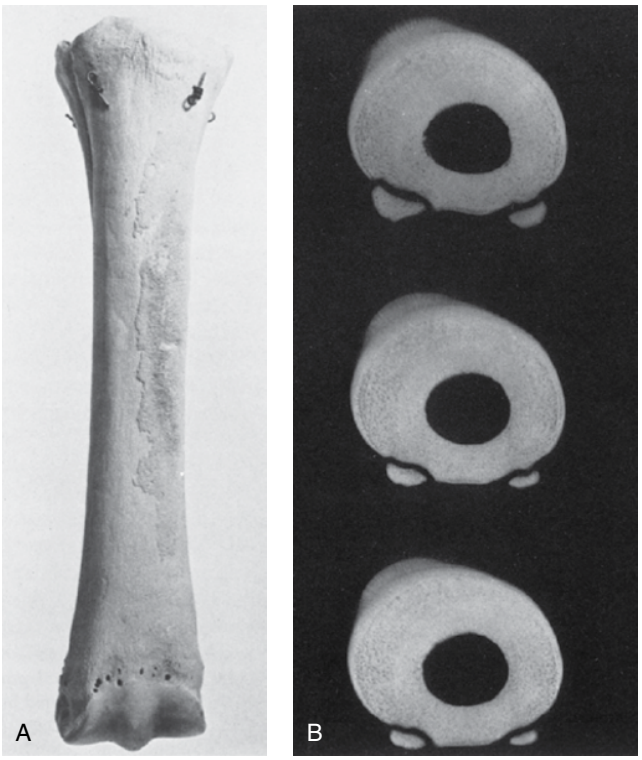


Figure 4.141. (A) A macerated specimen of the right third metacarpal bone illustrating subperiosteal new bone on the dorsal medial surface. (B) Cross section through the third metacarpal bone of a horse with dorsal metacarpal disease. Note that the dorsal cortex is thicker than the palmar cortex. *Source:* Courtesy of Dr. PF Haynes.

by 98.6%.[16,94] These studies also found that slow-speed exercise produced tensile strains on the dorsal cortex of the metacarpus, whereas high-speed exercise produced compressive stains on the dorsal cortex. Since that time, the incidence of bucked shins has dramatically decreased, as most Thoroughbred horses are now trained shorter distances, at higher speeds. The incidence of fatigue failure of the metacarpal dorsal cortex continues to be greater in Thoroughbreds than Standardbreds. This was not attributed to inherent differences in the mechanical properties of the bone, but rather gait differences and resultant bone stresses during training and racing.[95]

Clinical Signs

Early dorsal metacarpal disease is usually observed in young Thoroughbred racehorses (2–3 years old) as they begin intense race training and occasionally in older horses that have not been strenuously trained or raced as 2-year-olds. The disease has an acute onset and is most obvious after intense exercise. Horses can be described as having a short, choppy gait; however, overt lameness is usually not present. Additionally, any gait deficits often resolve after short periods of rest. A visible convex swelling overlying the surface of the affected portion of the cannon bone is common. On palpation, the dorsal cortex of the third metacarpal bone is acutely painful to pressure or concussion. Frequently the horse will withdraw the limb in response to pain. Radiographs taken at this time are usually negative; however, fractures are occasionally observed as well as a minimal amount of superficial cortical osteolysis.

Subacute to chronic dorsal metacarpal disease invariably develops as a result of acute disease that is unresponsive to therapy or has gone unrecognized. It is most frequently seen in horses 2–4 years of age.[86] Only mild degrees of gait deficit may be noticed at exercise. On palpation, varying degrees of pain may be elicited, but a more obvious enlargement is palpable on the dorsomedial cortex (Figure 4.140). The pain response is typically more profound after strenuous exercise, and the left limb is usually more severely affected. Periosteal new bone formation is usually observed on radiographic examination (Figures 4.140 and 4.141).

Dorsal metacarpal bone failure or stress fractures usually occur in the dorsolateral cortex, although fractures can occur in the dorsal or dorsomedial cortex. It is usually observed in older horses, 3–5 years of age, that have a history of bucked shins. As with dorsal metacarpal disease, the lameness may not be prominent while the horse is rested. However, it is usually prominent after strenuous exercise. On palpation, a rather discrete painful area can be palpated on the dorsolateral, or less commonly dorsomedial, surface of the left third metacarpal (cannon) bone at the junction of its middle and distal third. Only rarely is the right third metacarpal bone involved. Radiographs usually point to a cortical fracture on the dorsolateral surface (Figure 4.142).

Diagnosis

A tentative diagnosis of bucked shins or a dorsal cortical stress fracture can be made from the clinical findings and the age relationship. Little information is derived from local direct infiltration anesthesia of the painful area because it provides only partial relief in the lameness. Median and ulnar nerve perineural anesthesia or proximal metacarpal regional anesthesia with a ring block is required for total relief of pain, but this is rarely indicated.

Radiographs can assist with the diagnosis of dorsal cortical stress fracture. A series of four radiographic views should be taken: dorsopalmar (DP), lateromedial (LM), dorsolateral to palmaromedial oblique (DLPMO), and dorsomedial to palmarolateral oblique (DMPLO). The DLPMO and the LM best identify the dorsal medial bone proliferation, and the DMPLO and LM best identify the dorsolateral cortical fractures. Horses continuing to train and race with sore shins can be screened for the development of fractures with radiographs. Some fracture lines can be difficult to see; serial oblique radiographs can be helpful.

It is rare to see radiographic abnormalities in acute dorsal metacarpal disease. In subacute dorsal metacarpal disease, periosteal new bone formation (of reduced density compared to underlying cortex) and surface cortical bone resorption may be observed. In chronic disease, a thickening of the dorsomedial cortex with associated periosteal new bone formation is seen. Occasionally an endosteal response may be observed in the advanced cases. Dorsal cortical fractures have a characteristic radiographic appearance and usually enter the cortex distally and progress proximad at a

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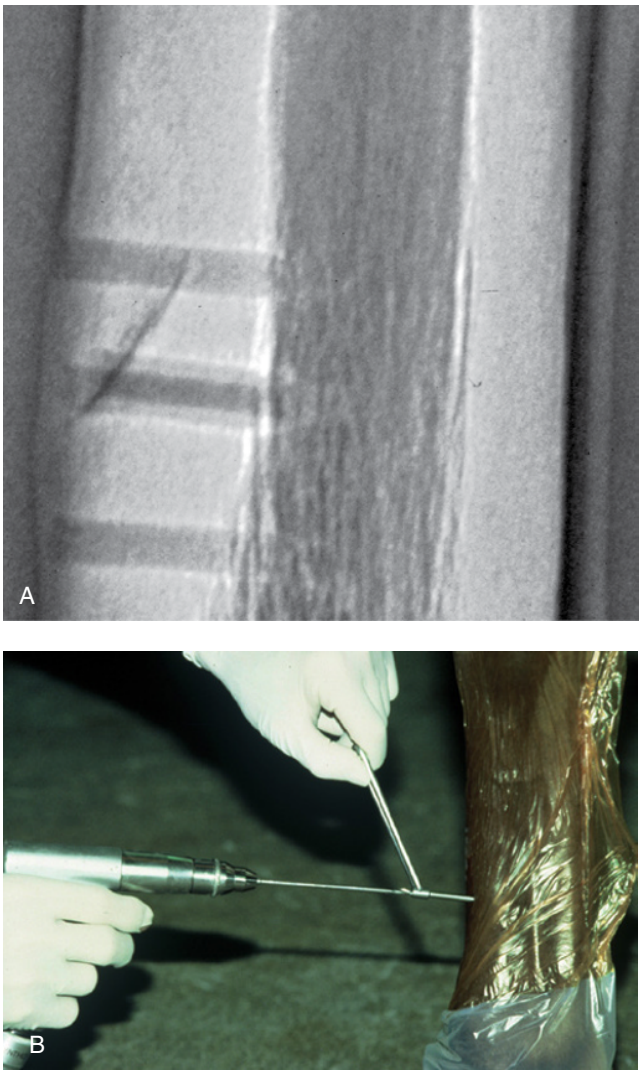


Figure 4.142. Radiograph of a dorsal cortical fracture treated with osteostixis procedure (A) that was performed standing (B).

35–45° angle. On occasion, dorsal cortical fractures can enter the cortex proximally and course distally.

Most frequently the fracture appears in radiographs as a straight or slightly concave fracture line (tongue fracture) (Figure 4.142). Occasionally, the fracture line exits the proximal (or distal) cortex such that a saucer fracture is produced. Rarely, the fracture continues to enter the medullary canal. Multiple fractures may emanate from the distal site of the cortical entry (often termed "fissure fractures"). Periosteal callus is often present at the site of fracture and is a function of the chronicity of the disease. Endosteal proliferation is observed occasionally in fractures that are completely through the cortex. Repeated radiographs at 7- to 10-day intervals may be necessary to identify a fracture that is suspected but not observed on initial radiographic examination.

Nuclear scintigraphy can provide information about the stage of disease in horses showing dorsal cortical pain or in those with undiagnosed forelimb pain. The sensitivity of this technique to identify bone metabolism and turnover is high, and it allows detection of abnormalities in horses in the acute–subacute stages that are still in training. Racehorses may have bone scintigrams performed to determine whether training should continue or to identify focal uptake indicative of impending fracture. In a review of 121 bone scintigrams of racing Thoroughbreds with clinical history of dorsal metacarpal disease, horses without fractures (bucked shins) had a mild to moderate diffuse uptake of radioisotope, unlike the focal intense uptake typical of dorsal cortical fracture. Chronic fractures are less focal and intense because of surrounding bone remodeling; however, they still appear relatively focal compared to early disease.

Standing robotic cone-beam computed tomography (CT) might be useful in defining difficult-to-see fractures. Use of quantitative CT to measure bone mineral density and three-dimensional morphologic changes in the metacarpus may provide improvements in identifying risk factors or imminence of fracture.

Treatment

There is no specific medical treatment for dorsal metacarpal disease, necessitating that horses be laid up or put on a convalescent exercise program to provide time for the early acute changes to subside. Many horses with acute bucked shins can continue to train after 5–10 days of rest and anti-inflammatory analgesics. Hand walking, ponying, cold water hosing, and bandaging should continue until the dorsal cortex can be palpated without eliciting pain. Speed and distance are introduced slowly, with constant monitoring of dorsal cortical pain. Initially, daily galloping distance is reduced to 50%. An overall modification of the training program with less galloping miles and more short-distance breezing may be necessary. The overall goal is to gradually increase the stress to the dorsal surface of the metacarpal bone at such a rate such that this surface can model according to compressive demands without producing structural damage.

Subacute and chronic dorsal metacarpal disease can be the most difficult to treat. After an exacerbation, many of these horses may not be suitable for the modified training regimen described above, and pain immediately returns with any sustained galloping. These horses may have marked periosteal new bone formation. More prolonged rest is usually necessary for bone remodeling of this new periosteal bone and remodeling of fatigued bone. The time required is usually 110 days.

Dorsal cortical fractures in young horses may resolve with the conservative approach outlined above for subacute or chronic bucked shins. Convalescent periods may extend from 4 to 6 months because fracture healing is slow at this site. In either case, serial radiographic studies should be performed at least every 30–45 days to assess the bone healing.

Several surgical procedures have been recommended for treatment of dorsal cortical metacarpal fractures, including placement of a unicortical screw in lag fashion, placement of a neutral unicortical positional screw, and dorsal cortical drilling or osteostixis (Figures 4.142B and 4.143). Transcortical screws are not recommended due to the expected differences in strain between the palmar and dorsal cortices, risk of fracture, and risk of damage to the suspensory ligament (SL). Placement of a dorsal unicortical screw in lag fashion can be technically

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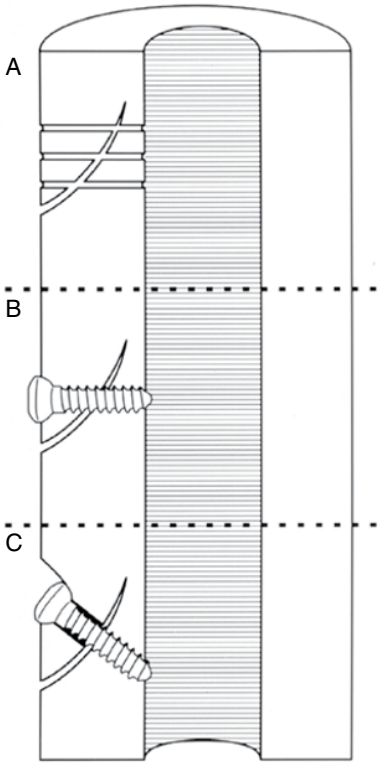


Figure 4.143. (A) Dorsal cortical drilling (osteostixis) of a dorsal metacarpal stress fracture. (B) Positional unicortical screw placement. (C) Unicortical screw placement in lag fashion.

difficult due to the short depth of the cortex (about 22 mm) and need for radiographic control, but provides the best fracture stability. Placement of a neutral dorsal unicortical screw to help stabilize the fracture, combined with dorsal cortical drilling, is often elected to provide added stability over dorsal cortical drilling alone. For screw fixation, cortical bone screws (4.5 or 3.5 mm) are used.

Many surgeons currently recommend removal of the screw after a sufficient time of healing (2 months). Many horses have and will race successfully with the dorsal cortical screw in place. However, reoccurrence of dorsal cortical pain may occur (regardless of the presence of a screw), and the screw will be presumed to be the cause. If any fracture occurs in this horse in the future, such as condylar fracture or complete metacarpal failure, the screw may be considered a cause. Screws can be easily placed and removed standing.[23,27,57,129] Many surgeons prefer standing fracture fixation to avoid risk of catastrophic fracture during recovery from general anesthesia. Fractures can be repaired standing with sedation and perineural anesthesia using radiographic guidance.

Postoperative management includes stall rest and bandaging for 2 weeks, followed by 6 weeks of stall rest with hand walking. Screw removal is usually performed at 8 weeks, followed by an additional 2 weeks of hand walking. Tack walking and light jogging can be introduced 2 weeks after screw removal; however, more intense race training should not commence until 4 months postoperatively. Greater than 95% of horses can return to racing in approximately 8 months.[27]

Osteostixis can be performed under general anesthesia or in the standing horse (Figure 4.142B). Holes (5–7) are made with a small drill bit (2.0–2.5 mm) in a diamond pattern through the dorsal cortex into the marrow cavity. This procedure can be combined with unicortical screw application. Success for return to racing is similar (greater than 80%) to that recently reported for screw fixation, and a quicker return to racing (4–6 months) has been reported.[57] Clustered drill holes act as a stress concentrator and significantly decrease the stress required for metacarpal failure in cadaver limbs, but catastrophic failure through the drill holes is not a reported complication of the procedure *in vivo*.[129,130]

Other adjunctive treatments have been recommended with or without surgical treatment, including electrical stimulation at the fracture, extracorporeal shockwave therapy, injection of osteogenic substances (sodium oleate), intralesional injection of steroids, thermocautery (pin firing), chemical vesication (blistering), needle drainage of the hematoma, and cryotherapy (point freezing). These treatments have met with varying degrees of success, and no controlled studies have been performed. In all cases, no matter what the treatment, an adequate period of rest combined with a controlled exercise program is required.

In one study of Thoroughbred racehorses, distinct training strategies were used at various stables, and the allocation of exercise to breezing (15 m/s), galloping (11 m/s), and jogging (5 m/s) was associated with lack of bucked shins for 1 year (survival of bucked shin syndrome). Survival was significantly reduced by allocation of exercise to breezing and increased by allocation to galloping. It was recommended that to reduce the incidence of bucked shins, trainers should allocate more training effort to regular short-distance breezing and less to long-distance galloping.[16]

Prognosis

The prognosis is good to excellent for return to racing with surgical treatment of dorsal cortical fractures; reports range from 80% to 98%. However, this underestimates the loss of racing days of horses with sore shins that remain in training and recurrence of pain or fracture once subacute or chronic disease occurs. The impact of this syndrome, particularly in 2-year-olds, is evidenced by the observation that if 2-year-old racing Thoroughbreds were not permitted to race for 6 weeks if sore shins are palpated prerace, significant improvements in predictable finishes occurred.[55] Adjustment of training regimens may assist with prevention, and training on grass, wood fiber, or softer surfaces without toe grabs is recommended.

Other Stress Fractures of the Third Metacarpal/ Metatarsal Bone

Although dorsal cortical stress fractures are by far the most common location of stress fracture in horses, particularly racehorses, other sites and variations within the metacarpus can occur. Dorsal cortical fractures may extend more proximal than the site of exit from the cortex, and fissure lines can sometimes be identified, most typically in the proximal metacarpus on other views. If a fracture line is noted on the craniocaudal view, a spiraling

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fracture or nondisplaced complete metacarpal fracture should be suspected. Fractures may be limited to the dorsal cortex and respond to lag screw fixation. Detailed radiographic investigation should be performed to define the fracture and determine whether confinement to an overhead wire, casting, or surgery and compression of the fracture with screws is recommended.[102,132,138] Similar fractures can occur in the metatarsus of the hindlimb.[102]

Unicortical fractures of the distal palmar/plantar condyle have been reported with increasing frequency due to more sensitive diagnostic imaging techniques; nuclear scintigraphy, the 125° dorsoproximal to dorsodistal view, CT, and magnetic resonance imaging (MRI) can be used for identification. Treatment can involve rest for 3–4 months or lag screw fixation across the palmar aspect of the condyles. Horses have a good prognosis for return to racing.[68,106]

Primarily in racing Standardbreds, longitudinal stress fractures or focal intense radioactive areas of uptake on nuclear scintigraphy can occur in the proximal palmar/plantar aspect of the third metacarpus/metatarsus and is a recognized syndrome. Physical findings are very subtle, but lameness can be blocked to the proximal metacarpus and scintigraphy is diagnostic. Most horses have abnormal radiographs, although high detail film and several projections may be needed to see the fracture. Rest resulted in a 64% return to the previous level of performance.[78,103] Internal screw fixation[118] and surgical drilling[145] also resulted in success. Dorsomedial articular fractures of the third metacarpal bone also can occur in racing Standardbreds. Lameness is abolished with intracarpal anesthesia. Periosteal new bone production can be seen on radiographs near the distal aspect of the fracture. All seven horses in one study healed and returned to soundness (one with internal fixation).[117]

FRACTURES OF THE CONDYLES OF THE THIRD METACARPAL/METATARSAL BONES (CONDYLAR FRACTURES, LONGITUDINAL ARTICULAR FRACTURES)

Fractures of the condyles of the third metacarpal and metatarsal bones occur most frequently in racing Thoroughbreds, less frequently in Standardbreds, and occasionally in Quarter horses and Polo ponies; they comprise a subset of catastrophic breakdowns on racetracks.[8,96,97] Fractures usually occur at high speeds. Males are overrepresented (59% in one study and 75% in another), and the distribution of fractures is approximately one-third incomplete nondisplaced, one-third complete nondisplaced, and one-third complete displaced (Figure 4.144).[147] Fractures affect the lateral condyle more commonly. Fractures in the lateral condyle usually propagate toward the lateral cortex, whereas fractures in the medial condyle tend to spiral proximally with the dorsal fracturing line moving medial or develop a transverse component in the mid-diaphysis causing a "Y" fracture configuration. For these reasons, medial condylar fractures should be considered to be at a higher risk for catastrophic failure and treated appropriately. In a study of 145 condylar fractures in mostly Thoroughbreds, forelimbs (81%) and lateral condyles (85%) were more frequently involved. The right and left forelimbs were approximately equally involved. Most fractures (59%) were initiated in the middle of the condyle and extended a mean of 75 mm proximally to exit the cortex. Axial fractures and medial condylar fractures tend to be longer. Articular comminution can occur, usually at the palmar/plantar articular margin, and was identified in 15% of fractures. The more axial fractures were more likely to have articular comminution (23%). Eight of 145 fractures evaluated spiraled proximally. Concurrent lesions included proximal phalanx eminence fractures, sesamoid fractures, and sesamoiditis.[147]

In another study of 233 condylar fractures, Thoroughbreds were overrepresented, and Standardbreds underrepresented based on the hospital population. Standardbreds had more metatarsal condylar fractures. Medial metatarsal condylar fractures had a 25% chance of suffering a mid-diaphyseal fracture ("Y" fracture). In this study, the distribution of Thoroughbred condylar fractures was 66% forelimb, with approximately 50% complete. Seventy-nine percent of Thoroughbreds with incomplete lateral condylar fractures (77% treated surgically) returned to racing, whereas a significantly lower percentage (33%) of complete lateral condylar fractures returned to racing (all treated surgically). Eighty percent

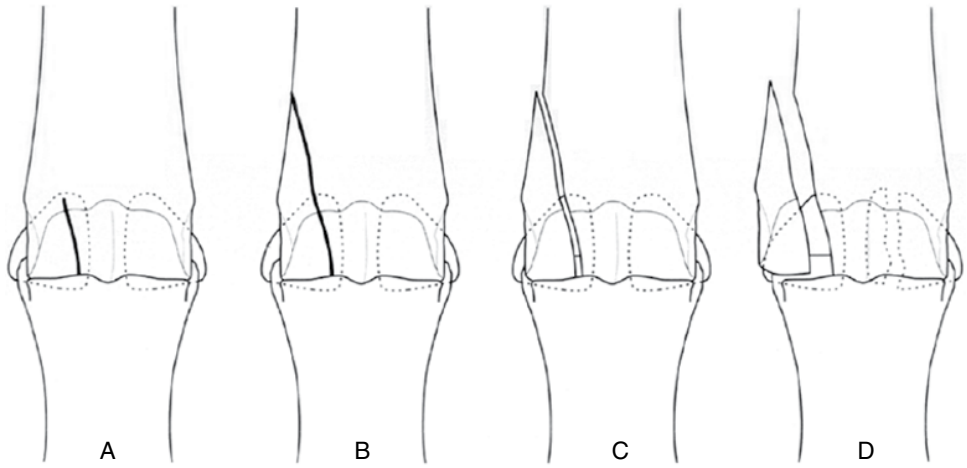


Figure 4.144. Condylar fractures. (A) Incomplete. (B) Complete nondisplaced. (C) Complete separated. (D) Complete displaced.

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of Standardbreds (6 of 7 treated surgically) raced after treatment. In summary, return to racing in Thoroughbreds was significantly reduced with complete fractures, forelimb fractures, evidence of sesamoid fracture, or female sex (presumed retired for breeding).[8] Complete condylar fractures tend to be longer (mean 85 mm) than incomplete fractures (mean 57 mm).[112] Axial sesamoid fractures are associated with displaced lateral condylar fractures that disrupt the collateral ligament and avulse the intersesamoidean ligament complex.[6]

Another report of 124 condylar fractures in Thoroughbreds corroborates the above findings of the more recent reports. It was noted that 90% of condylar fractures occurred in 2- and 3-year-olds and that most were acquired during training between April and October. In this report, 17% of displaced lateral condylar fractures returned to racing.[45] The variation in successful return to racing of displaced condylar fractures may be related to the duration of fracture prior to surgery. Destruction of the articular surface occurs quickly following a displaced fracture; therefore, immediate immobilization and repair are critical to a successful outcome.[111]

Condylar fractures of the hindlimb are more common in Standardbreds and are more likely to be medial and to not exit the cortex. These fractures can propagate proximally or progress to a complete "Y" fracture, even with stall confinement.[110,111]

Etiology

The etiology includes trauma from high compressive loads, asynchronous longitudinal rotation of the cannon bone, and exercise on uneven surfaces. The risk of fatal condylar fracture in Thoroughbred racehorses is 7 times and 17 times more likely if horses are shod with low or regular toe grabs, respectively.[66] The toe grab changes the hoof angle and presumably places more stress on the suspensory apparatus and plants the foot more securely. This likely alters the compressive and rotatory forces on the distal metacarpus.

Recent structural studies of the distal condyle of unexercised and exercised horses have demonstrated that the normal mineralized articular cartilage tends to cleave in the sagittal plane and that the main subchondral bone trabeculae are arranged in columns and run in the sagittal direction with fewer mediolateral connections. Blood vessel canals lie inside these sagittally oriented structures. The sagittal column orientation provides maximum strength and protection in the sagittal plane in which the joint rotates but offers minimal resistance to fracture propagation in this plane. The anatomical course of condylar fractures of the third metacarpal bone can be explained by the anisotropic structural arrangement of the mineralized tissues.[18] In horses in race training, densification (sclerosis) of the subchondral bone of the palmar/plantar surface of the condyles was identified by CT techniques,[114] and this correlated with linear defects in the mineralized cartilage and subchondral bone in the same region with intense surrounding bone remodeling.[115] Microcracks in the subchondral bone of the metacarpus may coalesce and represent a preexisting pathology in horses with catastrophic fracture.[87,97]

In a histopathologic study on North American racehorses, similar palmar condylar cartilage fibrillation with underlying bone sclerosis and areas of bone necrosis were noted. Evidence of active bone remodeling was present around the sclerotic zones with osteocyte necrosis apparent as prominence of subchondral vessels and osteoclastic activity. Fragmentation lines indicating matrix fragility and microfractures were apparent. This area of the condyle was grossly flattened.[92] Similar microdamage including microfractures in regions of osteoporosis and thinning of the zone of calcified cartilage was seen histologically in the bone of lateral condylar fractures obtained from fatal injuries.[134] In an experimental study with controlled training exercise, bone density, particularly in this palmar condyle region, was significantly greater in horses with high intensity exercise.[113] In summary, the palmar condyle is the site of maximum loading in racing, and the bone responds by increasing density (sclerosis). Bone fatigue failure occurs and, due to the normal columnar arrangement of the bone trabeculae, propagates acute failure in the configuration seen in condylar fractures (Figure 4.145).

Clinical Signs

The clinical signs may vary from a mild lameness that is exacerbated by exercise with little heat or swelling present with nondisplaced, incomplete fractures to severe lameness with heat, pain, and swelling in the acute, displaced fracture. The incomplete, nondisplaced fractures are often so subtle that they are missed on physical examination, but may be detected by radiographs, nuclear scintigraphy, or CT. However, fetlock joint effusion is usually present because fractures originate at the articular surface. This is best seen in the palmar or plantar recess of the fetlock joint capsule. The external swelling of the lateral side of the large metacarpal or metatarsal bone depends on the degree of separation of the proximal end of the fragment and is readily palpated in displaced fractures. More swelling is observed with greater separation of the fracture fragment. The degree of pain associated with the palpation and the amount of heat that is felt depends on the acuteness of the fracture. Fracture movement and crepitation also may be detected.

Increased lameness can be observed in a horse after it has been exercised and when the horse is circled to the affected side. Flexion and rotation of the fetlock usually result in sufficient pain to cause withdrawal of the limb. In the acute, displaced fracture, crepitation may be felt with rotation of the fetlock. Increased lameness can be observed when flexion tests are used in cases in which there is a fissure fracture. Preferably, if a fracture is suspected based on history, clinical signs, and joint effusion, radiographs should be immediately taken without further lameness examination or joint manipulations to decrease the risk of propagating the fracture or worsening the articular cartilage injury.

Diagnosis

It is advisable to take radiographs if a condylar fracture is suspected. Perineural and intrasynovial anesthesia should be considered only after it is confirmed that a

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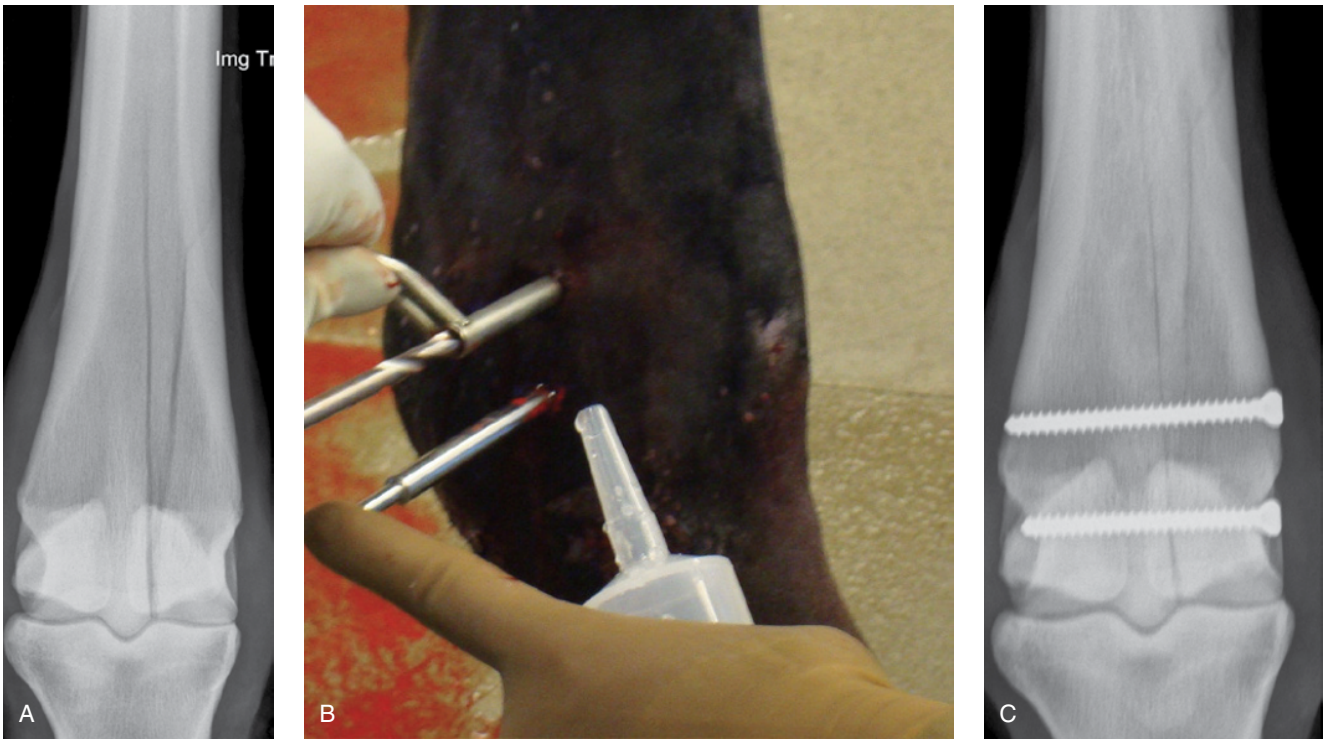


Figure 4.145. Radiograph of a spiraling medial condylar fracture in a racing Standardbred (A) repaired standing (B) with two parallel 5.5-mm cortical bone screws. At 4 weeks postoperatively, the compression at the screws is apparent and bone healing is progressing (C).

fracture is not present. A small percentage of these fissure fractures are difficult to identify radiographically, so it is important to closely scrutinize the radiographs.

Diagnosis is made with a standard series radiographic examination (DP, LM, DLPMO, and DMPLO). Additionally, a flexed DP view should be obtained to highlight the palmar/plantar surface of the condyles and evaluate the fracture line for comminution. Long cassettes are recommended so that the fetlock joint as well as the proximal cannon bone can be included in the study (Figure 4.145). Close evaluations of the study should follow to rule out the possibility of other injuries. Lesions that have been associated with condylar fractures include osteochondral fractures of the proximal phalanx (P1), fractures of the proximal sesamoid bone, osteoarthritis (OA) of the fetlock, palmar and plantar osteochondral lesions of the distal third metacarpal or metatarsal bone, SL desmitis,[73] and longitudinal fractures of the metatarsus. These associated lesions may be significant regarding the recommendation of treatment and the prognosis. Standing robotic cone-beam CT and nuclear scintigraphy can also be used to locate bone damage prior to fracture or to identify a fracture that is difficult to detect radiographically.

Treatment

The recommended treatment of most condylar fractures is internal fixation with transcortical screws placed in lag fashion for return to full athletic performance and health of the fetlock joint (Figures 4.145 and 4.146).[126] Incomplete, nondisplaced fractures can be treated conservatively with successful return to racing, and surgery is not always required. The advantages of screw fixation include the original fracture that will not become further displaced, shorter convalescence, a reduced incidence of refracture at the same site, and decreased risk of posttraumatic OA within the fetlock.

Horses with incomplete fractures that are treated with cast or support bandages should be confined to box stalls and observed closely for 2–3 weeks for any signs of increased pain and possible displacement. Bandages should be reset at least every other day for limb inspection. For conservative management of nondisplaced fractures, stall rest for 2 months followed by 30 days of hand walking and then 30 days of light turnout is recommended. Fractures in young horses can heal by 90 days, but the most articular edge of the fracture often requires additional time. Horses put in training at 90 days are at risk of articular osteolysis upon return to training.

Internal fixation should be used in all complete condylar fractures (nondisplaced and displaced) in horses intended for athletic performance (Figures 4.145 and 4.146). Immediate diagnosis and immobilization of the limb are critical preoperatively to enhance success. Surgery should be performed quickly but need not be performed on an emergency basis. The horse can be safely transported and permitted to recover from the incident if the limb is immobilized in a cast or complete commercial splint.

For surgery, the horse is positioned in lateral recumbency under general anesthesia with the affected limb up. Lag screws can be placed through stab incisions in the skin. The location and spacing of the screws can be preplanned by measurements made on the radiographs or preferably by using intraoperative fluoroscopic imaging. Regardless, the first screw is always placed in

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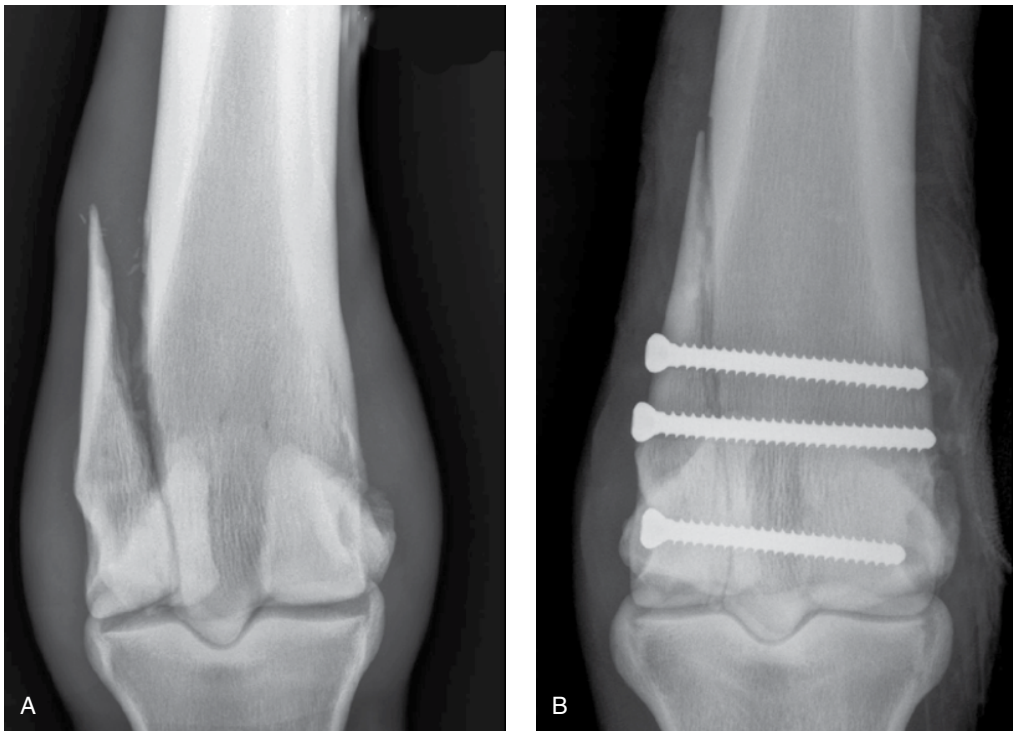


Figure 4.146. Craniocaudal radiograph of a displaced lateral condylar fracture in a racing Thoroughbred (A) repaired with three 5.5-mm cortical bone screws (B).

the supracondylar fossa, and subsequent screws are placed approximately 20 mm proximal to the first one. Most lateral condylar fractures can be repaired with two screws. Ideal screw placement is parallel to the joint with the first screw in the supracondylar fossa, engagement of both cortices without depositing bone material in the opposing collateral ligament, and compression of the fracture so the fracture line is eliminated or barely identifiable on the immediate postoperative radiograph. Increased duration and displacement of the fracture reduce the success in obtaining the latter result. Fractures are usually repaired with 4.5- or 5.5-mm cortical bone screws, although some success using headless titanium compression screws has been reported.[47,105] CT should be performed prior to starting the surgery to understand the fracture propagation if there is any question about the presence of a spiraling component to the fracture. This may affect the decision to use a bone plate in addition to screws and screw placement.

Displaced fractures can be compressed with Association for the Study of Internal Fixation (ASIF) bone clamps prior to drilling to stabilize the fracture and initiate compression or can be held in reduction by a Steinmann pin placed in the glide hole. Reduction of displaced condylar fractures can be a challenge, particularly in the cranial to caudal direction that may not be apparent on craniocaudal radiographs taken at surgery. Arthroscopic evaluation of the articular alignment should be used in all displaced fractures. Arthroscopic inspection of the dorsal articular surface often reveals a 1- to 2-mm step in the cranial direction of the fractured component. Arthroscopy also allows debridement of other bone fragments such as P1 eminence fractures or comminution of the palmar fracture line. Articular damage also can be assessed, which may affect prognosis in displaced fractures, particularly those of more chronic duration (days to weeks).

In displaced fractures, an open incision extending the full length of the fracture, use of multiple bone clamps, and initial focus on the reduction of the most proximal portion of the fracture may help maximize reduction. Screws (4.5- or 5.5-mm cortical bone screws) are tightened distal to proximal and retightened to maximize compression.

Medial condylar fractures, especially in the hindlimbs, are at risk of catastrophic failure if they propagate proximally and either spiral or develop a Y component middiaphysis.[3] Lateral condylar fractures can uncommonly spiral proximally. These fractures can be treated with multiple screws placed in lag fashion,[119,126,144] although plating techniques are likely the most reliable fixation method.[53] Locking compression plates (LCPs) can be applied using a minimally invasive approach to reduce incisional complications. Lag screw fixation can be performed with the horse standing to reduce the risk of catastrophic breakdown on recovery from anesthesia (Figure 4.145).

Casting is usually not performed for recovery. A full-limb cast has been recommended for long spiraled fracture repairs, but not all surgeons use this because there is a risk of complications in recovery.[110] Horses are usually comfortable on the limb immediately after repair and walk in the stall without gait deficit when on low doses of phenylbutazone. Continued lameness postoperatively is a red flag for problems and radiographs should be obtained.

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Postoperative exercise should be limited for at least 3 months. Stall rest confinement is recommended for this period, and after 2 months hand-walking exercise can begin. Follow-up radiographs should be taken 3 months postoperatively, and if the healing is progressing normally, the horse can be turned out into a small paddock for an additional month. The training schedule depends on the type of fracture but could begin as early as 4 months or as late as 6 months. In one study horses were more likely to race if the fracture line could no longer be identified at the 2- to 4-month radiographs.[146]

Screws are generally not removed after healing has occurred unless residual lameness results that can be directly attributed to their presence. In a review of 233 cases of condylar fractures, most repaired by interfragmentary compression, screws were only removed in 20 of the cases, and there appeared to be no consistent difference in the performance between this group and the group with screws in place.[8] This study noted that horses with screws placed closer to the joint had a lower return to racing. Subchondral sclerosis was proposed as a cause for this pain. In general, removal of screws is not routinely performed for condylar fractures except if screws are placed in the diaphysis such as in medial condylar fractures of the metatarsus or spiraling fractures. Screw removal is easily performed under general anesthesia; the location of the screws can be confirmed with marker needles and radiographs. In most cases the screws can be removed through small stab incisions alone. Bone plates are always removed in horses intended for athletic pursuits following surgery. Standing plate removal can usually be performed 3 months following surgery, provided the fracture has healed well. Stall rest for an additional 30 days is required after plate or screw removal, followed by stall rest with hand walking for an additional 30 days.

Prognosis

The general prognosis for athletic performance and returning to racing is excellent for nondisplaced incomplete fractures, whether treated conservatively (82% and 87% in two reports)[8,146] or following internal fixation (74% and 79% in two reports) for forelimb fractures,[8,146] and 93% for metatarsal fractures in one report.[8] Prognosis for athletic performance and return to racing is fair for complete displaced and nondisplaced fractures following internal fixation (58% in one report).[146] In two other reports, 33% of complete fractures (displaced and nondisplaced)[8] and 19% of displaced fractures[45] returned to racing. Rapid immobilization and repair of displaced fractures probably can dramatically affect outcome. In cases of complete displaced fractures or when there has been a delay in diagnosis and treatment and/or improper immobilization has been selected, a guarded to poor prognosis can be expected.[112] Only 12 of 38 cases in one report were able to return to racing.[112] This prognosis is altered because of increased damage to the articular surface that is likely to result in OA of the fetlock. The prognosis for return to racing is considered poor for comminuted fractures or subchondral bone lesions in the palmar or plantar surface of the distal end of the cannon bone.[112]

FRACTURES OF THE THIRD METACARPAL/METATARSAL (CANNON) BONE

Fractures of the third metacarpal/metatarsal (cannon) bone occur commonly in all ages, but more commonly in young horses, and all breeds of horses. The metacarpus/metatarsus is particularly susceptible to fracture because of the distal location and because little soft tissue covers the bone to help absorb impact energy in blunt trauma.[120]

Although fractures of the cannon bone can assume a variety of configurations, ranging from a simple fissure to severe comminution, younger horses seem to sustain simpler fractures than adults, possibly because of more elastic and less brittle and shatter-prone bone. Frequently, distal fractures that involve the growth plate, or rarely persistent proximal physes, in young animals are Salter type II fractures (Figure 10.7).[79] Because of the minimal soft tissue covering, the fractures are commonly open or become open soon after the injury occurs. More than half of referred metacarpal/metatarsal fractures are open.[84] Concurrent fractures of the small metacarpal bones are common. Stress fractures of the metacarpus and metatarsus in racehorses that can progress to acute and complete failure of the bone are discussed earlier in this chapter. Once catastrophic failure occurs, fractures are treated as discussed below.

Etiology

External trauma in any form can cause fracture of the cannon bone, e.g. kicks and falls. When foals are affected, the dam has often stepped on the limb, causing a fracture. Propagation of stress fractures or propagation of forces through screw or transfixation pin holes can result in similar complete cannon bone failure.

Clinical Signs and Diagnosis

Complete yet nondisplaced fractures of the cannon bone secondary to direct trauma can occur and may be difficult to initially diagnose (Figure 4.147). The lameness may be nonspecific and variable. Heat, swelling of the soft tissues overlying the fracture, and pain on deep digital palpation are usually present. A wound at the site of impact is also often present. Diagnosis may be delayed if soft tissue injury is evident because the lameness may be attributed to this cause. As lameness persists or worsens, radiographs are often taken and may reveal fracture lines. If the horse is turned out, invariably complete bone failure occurs. If stall confinement is maintained, the diagnosis is often made at the second radiograph when periosteal reaction is noted at sites of cortical exit and bone resorption of the fracture line widens the fractures.

In complete bone failure, the diagnosis of fracture of the cannon bone is obvious (Figure 4.148). In all cases, these fractures should be immediately supported and then radiographed to identify the type (simple vs. comminuted) and location in relation to joint surfaces. The limb should not be manipulated excessively during the physical exam because this may lead to penetration of bone fragments through the skin. If treatment is to be considered or further diagnosis is to be obtained, a cast

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Figure 4.147. This oblique radiograph of the tarsus revealed an incomplete proximal metatarsal fracture. This fracture occurred from an accident at a jump and was initially thought to be a proximal suspensory injury. *Source*: Courtesy of Dr. Ty Wallis.

or PVC full-limb splint should be immediately secured to the limb without moving the horse.[13]

Treatment

The selection of treatment of cannon bone fractures depends on the type of fracture (open vs. closed, simple vs. comminuted), location of the fracture (articular vs. nonarticular, proximal vs. distal), animal's age and intended use, presence of wounds, vascular compromise, and economics.

The preferred treatment for most cannon bone fractures is internal fixation with one or two DCPs (Figures 4.148 and 4.149) or LCPs. LCPs are ideal for minimally invasive plate fixation extending the length of the bone.[64] Individual lag screw fixation can also be combined with plates where appropriate. Metacarpophalangeal arthrodesis may be necessary in fractures with distal comminution.[19] Severe fractures may heal with transfixation pins and external fixators or casts. These methods are more successful in foals with rapid healing and low body weight. In adult horses, these severe fractures have a guarded prognosis, but use of external coaptation, plating in the face of open wounds and vascular compromise, or sequential procedures have been successful in rare instances.[14,64,76]

Stall rest should be anticipated for 4 months in foals and 6 months in adults, although hand walking can begin as soon as fracture callus bridges the fracture and other complications of implant failure have not occurred or have arrested. In general, horses that are to be used in athletic competition should have their plates removed in stages, 2–3 months apart. After plate removal, stall rest is continued for an additional 2 months and then exercise is slowly initiated.

Prognosis

The prognosis for successful treatment of third metacarpal and metatarsal bone fractures depends on multiple factors. In general, transverse, slightly oblique, and slightly comminuted fractures in the mid-cannon bone region in foals under 7 months of age have a good to excellent prognosis with internal fixation. Older horses with similar fractures have a more guarded prognosis due to their size and the risk of supporting limb laminitis, but in general have a fair to good prognosis. Older horses with open, comminuted, or articular fractures have a guarded to poor prognosis for recovery. Unfortunately, older horses have a greater risk of comminuted open fractures that involve the nutrient foramen.[84]

METACARPAL/METATARSAL EXOSTOSIS (SPLINTS)

Exostoses of the second and fourth metacarpal/metatarsal bones, or "splints," are a condition most commonly diagnosed in young, immature horses but can be seen in older horses. The condition is thought to be associated with direct trauma causing subperiosteal hemorrhage and periosteal bone proliferation or instability between the second and fourth metacarpal/metatarsal (MC/MT) bones and the third MC/MT bone (Figure 4.150). Inflammation or tearing of the interosseous ligament can occur during training, especially in musculoskeletally immature horses. Splints are most commonly seen in the proximal half of the bones and metacarpal II is most commonly affected. Conformation abnormalities such as offset carpi, improper hoof care, and mineral imbalances may exacerbate the condition.[108]

The second and fourth MC/MT bones are commonly called splint bones. Each is attached intimately to the respective third MC/MT bone by an interosseous ligament. The splint bones articulate with the carpometacarpal joint or tarsometatarsal joint and are exposed to loads on weight-bearing. The interosseous ligament consists of dense fibrous tissue that can tear with the strains applied during independent motion of the splint bones and the cannon bone. Initially, inflammatory periostitis and desmitis develop. Subsequently, new periosteal bone is produced, creating the characteristic profile of a splint (Figure 4.150).

The terminology used to identify the condition is variable. A true splint refers to a sprain or tear of the interosseous ligament. Blind splint refers to an inflammatory process of the interosseous ligament that is difficult to detect on physical examination because the swelling occurs on the axial (inner) side of the splint, between the small metacarpal bone and the SL. Osteolysis between the second and third metacarpal bones may be observed on radiographic examination. Periostitis of the splint bones results from superficial trauma to the periosteum, which, in turn, causes a proliferative periostitis. Although a residual blemish remains, the horse is usually not lame. A knee splint is the enlargement of the proximal portion of the splint bone that may lead to OA within the carpometacarpal joint.

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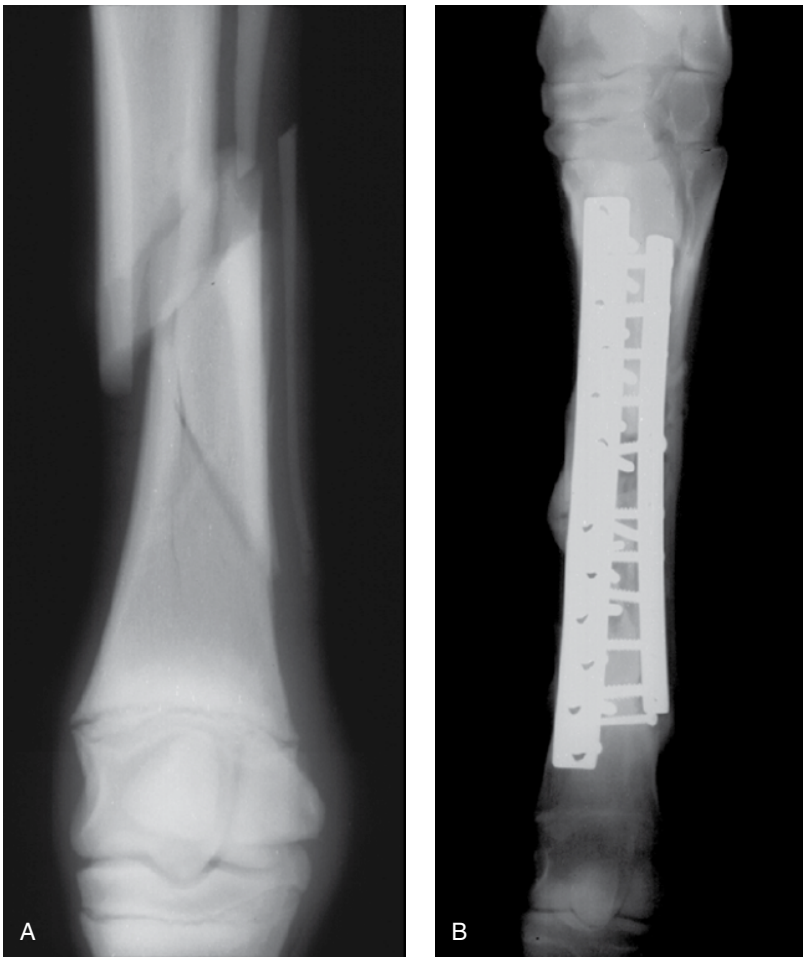


Figure 4.148. (A) Open comminuted mid-diaphyseal fracture of the metatarsus in a 7-month-old weanling. (B) Postoperative view demonstrating fracture repair with two broad dynamic compression plates on the dorsolateral and dorsomedial sides of the metatarsus. Additional screws were placed in lag fashion from outside the plates to secure the butterfly fragments.

Etiology

The enlargement of the splint bones associated with this disease results from proliferation of fibrous tissue and osteoperiostitis. The causes are tearing of the interosseous ligament that binds a small metacarpal bone to the large metacarpal/metatarsal bone, external trauma, and healing of a transverse or longitudinal fracture. If the inflammation associated with the periosteum is sufficient, over time it will result in ossification (proliferative exostosis) of the splint bone. The size of the splint usually depends on the degree of inflammation and the surface area involved. In any case, the splint usually assumes an elongated form, lying parallel to the small metacarpal bone.

The second metacarpal bone is more frequently involved because of the difference in its articulation with the carpus. The second metacarpal bone is entirely articular, and its articulation is flatter than that of the fourth metacarpal bone. *Ex vivo* biomechanical evaluation of the second metacarpal bone has shown that it plays a significant role in torsional stability of the carpus.[121] It is proposed that excessive loading can lead to tearing of the interosseous ligament in the region that is most frequently affected. Metacarpal fusion progresses normally with aging, and 78% of all horses 2 years old and older had two or more sites of fusion. The rate of metacarpal fusion/horse/year appeared to be at least 10 times higher than the rate of painful exostoses causing fusion, indicating that this process occurs subclinically in most horses.[75]

Imbalanced nutrition or overnutrition in young horses has also been implicated in the development of splints.[108] Imbalances and deficiencies in calcium and phosphorus have been associated with an increased incidence of splints. However, no well-documented studies have proven this to be true. In many cases horses that are suspected of having a calcium and phosphorus imbalance also grow rapidly. It may be that their increased weight causes sufficient compressive forces so that splints develop as a result of this, rather than the imbalance. In general, young horses that are poorly conformed, overweight, and overexercised have a greater chance of tearing the interosseous ligament before metacarpal fusion is complete.

Clinical Signs

The condition is most common in young horses undergoing heavy training, but cases occasionally occur in older, mature horses. Heat, pain, and swelling

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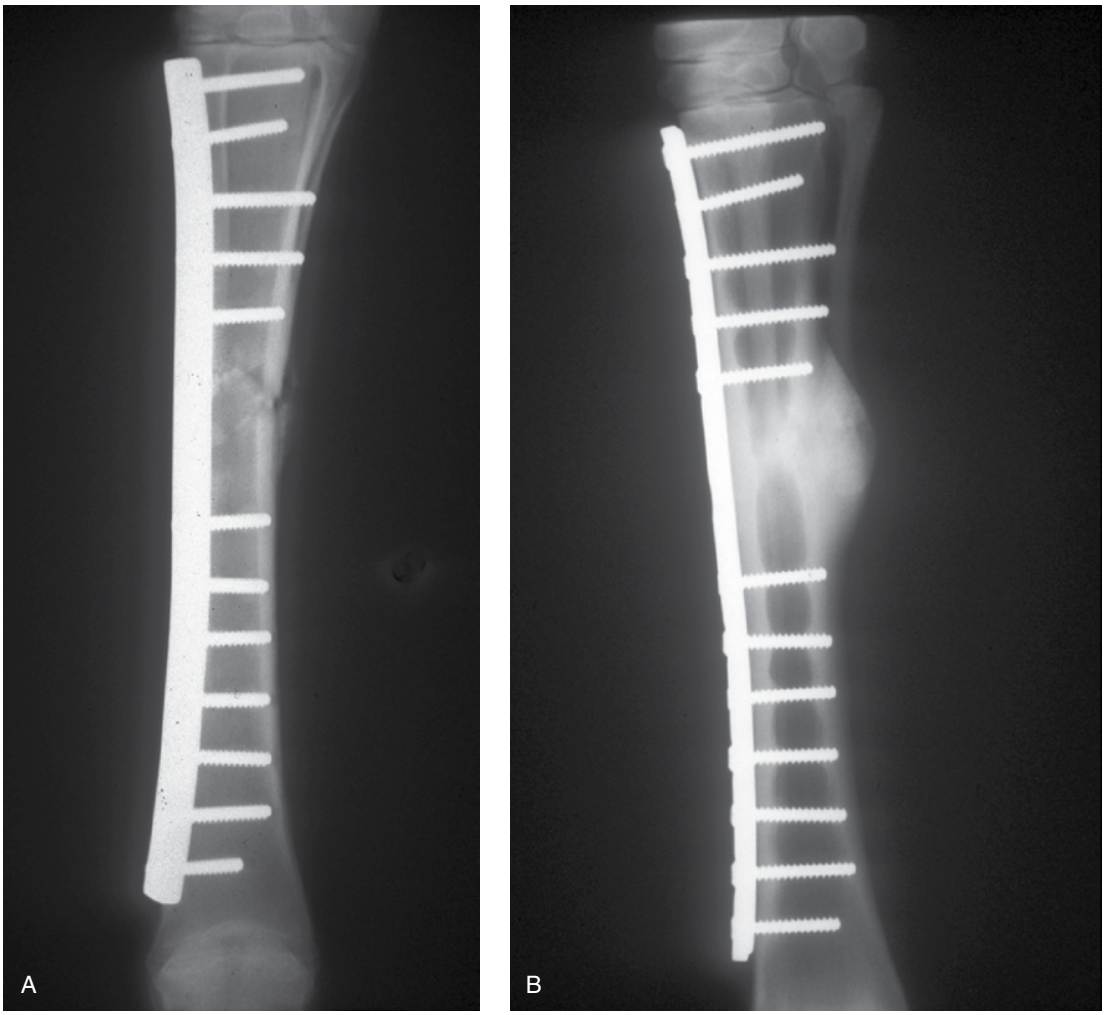


Figure 4.149. This transverse metatarsal fracture in a foal was repaired with a single broad DCP (A) and healed well with a relatively large callus (B). *Source*: Courtesy of Dr. Gary Baxter.

over the affected region are usually present in active splints. Splints most commonly occur about 3 inches below the carpal joint (Figure 4.150). One large swelling or a number of smaller enlargements may occur along the length of the splint bone at its junction with the third MC/MT bone. Horses have variable lameness but generally worsen with work and seem to be worse on hard ground. In mild cases no lameness may be evident at the walk, but lameness is exhibited during the trot. Palpation of the axial aspect of the splint is useful to determine if there is obvious impingement of the SL.

New bone growth that occurs near the carpal joint may cause carpal OA (knee splints). Extensive new bone formation on a splint bone may also encroach on the SL and cause chronic lameness unless it is removed. Growths of this kind can be determined by palpation, radiographs, and ultrasound examination. After the original inflammation subsides, the enlargements usually become smaller but firmer as a result of the ossification. The reduction in swelling is usually the result of resolution of fibrous tissue, rather than a decrease in size of the actual bone formation. In the early stages, the greatest bulk of the swelling is from inflammation, and this normally resolves to a much smaller size. Some cases of splints may not cause lameness.

Diagnosis

The obvious signs lead to a diagnosis when the affected limb is examined carefully. Heat, pain, and swelling over the regions mentioned, plus lameness, are enough to make the diagnosis. Diagnostic analgesia may be required to prove that the splint is a true cause of lameness. The diagnosis should be confirmed with a radiograph (Figure 4.151) as fractures of the splint bone can be confused with splints. Ultrasonographic examination can demonstrate concomitant injury to the suspensory and possible ligament impingement. In some cases, nuclear scintigraphy may be needed to confirm a blind splint. New bone growth resulting from trauma may occur on the third metacarpal or third metatarsal bones, close to the splint bone, and may be mistaken for splints. Palpation and radiographs, however, show that these swellings are dorsal to the junction with the splint bones. This type of new bone growth is most often caused by interference.

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Treatment

Treatment for splints includes anti-inflammatory agents and rest for the acute phase and occasionally surgery in the more chronic stages. Counterirritation is still practiced for the chronic phases but is of unknown significance. Other treatments include extracorporeal shockwave therapy, icing, topical diclofenac liposomal cream (Surpass), acupuncture, and massage.

Inflammation and swelling are the hallmark of this disease in the acute phase. The administration of nonsteroidal anti-inflammatory drugs (NSAIDs) coupled with the application of hypothermia and pressure/support wraps appears to be most beneficial to decrease the heat, pain, and swelling. Hypothermia can be attained with ice or ice/water packs or whirlpool boots. They should be applied for 30 minutes, 2–3 times a day for at least 2–3 days. Some recommend hand massage for 10 minutes after each treatment, after which a support bandage is applied. Affected horses should be confined to a stall for at least 30–45 days, and hand-walking exercise for 15–20 minutes twice a day should be begun after the acute inflammation subsides.[108]



Figure 4.150. Visible enlargement of the medial splint area just distal to the carpus typical of horses with "splints." *Source*: Courtesy of Dr. Gary Baxter.

Intralesional corticosteroid can reduce inflammation and may help prevent excessive bone growth. Corticosteroid therapy should be accompanied by counterpressure bandage. In this case the horse is generally rested longer than 30 days and should not resume training as rapidly as when counterirritation is used. However, the swelling may be considerably less. It is also true that splints will heal without therapy, provided adequate rest is given.[108] If the splint results from interference, splint or shin boots (guards) may help prevent further trauma. If the horse interferes because of improper trimming and shoeing, this should be corrected.

If the proliferative bone is excessive, surgery may be indicated in a very small percentage of the cases (Figure 4.151). Surgery to remove exostoses for medical or cosmetic reasons has resulted in fair to good success. In one study, 15 exostoses removed for cosmetics or lameness resulted in minimal recurrence of the bone proliferation.[5] In a larger study of 95 Standardbreds, the splint bones were amputated to remove excessive bone callus. In horses with proximal splint bone removal in which the proximal portion was stabilized with screws or bone plates, horses were still limited in performance at 12 weeks. Horses in which a subperiosteal removal of the exostosis was performed were sound 12 weeks postoperatively.[141] It is recommended to retain the splint bone lever arm by reflecting the periosteum to prevent

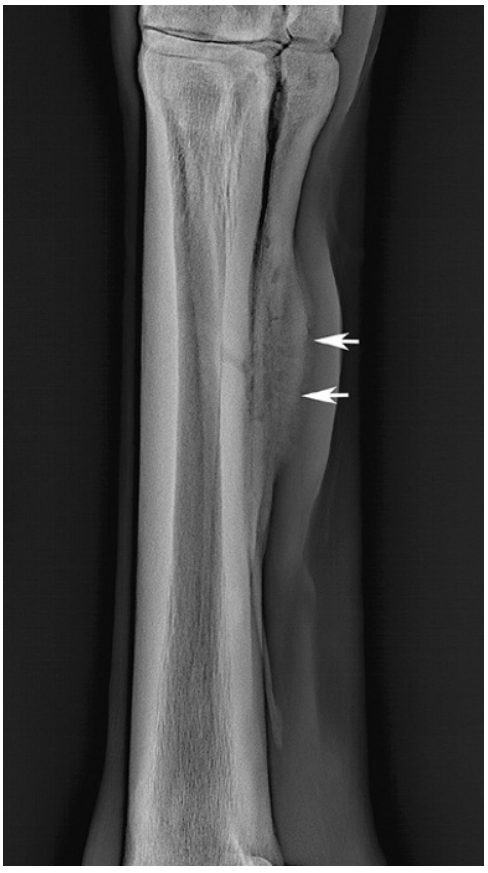


Figure 4.151. This large exostosis of the medial splint (arrows) was contributing to lameness and was removed surgically. Just the exostosis was removed and the underlying splint bone was left intact.

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formation of excessive new bone and associated irritation of the SL. In some cases, it is necessary to surgically remove a bony exostosis that interferes with the action of the SL or the carpal joint or one that is so large that the opposite foot hits it repeatedly. These require care in dissection of the proximal structures, including ligaments of the palmar carpal support. If the bone growth has been caused by trauma from interference, the surgery will not be successful unless corrective shoeing or use of splint boots will stop the interference.

Prognosis

Prognosis is good to excellent for soundness except for those in which the exostosis is large and encroaches on the SL or the carpal joint. Chronic recurring lameness can occur in horses that are not rested long enough, which can be 5–6 months. Surgery to remove the excess bone callus can successfully alleviate lameness, and recurrence does not occur in most cases. Surgery may speed up the return to athletic soundness and improve the cosmetic blemish.

FRACTURES OF THE SMALL METACARPAL AND METATARSAL (SPLINT) BONES

Fractures of the small metacarpal and metatarsal bones (splint bones) can occur anywhere along their length, but they are most commonly located at the distal third.[3,17,31,32,35,136] In most cases fractures located at the distal third are simple fractures (Figure 4.152), in contrast to fractures of the middle and proximal portion, which are often complicated by comminution, osteomyelitis, and bone sequestration (Figure 4.153).[10,31,58,101]

Fractures of the distal part of a small metacarpal or metatarsal bone usually occur in older horses (5–7 years of age) and only rarely occur in horses under 2 years of age.[17] This is thought to occur as a result of decreased pliability in the interosseous ligament and more strenuous training programs in older horses.[17] In contrast, younger horses tend to sustain damage to the interosseous ligament supporting the small metacarpal bones, resulting in the condition referred to as splints. The forelimbs are more frequently involved than the hindlimbs; the fourth metacarpal bone in the left forelimb is affected more commonly than the hindlimb.[17,136] The relationship between SL desmitis, sesamoiditis, and fetlock OA is more than casual. It appears that the enlarged fibrotic SL decreases the absorptive capacity of the fetlock and creates a space-occupying mass that may lead to fracture of the small metacarpal/metatarsal bones followed by further displacement of the fracture fragment.

Fractures of the proximal half of the small metacarpal or metatarsal bone are often comminuted. Open fractures can often be complicated by osteomyelitis with or without sequestrum formation. Fractures of the proximal lateral splint bones are usually a result of direct trauma (Figure 4.153).[10,58,101]



Figure 4.152. Fractures of the distal splint bones such as this rarely heal and are usually removed.



Figure 4.153. Proximal fractures of the fourth metatarsal bone are often comminuted and open and are due to traumatic injuries. *Source*: Courtesy of Dr. Gary Baxter.

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Etiology

Fractures of the distal part of the small metacarpal or metatarsal bone result from external and internal trauma. External trauma can result from a kick from another horse, interference, direct blows from hitting another object, or traumatic wounds. Internal trauma occurs from increased axial compression forces on these bones during races or from pressure from the SL or increased tension from the fascial attachments. It is conjectured that the increased incidence of left fourth metacarpal bone and right second metacarpal bone fractures observed in Thoroughbreds may be the result of increased weight-bearing on the bones when they are racing in a counterclockwise direction. In contrast, the SL and supporting fascia may put these bones under tension sufficient to cause fracture in the hindlimbs.[3,17,31,32,35,136] Because there is an increase in incidence of left second metatarsal bone and right fourth metatarsal bone, which is the tension side of the hindlimb in horses that run counterclockwise, it is logical to assume that tension created by the bowstring effect of the SL or increased tension developed by the internal fascia may lead to fracture.

It is difficult to decide whether the incidence of SL desmitis is a result of distal splint bone fractures that may cause irritation to this structure, or, conversely, that the swollen SL becomes space occupying enough to cause these fractures. Whatever the case, a higher incidence of SL desmitis is noted in the forelimb in association with distal splint bone fractures.[3,17,31,32,35,136]

More complicated fractures of the proximal part of the small metacarpal or metatarsal bones result from direct trauma, either from interference or direct blows to the surface. These fractures are often open initially, which frequently results in osteomyelitis or septic arthritis of the carpometacarpal or tarsometatarsal joint. In some cases there is not a break in the skin initially, but the comminuted fractures become sequestered and result in recurrent draining tracts. Fractures of the second metacarpal bone of racehorses also have been associated with excessive torsional forces that may occur in the starting gate.

Clinical Signs

On visual observation, swelling is usually a prominent feature of proximal splint fractures, but it may or may not be present with distal splint fractures. In general, the degree of swelling associated with the distal splint fracture depends on the acuteness of the fracture. The more acute the fracture, the more the swelling. Associated swelling in the SL may also be observed.

In the acute case, in both instances, horses frequently point their foot. Trotting exercises may or may not cause lameness, but this is totally dependent on the acuteness and type of fracture that has resulted. Circling or fast work may be required to cause sufficient lameness to be observed.

On palpation, heat, pain, and swelling are obvious features of acute fractures, and in some cases draining tracts are also present. The pain and heat decrease with time. However, because callus formation is a frequent sequela to the nonsurgically treated fracture, the fracture site will become enlarged over time (Figure 4.154).



Figure 4.154. This lateral radiograph was taken 3 months after occurrence of a fourth metatarsal fracture. A large callus had developed and there is minimal bridging of the fracture line. *Source*: Courtesy of Dr. Gary Baxter.

In cases in which only mild pain is evidenced with palpation, yet the horse is quite lame, the limiting features of this horse resuming performance may be associated with SL desmitis.[3,17,31,32,35,136] To gain a full appreciation of the involvement of the small metacarpal or metatarsal bone, the limb is flexed so the full extent of this bone can be palpated. A thorough physical examination of the SL should follow.

Diagnosis

A persistent swelling over the affected splint bone, exhibiting heat and pain when pressure is applied, should lead one to suspect a fractured splint bone. Some fractured splint bones closely resemble the disease called splints. Some such fractures heal, but the bony swelling is confused with "splints." Radiographs are necessary for a positive diagnosis of a fractured splint bone and to differentiate between a fracture of the splint bone and splints or soft tissue injury. Direct infiltration of local anesthesia of the fractured small metacarpal or metatarsal bone may be used in cases in which a concomitant SL

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desmitis is present. This helps to define the contribution of the fractured splint to the lameness. This is particularly important in cases in which minimal pain is elicited on deep palpation of the bone, but the horse is quite lame.

Radiographs should be taken in all cases to identify the fracture, its limits, and whether sequestration and osteomyelitis exist in association with a complicated fracture. The proximal fractures may extend toward or into the carpometacarpal or tarsometatarsal joint (Figure 4.155).

Treatment

Small Distal Fractures

Small distal fractures of the splint bones are traditionally treated by removing the distal fragment, but this approach is not universally recommended.[35,136] Up to two-thirds of the length of the splint can be removed without untoward sequelae; however, distal splint fractures can heal spontaneously and are not usually a continued cause of lameness. Nonunion distal splint fractures are frequently not the cause of lameness, and evaluation of the concomitant suspensory desmitis is warranted. In conclusion, surgical removal of these distal splint fractures is often not necessary, but may be performed, particularly for persistent fractures. At least 70% of horses with distal splint fractures have suspensory desmitis, which is generally agreed to be the cause of continued lameness.[136]



Figure 4.155. A dorsomedial to palmarolateral oblique radiograph of the carpus showing an oblique articular fracture of the second metacarpal bone.

Closed Nonarticular and Nondistracted Proximal Comminuted Fractures

Closed nonarticular and nondistracted proximal comminuted fractures of the splint bones may heal successfully with 2–4 months of rest. In hindlimb lateral metatarsal fractures, the horses will be minimally lame and training can continue after the acute inflammation is resolved (approximately 30 days). Surgery may become necessary if:

1. Draining tracts develop.
2. Lameness and pain associated with the fracture are moderate to marked.
3. Exuberant bony callus impinges on the SL.
4. Infection is present.
5. A nonunion or sequestra is developing on follow-up radiographs.
6. A faster recovery and return to performance is desired.

If surgery is required, it should be performed aseptically with the horse placed in lateral or dorsal recumbency.

Two surgical approaches have been successful: removal of the fractured fragments only or removal of the fracture and distal splint bone. If the fracture is in the proximal third, any contiguous piece of bone present should remain to stabilize the proximal component. In closed fractures in which more than two-thirds of the splint bone is to be removed, a small bone plate is recommended to stabilize the remaining proximal fragment.[17,101] If the proximal fragment is not anchored, excessive movement of this fragment may occur and result in interosseous desmitis, degenerative OA of the carpometacarpal or tarsometatarsal joints, or avulsion of the proximal fragment. Screw fixation is less frequently successful. The small metacarpal bones are more predisposed to avulsion after proximal fractures than the metatarsal bones because of the major attachments of the collateral ligaments.

Open Distal and Middle Fractures

Open distal and middle fractures of the splint bone often lead to draining tracts or sequestra, so it is recommended to treat these surgically with open removal of the fracture with or without the distal splint (Figure 4.156). The distal splint bone is left if it seems to provide some stability to the proximal fragment.

Open Proximal Fractures

Open proximal fractures of the splint bones can be more difficult to treat due to draining tracts, sequestra, septic arthritis, and unstable proximal fragments. Several treatment options are available including medical management, fracture debridement, internal fixation,[101] and complete removal if it involves the fourth metatarsal bone[10] (Figure 4.157).

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Figure 4.156. Chronic middle splint fracture with secondary drainage and periosteal reaction that is best treated with surgical debridement and removal of the distal fragment.

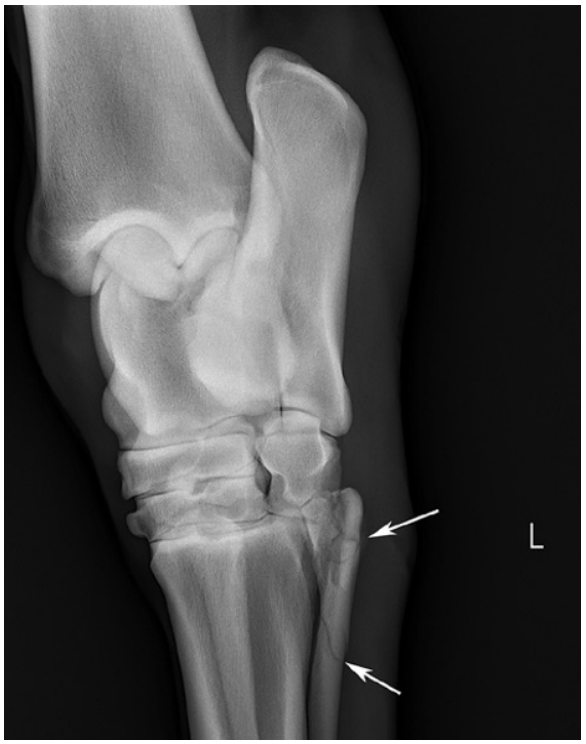


Figure 4.157. Open, articular, comminuted proximal fourth metatarsal fracture (arrow) that was initially debrided and treated conservatively. Infection persisted and the entire fourth metatarsus was removed.

Conservative treatment with local lavage, systemic antimicrobial therapy, and local antimicrobial therapy using regional limb perfusion with or without intra-articular injections can be successful. A recent study found that 11/12 horses with open proximal fractures responded favorably to medical treatment.[63]

Surgical treatment can involve simple debridement of the wound with removal of any loose bone fragments. If there is any concern about intra-articular communication, the joint should be sampled and lavaged during surgery. These procedures can be performed under standing sedation and local anesthesia or can be performed under general anesthesia. A drain can be placed to facilitate drainage postoperatively. Sequestrum formation may occur and require further surgical intervention at a later date. Cancellous bone grafts are useful for treating nonunion fractures in this area.

If more than two-thirds of the splint bone is removed and it appears unstable and active infection is not present, a small bone plate can be secured to the proximal portion of the splint and the cannon bone using 3.5-mm screws. If the fourth metatarsal bone is involved, bone plates may be abandoned for complete removal of the splint bone. If placement of a plate is felt to result in implant infection, the proximal portion can just be left and, if luxation occurs, can be addressed at that time. This may give adequate time for the wound to clean up and decrease the chance of infecting the repair.

For an optimal cosmetic end result, bandages are maintained postoperatively. The horse should be maintained in a stall for at least a total of 6 weeks. Hand-walking exercise can begin at about the second week after surgery, and free exercise can begin after 6 weeks. Training is usually initiated after 2–3 months of rest and is totally dependent on the degree of soft tissue damage associated with the fracture site.

Prognosis

For distal splint bone fractures, the prognosis for return to performance depends on the severity of suspensory desmitis, not on the radiographic healing of the fracture.[17] In a retrospective study of 34 cases of distal splint fractures without SL desmitis, 75.3% of the horses returned to previous levels of performance, and only 50% of the horses with SL desmitis accompanied by the fractured splint resumed to previous levels of performance.[65] In another series with an increased percentage of SL desmitis, only 25% of the horses returned to previous levels of performance.[17]

The prognosis for comminuted closed splint bone fractures is good to excellent with or without surgery, par-

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ticularly if the horse is used for performances other than racing.[17] The prognosis for open comminuted splint bone fractures is good to excellent with surgery if approximately one-third of the proximal splint remains. The prognosis for open comminuted fractures of the proximal splint is more guarded, and about 60% return to performance without lameness. These cases can be complicated by sequestration, osteomyelitis, and infectious arthritis.

ENOSTOSIS-LIKE LESIONS

Enostosis-like lesions (ELLs) have been identified in the third metacarpal and metatarsal bones on nuclear scintigraphic examinations.[1,9] Lesions are identified as areas of focal, moderate to intense increased radiopharmaceutical uptake (IRU) in the medullary cavity (Figure 4.158). Radiography of the affected bone may reveal areas of increased radiodensity in the medullary cavity, often near the nutrient foramen. ELLs in the metacarpal/metatarsal bones may or may not be associated with lameness. Other causes of lameness should be ruled out first. Treatment consists of stall or small paddock turnout for 3 months. Prognosis is generally good for return to athleticism; however, horses with more than one ELL appear to have a decreased prognosis.[1]

SUSPENSORY LIGAMENT DESMITIS

The SL is predominantly a strong, tendinous band containing variable amounts of muscular tissue (2–11%).[39,42] It originates from the palmar carpal ligament and the proximal palmar surface of the third metacarpal bone in the forelimb and descends between the second and fourth metacarpal bones. In the hindlimb, the SL originates primarily from the proximoplantar aspect of the third metatarsal bone. An accessory ligament originating from the plantar fourth tarsal bone also joins the SL. In the distal metacarpus/metatarsus it divides into two branches that insert on the proximal sesamoid bones. The extensor branches pass obliquely dorsad to join the common digital extensor tendon in the proximal phalangeal region. The suspensory apparatus is continued distally as the straight, oblique, cruciate, and short distal sesamoidean ligaments. The SL phylogenetically represents the median interosseous muscle and is also known as the interosseous muscle or proximal (superior) sesamoidean ligament.[36,37,39,42]

The percentage of muscle in each region has been identified; Standardbreds had 40% more muscle in their SL than Thoroughbreds, and it has been suggested that this may be associated with biomechanical differences in gait between the two breeds or genetic factors. *In vitro* strength testing of the suspensory apparatus in training and resting horses suggests that there is an increase of strength with training. The absolute load to failure in a single load-to-failure compression test was higher in horses that had been in racehorse training, and failure in the trained group was usually by fracture of a proximal sesamoid bone. In the untrained group, the SL failed.[22]

Injuries to the SL can be divided into three areas:

1. Lesions restricted to the proximal one-third (proximal suspensory desmitis [PSD])
2. Lesions in the middle one-third, sometimes extending into the proximal third (body lesions)
3. Lesions in the medial and/or lateral branch (branch lesions)

PSD, or inflammation of the origin of the SL, is the most common cause of soft tissue injury to the limbs, comprising approximately 30% of tendon/ligament injuries[143] and approximately 60% of soft tissue injuries that localize to the proximal metacarpus/metatarsus (MC/MT).[22] PSD occurs most commonly in sport horses, such as event horses, jumpers, dressage horses, and racehorses. The hindlimbs are more frequently affected than forelimbs and have a lower success rate of returning to performance after rest and convalescence: 69% for hindlimbs vs. 80% for forelimbs.[22] Overall, lameness associated with the origin of the SL occurs in about 5% of horses (36 of 1,094 horses and 31 of 638 horses) with a forelimb lameness.[36,37,39,42]

Injury can be exclusively within the ligament, involve tearing of the Sharpey fibers at the origin of the SL, or be associated with avulsion fracture(s) of the origin of the SL that involves the proximal MC/MT. High PSD and acute tearing of Sharpey fibers may present with similar signs as those described for blind splints and desmitis of the carpal (distal) check ligament. In chronic cases of tearing of the origin of the SL, radiographic evidence of bone resorption and surrounding sclerosis is often present. With avulsion fracture of the origin of the SL, either the lateral or medial branch may be involved in one or both limbs and will likely be noted on radiographs or seen as a displaced piece of bone on ultrasound (Figure 4.159). This lameness most frequently

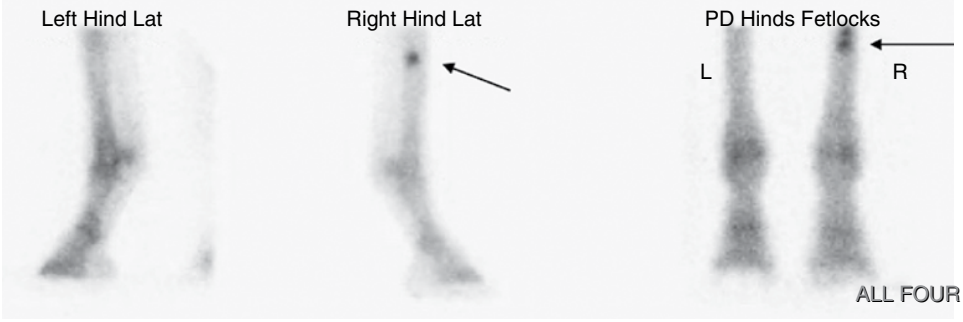


Figure 4.158. Lateral and dorsopalmar nuclear scintigraphic images of the metacarpal regions in a horse with right front lameness. There is a focal, intense area of increased radiopharmaceutical uptake in the proximal medullary cavity of the right front limb (arrows) consistent with an enostosis-like lesion (ELL).

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occurs in Standardbreds, hunters and jumpers, and polo ponies, but other breeds are affected as well.[137]

Injury to the body of the SL is less common and occurs most frequently in racehorses, specifically Standardbred racehorses. The body of the ligament also is usually involved in cases of degenerative suspensory ligament desmitis (DSLD).[56] In general, lesions of the body of the suspensory have a more guarded prognosis for return to full athletic work than proximal injury or injury of the branches of the suspensory.[143]

Injury to the SL branches occurs most commonly in Standardbred racehorses or jumping horses. "Bowing" or "springing" of the distal ends of the splint bones and fracture of the distal splint bones is associated with desmitis of the branches of the suspensory. This finding is considered a result of the enlargement of the branches physically forcing the ends of the splint bones abaxially. This predisposes the splint bones to fail or fracture at their thinnest point. These fractures do not heal spontaneously because the splint bones are under tension. Lameness is often due to the suspensory problem and not the fracture.

Etiology

The SL is primarily made up of dense white fibrous connective tissue. Proximally, the body of the SL separates and attaches into two palmar depressions just distal to the carpometacarpal or tarsometatarsal joints (Figure 4.160). Overloading of the SL may cause trauma to any portion of the ligament, but origin injuries are seen more commonly in sport horses. Hyperextension of the carpus/tarsus in conjunction with severe overextension of the fetlock joint has been proposed to cause proximal lesions. In general, the more severe the trauma, the more severe the ligamentous lesion. Working horses in deep, soft arenas or in eventing where there is excessive rotational movement of the limbs may increase the risk of injuries. Lesions in the body or branches of the suspensory also occur in sport horses worked in soft ground. Therefore, soft tissue injuries, including suspensory injuries, occur more frequently in Europe where horses race and train on turf. Lesions within the branches are also associated with fetlock lameness and suggest that high rotary motion of the fetlock may predispose to suspensory branch injury as may occur in racehorses and animals with dropped fetlock conformation.

Clinical Signs

Most horses with PSD present with a history of intermittent lameness of several days' or week's duration that is exacerbated by resumed exercise. The onset may be acute or insidious. Generally, heat and swelling are palpable on the proximal aspect of the limb only in acute cases. In chronic intermittent cases, physical findings are less obvious or may not be present to assist with a diagnosis. However, slight proximal swelling may be felt on the medial side between the SL and deep digital flexor

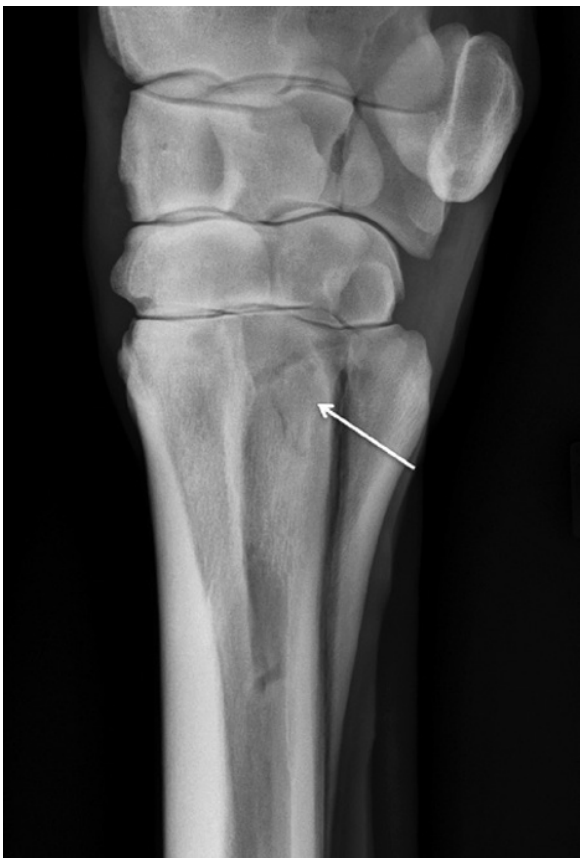


Figure 4.159. A dorsolateral to palmaromedial oblique radiograph of the carpus and proximal metacarpus demonstrating a saucer-shaped avulsion fracture (arrow) of the metacarpus at the origin of the suspensory ligament.

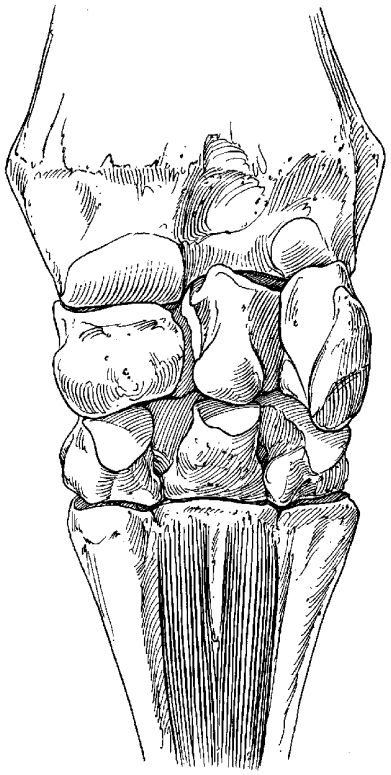


Figure 4.160. The attachments of the suspensory ligament at the proximal palmar surface of the third metacarpal bone. *Source*: Courtesy of Dr. Ted Stashak.

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tendon (DDFT) in some cases.[36–39,41] Additionally, firm digital pressure overlying the proximal SL in these cases usually elicits a nonfatiguable painful response (Figure 4.161). The lameness is generally mild to moderate (1 to 2+ out of 5) at a trot and may be more obvious when the horse is circled at a trot with the affected limb on the outside of the circle. Horses with hindlimb PSD can exhibit moderate to severe hindlimb lameness. Lower limb flexion exacerbates the lameness in 50% of horses with forelimb suspensory problems, and hock flexion exacerbates the lameness in 85% of horses with hindlimb suspensory pain.[39]



Figure 4.161. Location for digital palpation of the origin of the suspensory ligament in the forelimb. The origin is easier to palpate in the forelimb than in the hindlimb.

Horses with tearing of the Sharpey fibers at the origin of the SL or avulsion fractures most frequently present with a history of acute onset of moderate to severe lameness. Horses sustaining a fracture of the origin of the SL often have attained racing speeds in their workouts. Digital pressure at the origin of the suspensory may induce a painful response and exacerbate the lameness (Figure 4.161).

Horses with injury to the body or branches of the SL usually have visible and palpable swelling and signs of acute inflammation at the site of injury. In more insidious onset cases, the enlarged ligament has less inflammation and is firmer on palpation. Pain on digital pressure and a positive response to fetlock flexion are common in affected horses.

Diagnostic Analgesia

Proximal suspensory injury can be challenging to accurately diagnosis and requires a combination of diagnostic analgesia and diagnostic imaging. Diagnostic ultrasonography, nuclear scintigraphy, CT, and MRI can all be used following lameness evaluation and diagnostic analgesia to confirm and characterize SL pathology (Figure 4.162).[22,41,44,72,143]

Diagnostic analgesia is necessary to localize the lameness to the proximal suspensory. There are many different approaches to diagnostic analgesia of the proximal suspensory. The authors prefer anesthesia of the lateral palmar nerve or lateral plantar nerve. This technique may be more specific and also requires less volume of

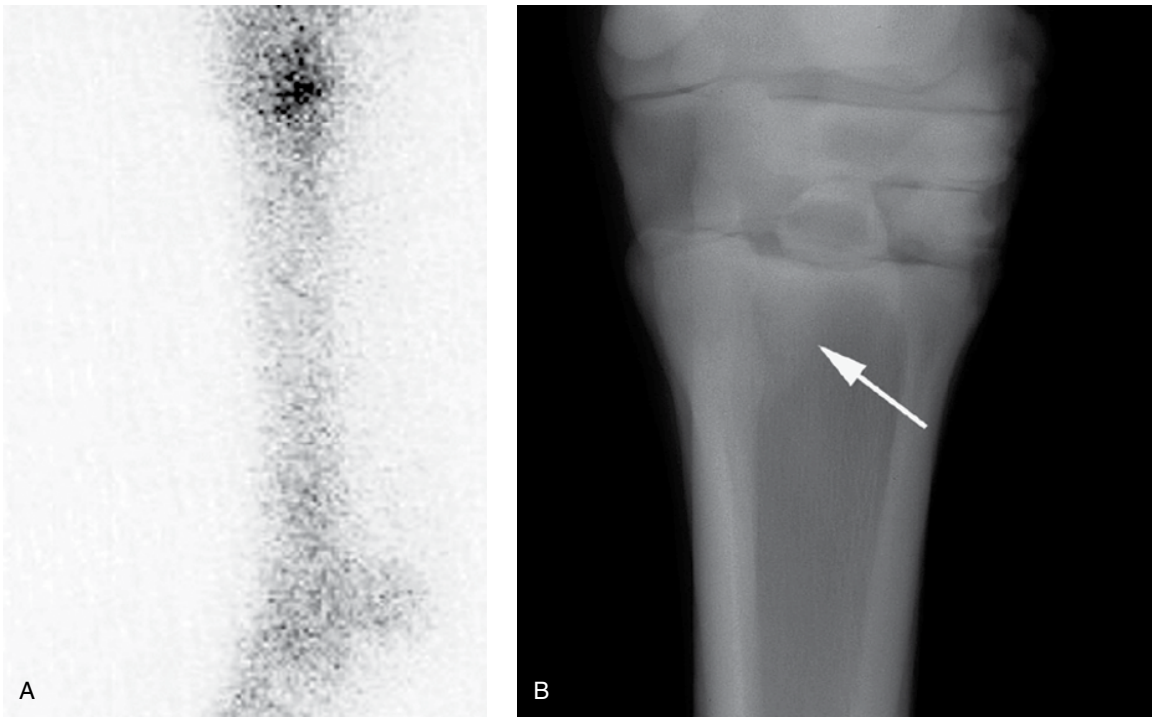


Figure 4.162. Nuclear scintigraphy can identify bone injury at the origin of the suspensory ligament (A). Radiographic lesions such as sclerosis (arrow) can be subtle (B) and usually only identified on the craniocaudal view due to the overlap with the splint bones on the lateral view.

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local anesthesia; therefore, the risk of diffusion is lessened, and interference with subsequent ultrasonography is decreased. Alternatively, direct infiltration of the origin of the SL or a high four-point block with anesthesia of the palmar/plantar nerves and palmar/plantar metacarpal/metatarsal nerves can be performed.

In the forelimb, perineural anesthesia of the lateral palmar nerve usually improves forelimb lameness and can be performed just below the accessory carpal bone from the palmar or lateral aspect of the limb or on the medial aspect of the accessory carpal bone. In a minority of horses, the ulnar nerve needs to be blocked to completely abolish the lameness. Diffusion of local anesthesia into the middle carpal joint following anesthesia of the proximal suspensory region has been reported.[89] Diffusion occurs most commonly with anesthesia of the medial and lateral palmar nerves with needle insertion just axial to the splint bones and can be minimized by anesthesia of the lateral palmar nerve performed just below the accessory carpal bone. Additionally, anesthesia of the lateral palmar nerve can lead to penetration of the carpal sheath.[46] Inadvertent anesthesia of the middle carpal joint and carpal sheath needs to be considered during the diagnostic process of a horse with suspected PSD. It should also be noted that low four-point block may cause partial improvement in lameness associated with PSD due to proximal diffusion of local anesthesia.[88]

In the hindlimb, the desensitization of the deep branch of the lateral plantar nerve is the preferred nerve block. This block can be performed just distal to the head of the lateral splint bone with the needle directed axial to the splint (Figures 2.156 and 2.157). As in the forelimb, diffusion of local anesthesia or direct penetration into the tarsometatarsal joint or tarsal sheath can occur, especially when the direct infiltration of the origin of the SL or both the medial and lateral plantar nerves is blocked. It is often worthwhile to perform intra-articular anesthesia of the tarsometatarsal joint on a separate occasion to confirm diagnosis of PSD.

In the forelimbs, desmitis of the distal accessory ligament of the deep digital flexor tendon (ALDDFT) must also be considered.[22] In one study, lameness due to lesions created in the flexor tendons or proximal SL with collagenase injection was not reliably abolished until the ulnar block was added.[69] Similar results were obtained with hindlimb blocks.[61,70]

Diagnostic Imaging

Ultrasonographic examination, radiographic examination, nuclear scintigraphy, MRI, and to some degree, CT, particularly contrast CT, are the current methods for determining the damage within the origin of the SL.[22,36,43,72,143,148] Ultrasonography is by far the most commonly used modality as it is accessible and relatively simple to perform. That being said, lesions in the proximal SL are often subtle and difficult to detect, especially considering interference by both splint bones (particularly in the hindlimb) and the variability in hyperchogenicity due to differences in the amount of muscle present. The entire SL should be imaged in transverse and longitudinal planes. Ultrasonographic abnormalities of the SL that are often identified include enlargement of the ligament (linear width and circumference), poor definition of margins, central or peripheral areas of hypoechogenicity, diffuse reduction in echogenicity, hyperechogenic foci, and irregularity of the plantar cortex of the third metatarsal bone (enthesophyte formation or avulsion fracture).[36,37] Normally, the SL is the most echodense structure in the palmar or plantar MC/MT.[37,143] Comparison to the contralateral limb should be performed, but the disease condition can be bilateral in 18% of horses,[37] so standardized normal comparisons should also be used. Repeat ultrasonographic exams can be performed every 60 days to evaluate response to treatment. Although ultrasonography is the most commonly used diagnostic tool, lack of ultrasonographic abnormalities in the proximal SL does not necessarily rule out a problem. MRI of horses should be considered in horses with lameness localized to the proximal SL with diagnostic analgesia but without ultrasonographic abnormalities. Increased signal intensity and enlargement or alteration in shape of the origin have been seen on MRI in horses with PSD, despite normal ultrasonographic exams.[22,38]

Radiographic examination may be negative for acute high SL desmitis and tearing of Sharpey fibers. However, bone sclerosis of the proximal plantar metatarsal bone, alteration of the plantar subcortical trabecular pattern, and enthesophyte formation were noted in 23 of 47 horses diagnosed with hindlimb PSD (Figure 4.162).[39] Additionally, radiography can be used to rule out causes of lameness attributable to the carpal or tarsal joints. CT can be used to better define this proliferation.[72] Early bone resorption can occur at the origin of the heads of the SL. Avulsion fracture associated with the origin of the SL can also be observed on radiographic examination.

Nuclear scintigraphy can diagnose cases with bone involvement or remodeling, but in horses with confirmed origin of suspensory lesions has been shown to be less sensitive than expected and cannot reliably diagnose the condition.[41] CT can identify enthesophytes that surround the ligament that are not well delineated on radiographs.

Desmitis of the body and branches of the suspensory can be diagnosed with a combination of ultrasonography and radiography. Radiographs should be included to identify the concomitant bone abnormalities such as distal splint bone fractures and proximal sesamoid bone fractures. Ultrasonography commonly identifies enlargement and hypoechoic areas within the body or branches of the ligament.

Treatment

Treatment for horses with a structural abnormality on ultrasound and/or radiography consists of a convalescence program of confinement and slow return to exercise. Immediate medical management, including anti-inflammatories, hydrotherapy, and bandaging, is recommended to reduce any swelling that may occur and to support the fetlock. If a lesion is present on ultrasound, initially, stall rest for 2 months followed by repeat ultrasound is recommended. Hand walking for 15 minutes twice daily for the first 4 weeks and for 20 minutes twice daily for the last 4 weeks can be performed if the horse is sound at the walk, the origin lesion is not greater than 50% of the cross-sectional area

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(CSA), and straight hindlimb conformation is not present in a hindlimb injury. Trotting can begin after 8 weeks if the lesion is reasonably improved on ultrasound and the horse is sound at the trot in hand. A slow and controlled (saddle) or rein lunging exercise program can continue until 16 weeks, when another ultrasound is performed. If soundness persists and the lesion appears healed on the 16-week ultrasound exams, the horse's exercise can increase. Total healing time is 8 months and return to full competitive performance may not be possible for 1 year.

Recurrence of injury is greater in horses that have been inadequately rested and for abnormalities within the hindlimbs. Balancing the foot, shoeing with egg-bar shoes, and using sport fetlock support bandages can improve the flexor surface support. A more prolonged convalescent period is required if pain returns after exercise has begun. Use of oral glycosaminoglycans, systemic polysulfated glycosaminoglycans, and systemic hyaluronic acid may have benefit, although unproven. Use of internal blisters, intralesional steroids, and mineralizing agents is contraindicated.

Regenerative medicine therapies consisting of ultrasound-guided injection of bone marrow aspirate concentrate (BMAC), autologous mesenchymal stem cells (MSCs), or autologous platelet concentrates have shown efficacy.[4,137] In one study of suspensory desmitis of the body, autologous platelet concentrate injection in Standardbred racehorses resulted in all nine cases returning to racing with good performance, equal to peers in money earned.[137] Surgical splitting,[60] ultrasound-guided desmotomy and fasciotomy,[61] and shockwave therapy[62,77] have also been described for PSD injury that has significant lesions on ultrasound or that does not respond as expected to confinement and medical management. Improvement in most horses occurs over 6 months of treatment. Neurectomy of the deep branch of the lateral plantar nerve with concurrent fasciotomy has a reported success of 77.8% of horses with PSD alone. However, in this study, all horses with concomitant straight hock conformation and/or hyperextension of the fetlock remained lame and horses with concurrent OA of the distal tarsal joints were reported to have a significantly worse prognosis following surgery (44.2%).[41] However, the surgery may be particularly useful in horses with compressive damage to the lateral plantar nerve, which can cause persistent lameness.[135]

In suspensory desmitis of the branches, treatment of concomitant disease of the fetlock, support bandaging, and a modified training program permits many horses to continue performing. Intralesional orthobiologic therapies described previously are also frequently performed. Surgical removal of the distal splint fractures is controversial and may not address the primary cause of lameness.

Horses with a dropped fetlock conformation have lost suspensory support to the limb and are at high risk of re-injury or complete SL breakdown. Use of support bandages and heel extension shoes, such as an egg bar, are recommended. Many of these horses are retired. In addition, horses with DSLD often have a bilaterally dropped fetlock conformation as part of the presenting clinical picture, suggesting severe damage to the SL.

Prognosis

The prognosis for acute forelimb PSD is good (greater than 80%) for return to full work in sport horses with forelimb PSD following 3–6 months of rest and controlled exercise.[22,100] For hindlimb lameness, the prognosis is significantly worse with only 14%–69% of horses returned to full athletic function without detectable lameness.[22,38] The wide variety of outcome with hindlimb PSD may be related to the greater range and tendency for more severe lesions, concomitant neuropathy and compartment syndrome, and conformational issues that predispose the SL to greater stresses, such as dropped fetlocks and straight hocks. Recently, it was reported that neurectomy of deep branch of the lateral plantar nerve resulted in an 80% return to performance for hindlimb PSD.[135] Osteostixis of the proximal metatarsus at the origin of the PSD in hindlimbs has also been reported.[61] The presence of upright hindlimb (straight hock) conformation is overrepresented in horses with hindlimb suspensory injury and may predispose the ligament to injury and recurrence—hence, the poor success rate for permanent resolution of lameness. Another proposed reason for the poor prognosis associated with hindlimb PSD is the development of a "compartmental syndrome" resulting from the limited space available between the prominent splint bones into which the inflamed SL can expand.[39]

Recurrence of PSD also relates to use. In one study, dressage and show jumping horses had the highest recurrence rate of PSD, 37% and 46%, respectively, compared with racehorses (27%), show hunters (19%), and field hunters (18%).[26] In addition, the larger the SL lesion, the greater the chance of recurrence. Generally, the recurrence of PSD after 1 year following successful treatment is low, but when it does occur, the prognosis is guarded for return to performance. The prognosis is also reduced when PSD is associated with other lesions such as tendinitis of the SDFT and DDFT or desmitis of the superior check ligament.[42]

The prognosis for avulsion fracture associated with the origin of the SL appears good for uneventful recovery to full work after 3–6 months of rest and controlled exercise. Horses with evidence of exostoses developing around the splint bones, but in association with PSD, may benefit from bone debridement after CT.[72]

Degenerative Suspensory Ligament Desmitis (DSLD)

DSLD is a debilitating disorder thought to be limited to the SL of Peruvian Pasos, Peruvian Paso crosses, Arabians, American Saddlebreds, Quarter horses, Thoroughbreds, and some European breeds. It primarily affects the body and proximal aspects of the ligament and is often bilateral. Both forelimbs or both hindlimbs can be involved, and frequently the disorder leads to persistent incurable lameness requiring euthanasia. Many horses have excessive fetlock extension (dropping) on presentation, and horses with hindlimb involvement often have the combination of straight hocks and fetlock hyperextension (Figure 4.163). Affected horses often have palpable enlargement and pain of the SL. The diagnosis is usually made based on patient signalment and history, clinical examination, and ultrasonographic abnormalities within the affected SL. Treatment is

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Figure 4.163. Severe dropping of the fetlock together with a straight hock conformation is typical of horses with DSLD. *Source:* Courtesy of Dr. Gary Baxter.

empirical and supportive but often not effective in altering progression of the disease. Horses often remain lame or worsen, and the prognosis is poor for recovery.

The etiology of DSLD is unknown, but the disease tends to run in families, suggesting hereditary influences. A recent study suggested that the disease may be due to excessive accumulation of proteoglycans within the SL and other tissues in affected horses.[56] Histopathologic examination of horses affected with DSLD revealed increased accumulation of proteoglycans within the SL, SDFT and DDFT, patellar and nuchal ligaments, cardiovascular system, and sclerae, compared with control horses. This study indicated that the abnormalities associated with DSLD are not limited to the SL but affect many other tissues and organs with significant connective tissue. The authors suggested that DSLD is actually a systemic disorder of proteoglycan accumulation and propose the term equine systemic proteoglycan accumulation (ESPA) as a more appropriate name for the condition.[56] It should be noted that 22/28 horses in this study were Peruvian Pasos; therefore, extrapolation to other breeds must be considered with caution. It has also been proposed that DSLD in non-Peruvian Paso breeds is due to progressive exercise-induced degeneration of the SL and not associated with a proteoglycan disorder. Regardless, the disease is almost always progressive and the prognosis for athleticism is poor.

SUPERFICIAL DIGITAL FLEXOR (SDF) TENDINITIS (BOWED TENDON)

Tendinitis of the SDFT is a common injury in horses working at high speeds including Thoroughbred and Standardbred racehorses. Thoroughbred racehorses have a reported incidence of 7%–43%.[34,116] Superficial digital flexor (SDF) tendinitis can also occur in other performance horses including show jumpers, event horses, dressage horses, and Quarter Horses. Unlike SL injury, SDFT injury is almost exclusively a forelimb problem. Lesions can range from peritendinous inflammation and pain without structural damage to complete tendon rupture. Tendinitis usually results in a high degree of morbidity with prolonged periods out of work.

Classical SDF tendinitis is localized to the mid-metacarpal region in the forelimb and appears as a convex "bow" to the visual profile of the metacarpus on the side view, hence the term "bowed tendon" (Figure 4.164). Anatomically, the SDFT is the most superficial of the flexor tendons/SL, and therefore enlargement is readily visible and palpable.[80] In normal horses, the SDFT develops in size and tensile strength with training. The SDFT musculotendinous unit originates on the caudal humerus and extends to a bivalved attachment on the palmar/plantar eminences of the second phalanx. Therefore, complete disruption of the SDFT can be associated with dorsal subluxation of the distal first phalanx as well as dropping of the fetlock. In the author's experience, rupture of the SDFT is uncommon, but occurs most frequently in eventing horses and geriatric horses that run when out in pasture.

Less common locations for injuries to the SDFT are the distal MC/MT, the branches of the SDFT in the pastern region, and the proximal SDFT at the level of carpus and above, including lesions at the musculotendinous junction. Lesions in the distal cannon bone are often referred to as "low bows" and can be associated with digital sheath tenosynovitis or constriction of the annular ligament (Figure 4.165). Tendinitis of the branches of the SDFT can be difficult to diagnose because this is not a common location of injury and the classical swelling seen at other locations may not be present.

Damage to the SDFT in its proximal aspect is a syndrome that more commonly affects older (>15 years) nonracehorses. In one study, the mean age of affected horses was 18 years (range of 11–23 years), and Quarter horses (9 of 12 horses) were the predominant breed.[24] Most horses have moderate to severe lameness and positive reactions to carpal flexion, and the majority required an ulnar nerve block to alleviate the lameness (9 of 12 horses). Swelling of the proximal SDFT can be subtle and easy to miss. The prognosis for return to soundness in these aged performance horses appears to be poor.[24]

Etiology

As the forelimb contacts the ground at the gallop, the fetlock is hyperextended, and the flexor tendons are placed under very high tensile loads.[54] Tendons are elastic and can store kinetic energy upon loading that assist with propulsion and support of the large body loads in a "bungee cord" effect. In the Thoroughbred racehorse, the tendon strain (elongation) can be as

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Figure 4.164. Classic appearance of mild (A) and more severe (B) SDF tendinitis of the left forelimb. Note the convex palmar surface to the middle region of the metacarpus (arrows).



Figure 4.165. A "low bow" of the SDFT as illustrated in this image can be associated with concurrent digital tendon sheath tenosynovitis. *Source:* Courtesy of Dr. Gary Baxter.

great as 16%, putting this structure close to its physiologic limit during maximal exercise.[142] The high demands placed on the SDFT put it at risk of overstrain injury. This can occur as a single overloading but is more commonly due to repetitive trauma leading to microdamage of the collagen structure that contributes to final failure of fibrils. An age-related effect on SDF tendonitis has been proposed. The SDFT matures early (by ~2 years) with increased strength in response to exercise, after which the tendons develop patchy areas of acellularity, matrix fibrillation, and an overall limited ability to adapt and repair.[98,99] Increasing age and exercise demands can lead to subclinical tendonitis due to accumulation of microtrauma without adequate repair. Full clinical tendonitis can result if biomechanical forces are sufficient.

Clinical Signs and Diagnosis

Diagnostic advances, particularly with ultrasonography and MRI, have greatly enhanced our ability to diagnose and define the degree and extent of damage in SDFT injuries. However, a careful clinical examination can identify early focal swelling and tenderness well before a detectable lameness occurs. Intervention at this early phase can prevent structural damage to the tendon. Excellent husbandry and observant animal caretakers are keys to this early recognition.

In more significant SDFT injuries, a more diffuse enlargement, tendon thickening, and local heat with lameness after exercise occur. Severe tendon injuries are recognized clinically as horses that do not want to put the hoof flat on the ground, thereby putting tension on the flexor surface of the limb. The acute phase of SDF tendinitis can last 2–7 days, the subacute phase 1–3 weeks. Aggressive treatment of the inflammation and

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swelling is paramount to limiting the fibrosis and tendon disfiguration that can be permanent.

Chronic SDF tendinitis is manifested by fibrosis and firm swelling on the palmar or plantar (flexor) surface of the limb (Figure 4.164B). Chronic SDF tendinitis may not have heat or edema associated with the condition, yet lameness may be present. The use of diagnostic ultrasonography enables initial and reasonably accurate determination of the extent and location of the lesions.

A complete ultrasound examination extends from proximal to metacarpus/metatarsus to the heel bulbs and determines the CSA of the SDFT and the lesions at multiple locations from proximal to distal. Comparison to the contralateral side may provide a comparison and possibly identify a subclinical condition. Longitudinal scans permit the validation of structural lesions and the assessment of fiber alignment. Hypoechogenicity can be caused by fibrillar disruption and/or inflammatory processes (hemorrhage, edema, and cellular infiltration in the early stages).[107,116] Ultrasound images correlate to histologic lesions. If an SDFT injury is acute/subacute and has clinical signs of inflammation, a repeat ultrasound within 2 weeks is indicated after the inflammation has subsided. Hypoechoic areas within the SDFT can be edema fluid or hemorrhage early on after the injury and may not necessarily be fiber disruption or structural damage (Figure 4.166). If the acute signs of injury have resolved and hypoechoic areas within the SDFT persist, they likely represent fiber disruption (Figure 4.167). MRI has demonstrated an advantage over ultrasound for distinguishing fibrosis from normal tendon in chronic tendinitis.[67]

Treatment

Immediate medical treatment of SDF tendinitis is recommended to reduce or eliminate the acute swelling and inflammation as rapidly as possible to prevent fiber damage or early fibrous tissue formation. Routine physical therapy such as icing, cryotherapy, pressure wraps,

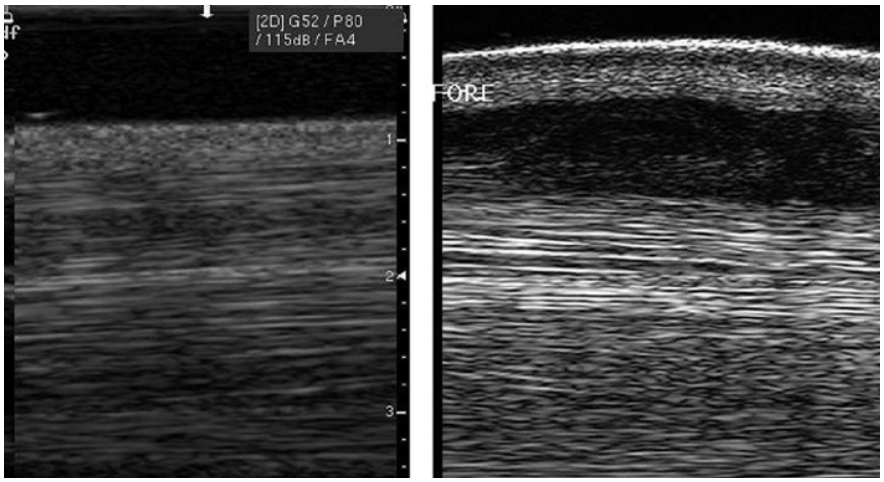


Figure 4.166. Ultrasound longitudinal sections through the midportion of a normal SDFT (left) and SDFT with acute tendinitis. Note the hypoechoic area (black) in the middle of the SDFT that usually corresponds to edema or hemorrhage in acute cases.

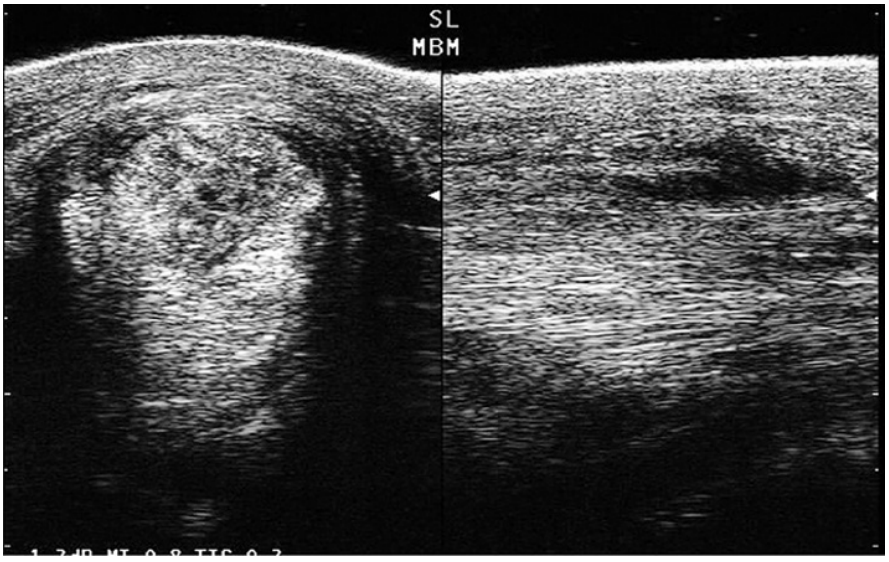


Figure 4.167. Ultrasound cross section (left) and longitudinal section (right) through an affected area of chronic SDF tendinitis. Note the hypoechoic areas (black) that usually correspond to fiber disruption in chronic cases.

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and topical hyperosmotic sweats or diclofenac liposomal cream, along with systemic NSAIDs, is instituted along with rest until the ultrasound can be performed. In acute, severe injury such as rupture, a soft cast, resin cast, or Kimzey Leg Saver splint (Kimzey Welding Works, Woodland, CA) may be indicated until radiographs or ultrasound can occur. The application of topical steroids ± dimethyl sulfoxide is controversial, but can be very effective at eliminating signs of inflammation. Systemic steroids are generally not recommended because they may impair the early healing response. Local injections in or around the tendon are usually considered after the results of the ultrasound examination at 1–2 weeks, when most of the fluid accumulations (edema, blood) have resolved and the extent of fiber damage can be accurately determined.

If ultrasound examination is normal and the acute inflammation surrounding the tendon resolves within 1–2 weeks, then hand walking followed by a return to exercise in 30 days or so may be effective. A cautionary re-ultrasound prior to work is indicated to be certain the original ultrasound is still normal, or further damage did not occur upon retraining. If areas of hypoechogenicity are noted within the tendon at ultrasound, then additional treatment and rest are indicated. Lesions should be quantified and horses rested at least until follow-up ultrasounds at 60-day intervals.[82]

Many medical treatments for SDF tendinitis are used in practice. Systemic hyaluronan and polysulfated glycosaminoglycans may be used to reduce adhesions and promote extracellular matrix quality, even though they have lower levels of evidence for success; they also carry a low risk of complications.[40,49–51,54,59,60,67,71,80,82] Intra- or perilesional injection of these same drugs is used less often, and controlled experimental studies have been unable to confirm a benefit.

Several medical therapies have been developed specifically for tendon or ligament injuries. Injectable autologous biologic therapies such as bone marrow, BMAC, MSCs, acellular collagen, platelet-rich plasma (PRP), and concentrated plasma are commonly used to treat SDF tendonitis (Figures 4.168 and 4.169). Acute, subacute, or chronic tendinitis with a persistent centralized hypoechoic area can be injected intralesionally.[4] In the acute and subacute phase, patient-side products like PRP, BMAC, and allogeneic MSCs are used due to their availability. Such therapies are likely beneficial even during the acute phase as they help modulate the inflammatory process.

At this time, bone marrow or fat can be harvested for culture expansion of MSCs, which requires 2–4 weeks.[71,124] Several experimental and clinical studies have described the treatment of SDF tendonitis with adipose-derived stem cells in PRP,[30] adipose-derived stem cells,[91] and bone marrow-derived MSCs,[51,127] with improved tendon organization and decreased re-injury rates in racehorses reported. Use of ultrasound guidance for injection facilitates the injection into the lesion (Figure 4.169). Intravenous or intra-arterial regional limb perfusion using stem cells has been used clinically in some cases of extensive SDF tendinitis due to the difficulty of adequately injecting the entire lesion. Although some studies have evaluated the localization of stem cells following regional perfusion,[128,131] studies evaluating the possible benefit of this technique in horses with pathology have not been published. Although these materials may aid tendon healing, proper rehabilitation and a convalescent exercise program remain paramount to success with any treatment.



Figure 4.168. Gravitational device for the isolation of platelet-rich plasma and platelet-poor plasma.

Electromagnetic and electroshock stimulations have been proposed as treatments for tendon injury. Studies suggest that these techniques should be used with caution because they can cause physical damage to the tissue.[15] Similarly, low-level laser therapy (also called soft or cold laser therapy) has not substantiated clinical claims of benefit.

Surgical Management of SDF Tendinitis

Two surgical procedures are described for treatment of SDF tendinitis: a tendon splitting procedure and proximal accessory ligament transection on the affected forelimb (frequently both, including the unaffected limb). The surgical technique of longitudinal tendon splitting was advocated to decompress the lesion (relieve the hematoma), provide a vascularization channel to central lesions,[2,140] and consequently promote healing, particularly of core lesions in injured SDFT. In experimental and clinical studies, tendon splitting decreased the core lesion size within 8 weeks.[2,60] Importantly, tendon fibers are separated in the longitudinal plane and not transected, with this procedure, or more fiber damage could occur.

Transection of the accessory ligament (superior check) of the SDFT has been reported to drop the fetlock angle

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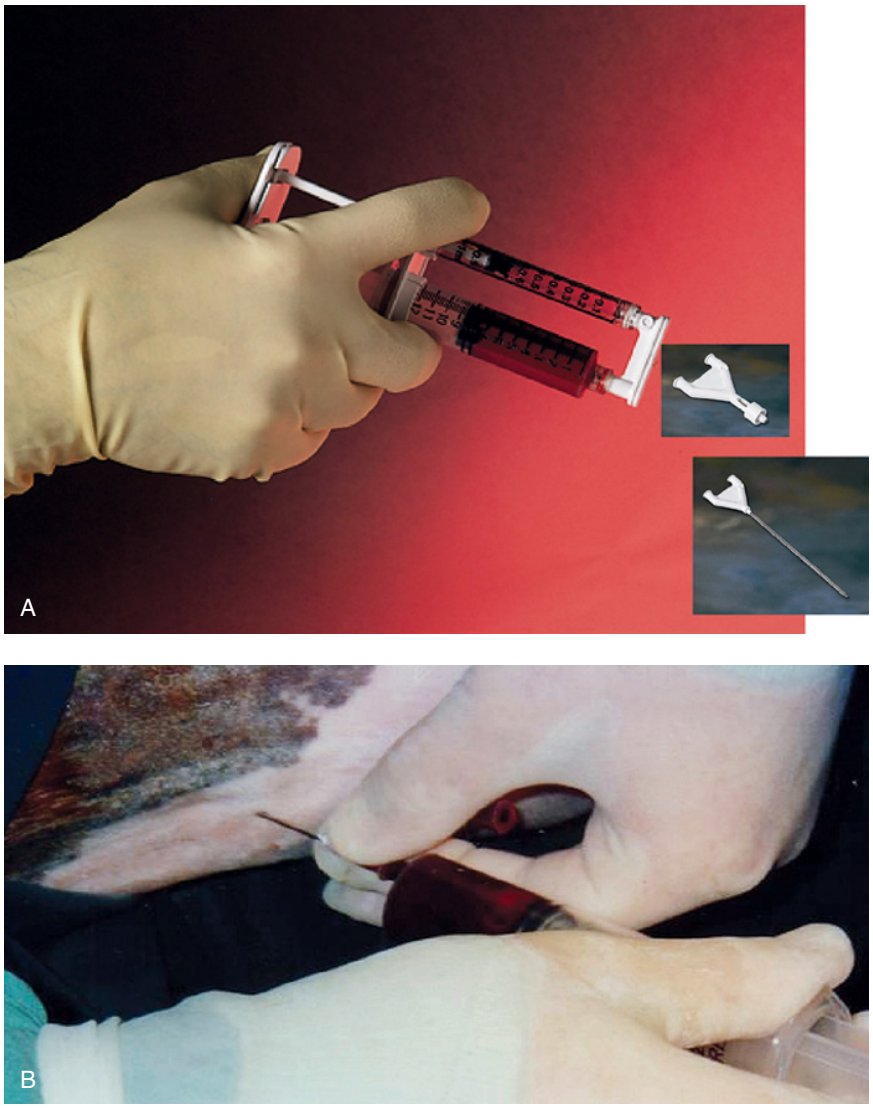


Figure 4.169. (A) A dual port injection syringe permits simultaneous injection of platelet-rich plasma (PRP) (red) and thrombin (clear) into SDFT core lesions. The thrombin serves to clot the PRP. (B) Injection of PRP into an SDFT after ultrasound identification of the site of the lesion. Small aliquots (less than 0.5 mL) of PRP can be injected 0.5 cm apart throughout the length of the lesion.

(increase hyperextension) in normal horses[123] and has been used as a successful surgical therapy for flexural deformity of the fetlock for many decades. Transection of this ligament releases the SDFT, and a gap forms at the transection site, altering the biomechanical forces on the SDFT and reducing peak loads on the SDFT at full weight-bearing.[20,123] However, an *in vitro* study has also shown that SDFT strain is increased following transection of the ALSDFT, which would be suboptimal. In a retrospective study in 62 Thoroughbreds, 92% of the horses were able to train and race after proximal check ligament surgery, and 66% started at least five races.[21] Another study in Standardbred racehorses reported that 92% of horses raced at least once after surgery and 87% raced at least five races, with most having had a bilateral procedure.[59] The evidence for efficacy of proximal check ligament surgery in treating horses with SDF tendinitis is somewhat controversial though, as a trend toward re-injury and a 5.5-fold increase in SL desmitis following surgery have been reported.[49]

Prognosis

Incidence of re-injury is an outcome measure of successful management of SDF tendinitis. Published reports of re-injury in horses treated conservatively with rest alone are 48%[116] and 56%.[124,125] Medical therapies such as hyaluronan, PSGAGs, etc. did not show a difference in re-injury rate.[40] Stem cell therapy reports a significantly reduced re-injury rate of 18%.[51,124] Surgical transection of the accessory ligament of the SDFT reports a reduced re-injury rate of 25%.[59]

Rehabilitation

The value of a carefully controlled rehabilitation program combined with regular ultrasound examination has been documented as providing the best chance for an equine athlete to return to full performance following SDFT (or SL or biceps tendon) injury.[107] Most structural lesions need a minimum of 6 months of restricted

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athletic activity to allow for the majority of healing to occur, and clinical signs of lameness should be absent after a short period of rest and before any forced exercise. Tendon rehabilitation protocols are tailored according to whether the structural damage is graded as mild, moderate, or severe. In mild structural injury, the SDF has a tendon CSA that is less than 20% greater than normal SDFT, a core lesion of 15% or less of the total tendon CSA, or a longitudinal lesion of 20% or less of the total length of the tendon. A moderate structural injury has a 20%–35% greater CSA, a core lesion of 15%–30% of the total tendon CSA, or a longitudinal lesion of 20–35% of the length of the tendon. A severe structural injury has a 35% greater CSA than normal, a core lesion greater than 30% of the total tendon CSA, and a longitudinal lesion greater than 35% of the length of the tendon.

All cases with structural injury to the tendon or suspicion of a structural injury should have an initial period of stall confinement and hand walking after acute inflammation subsides, usually a minimum of 30 days. Hand walking is generally 15 minutes twice daily from 0 to 30 days. Many mild tendinitis cases can begin riding at the walk at 30 days, but most moderate and severe injuries require at least 60 days of hand walking.

A second ultrasonographic examination is made at 30 and 90 days, and the tendon lesion compared to original scans. Repeat PRP or stem cell injections can be performed under ultrasonographic guidance every 30 days for three to four treatments. Horses without lameness and good grades of lesion can increase the rehabilitation exercise to include riding at a walk 30 minutes daily from 90 to 120 days, increasing over the next several months. After several months of walking, and in horses without lameness and improvement in ultrasound grade, horses can begin trotting under control. One method is to have horses trot under saddle only on the straight portions of the ring to avoid asymmetric loading on the limbs. The number of laps and times per day can be increased, gauged by the progress of the horse and tendon healing on ultrasound. Cantering can be added for 5 minutes every 2 weeks for 120 days for good and fair cases, whereas poor cases have a reevaluation and discussion of further treatment options. No active race or jump training should begin for at least 6 months from the injury.

Arguably, a controlled exercise program may be preferable to turnout after 60 days. This statement is supported by the results of a retrospective study of 50 Thoroughbred racehorses with SDF tendinitis.[50] The severity of the lesion and type of rehabilitation were significant. Of 16 horses kept in pasture, 2 of 8 raced. Of 28 horses kept in a controlled exercise regimen, 20 of 28 raced. Successful cases usually require 8–9 months of rest and rehabilitation to return to their previous full workload. Shortening this period or advancing too quickly often results in worsening of the tendon lesions. The premise for use of controlled exercise is to initially reduce inflammation, maintain gliding function, and improve healing.

Prevention

Based on changes seen in tendon matrix associated with development, aging, function, and exercise, it has been hypothesized that immature tendon may have a greater adaptive ability and may respond to an appropriate exercise regimen to produce a more functionally adapted tendon.[49] Recent studies suggest that foals allowed free pasture exercise develop a larger, stronger, more elastic tendon compared with those that were confined or subjected to a training program. Effects on the noncollagenous matrix appear to be responsible for these differences. In contrast, training or excess exercise may have permanent detrimental effects on the biomechanical and functional properties of the SDFT in the foal. The implication is that the determination of optimum exercise intensity and timing and the role of the noncollagenous matrix in tendon physiology in the young horse may hold the key to developing tendons that are more capable of resisting injury. Optimal conditioning of a young horse may result in improved performance and reduced tendon injury later in life.[33,125]

DEEP DIGITAL FLEXOR TENDINITIS

Strain injuries of the DDFT in the metacarpal or metatarsal region are uncommon compared with injuries of the SDFT, ALDDFT, or SL on the basis of both postmortem surveys[139] and ultrasonographic examinations.[48,109] However, many DDFT lesions are within the hoof and cannot be seen with ultrasound.

Etiology

Classic deep digital flexor (DDF) tendinitis in the distal metacarpal/tarsal area is usually traumatic in origin, but direct blunt trauma may be a more common cause than with SDF tendinitis. Excessive strain (deformation) of the tendon, usually as it passes over the fetlock, can induce inflammation or structural damage. The anatomic confluence of the digital sheath and annular ligament relatively immobilizes the DDFT at this site, possibly predisposing this area to injury. Horses that hit themselves around the fetlock region with other limbs during exercise can induce a DDF tendinitis. DDF tendinitis can be confused with SDF tendinitis and desmitis of the ALDDFT based on visual appearance (Figure 4.170).[139]

Many, if not most, DDFT abnormalities are degenerative in nature and are located in the distal phalangeal region of the forelimb. Distal injuries of the DDFT palmar/plantar to the flexor cortex of the navicular bone are a well-recognized postmortem and MRI finding and are considered part of the heel pain syndrome.[81,104]

Clinical Signs

DDF tendinitis is suspected with careful physical and lameness examination. It can be confused with distal SDF tendinitis (low bow) or desmitis of the ALDDFT on visual examination and palpation, especially if edema or hemorrhage blends the structures (Figure 4.170). Limbs should be palpated both with load on and off the limb. In unloaded limbs, the SDFT can be moved off of the DDFT, permitting better palpation. If injury of the DDFT has occurred within the digital sheath, sheath distension can also make palpation of the structures more difficult. In both SDF low bow and DDF tendinitis, a convex

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Figure 4.170. Horse with DDF tendinitis illustrating the location of many of these lesions in the distal aspect of the metacarpus (arrows). However, the swelling can appear similar to SDF tendinitis. *Source*: Courtesy of Dr. Gary Baxter.

appearance to the caudal limb just above the proximal aspect of the annular ligament occurs, suggesting a relative annular ligament constriction.

Horses with acute injury to the DDFT are usually lame. Ultrasound examination and/or MRI is used to determine the structures involved and the extent of structural damage. Contrast CT has been used to assess angiogenesis of DDF tendinitis and can be used as an alternative to MRI.[104]

In one study, classic traumatic-induced DDF tendinitis was seen in 24 cases with ultrasonographic lesions in the DDFT in the metacarpal/metatarsal region.[7] Clinical signs included a sudden onset of mild to moderate lameness associated with filling of the digital flexor tendon sheath. Intrasynovial analgesia of the DFTS consistently improved the degree of lameness in all cases. Regional four-point blocks also produced a significant improvement, but there was a less consistent response to abaxial sesamoid nerve blocks. On ultrasound examination, 20 of 24 cases had small, distinct, often circular focal hypoechoic areas within the DDFT in the distal metacarpus/metatarsus, usually within the digital sheath and proximal to the proximal sesamoid bones. These hypoechoic areas tended to be localized and extended only a short distance proximodistally (usually less than 1 cm). The lesions were considered focal rather than true "core" lesions as seen in the SDFT and could be easily missed if not carefully examined through the digital sheath. All cases had an increase in the amount of fluid in the DFTS.

Longitudinal tears of the DDFT in racehorses have been reported and can be initially thought to be DDF tendinitis. Ultrasound examination has failed to identify the tears in a significant number of horses. Most longitudinal tears occur within the sheath, and effusion should direct the clinician to further investigation of the digital tendon sheath. See the section on digital flexor tendon sheath tenosynovitis.

Treatment

Treatment of DDF tendinitis is similar to that of SDF tendinitis in terms of physical therapy, systemic medical management, and convalescence. DDF tendinitis may be treated surgically with distal accessory desmotomy for the same reasons that SDF tendinitis may be treated with proximal accessory desmotomy to relieve tension on the DDFT.[11] Literature supporting this treatment is limited, partly due to the rarity of the condition. Injuries to the DDFT within the sheath are frequently treated as a DDF tenosynovitis, in addition to treatment of the tendon injury. See the digital tenosynovitis in the fetlock section.

Prognosis

The prognosis for DDF tendinitis is guarded, depending on severity. In one study of 24 cases,[139] only 7 returned to athletic use.

DESMITIS OF THE ACCESSORY LIGAMENT OF THE DEEP DIGITAL FLEXOR TENDON (DISTAL CHECK LIGAMENT)

The ALDDFT (distal or inferior check ligament) is a direct continuation of the palmar carpal ligament and fuses to the DDFT in the proximal metacarpus.

Etiology

The ALDDFT provides stability to the hyperextended carpus in the midstance phase of the stride because it shares tensile load with the DDFT.[74] Desmitis of the ALDDFT has been reported as a fairly common forelimb injury, third behind suspensory desmitis and SDF tendinitis. This condition is reported in pleasure horses, ponies, and show jumpers; is less frequent in racehorses and event horses[37]; and may not be recognized as a serious problem initially.[85]

Clinical Signs

Lameness occurs suddenly after work and is associated with visual and palpable swelling in the proximal metacarpus of often older horses (older than 10 years) within 24 hours.[85] Ultrasound examination reveals enlargement of the diameter of the accessory ligament and hypoechoic areas indicating edema, hemorrhage, or fiber damage. Swelling in this region tends to persist and progress to a firm fibrosis. Lameness is consistent and improves with rest, but recurs with resumed exercise, similar to other soft tissue injuries to the flexor surface of the forelimb.

Treatment

Treatment of ALDDFT differs from that of SDF or DDF tendinitis in that the accessory ligament structure

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is not considered paramount to supporting the load in the working horse. Therefore, treatments may be more geared toward eliminating pain and lameness. Treatment has empirically included oral nonsteroidal medication, intralesional hyaluronan injection to reduce adhesions, and intralesional injection of steroid to reduce pain and inflammation as well as to reduce fibrosis. More recently, ALDDFT desmitis has been treated with injections of stem cells, BMAC, and PRP.

Desmotomy of the ALDDFT may relieve excessive tension on a fibrotic ALDDFT in chronic cases (greater than 3 months); in one study, only 14%–18% made a full recovery.[12] It has been suggested that desmotomy may prevent re-injury.[11] Horses that have received a desmotomy of the ALDDFT as a treatment for chronic rotation of the third phalanx can have pain at the desmotomy site when returned to full work. Similar medical treatment as described above can be effective in eliminating pain. Topical nonsteroidal and steroidal mediations can be effective.

As with all other soft tissue injuries to the flexor surface of the metacarpus/metatarsus, a controlled convalescent exercise program should be followed based on lameness and ultrasound examination at 60-day intervals.

Prognosis

The reported prognosis for resolution of pain and return to work is guarded. In one study of 27 horses, 12 (44%) resumed work within 5 months, and 10 (37%) eventually resumed full work. It is strongly recommended that controlled exercise be used, rather than pasture turnout, and that lameness is resolved long before healing is complete. The horse's return to work should be based on the results of serial ultrasonographic appearance.

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5

CHAPTER

Lameness of the Proximal Limb

THE CARPUS

Chris Kawcak

The carpus is composed of three joints: the antebrachiocarpal (radiocarpal) joint lies between the distal radius and the proximal row of carpal bones, the middle carpal (intercarpal) joint lies between the proximal and distal row of carpal bones, and the carpometacarpal joint lies between the distal row of carpal bones and the proximal third metacarpus (Figure 5.1). The proximal row of carpal bones is composed of the radiocarpal bone, intermediate carpal bone, ulnar carpal bone, and accessory carpal bone. The distal row of carpal bones is composed of the second, third, and fourth carpal bones, and occasionally the first carpal bone. These bones have been shown to move independently through strong intercarpal ligaments that help to dissipate the axial stress through the carpus.

Although the antebrachiocarpal joint is usually isolated from the other carpal joints, one report has documented a communication to the middle carpal and carpometacarpal joints.[37] The middle carpal and carpometacarpal joints communicate routinely, and on occasion the antebrachial carpal joint and the carpal sheath can communicate.[103] Of clinical significance is the fact that the carpometacarpal joint has distal palmar outpouchings that extend distally to the axial side of the second and fourth metacarpal bones. This area interdigitates with the proximal suspensory ligament; therefore, infusion of anesthetic into this area may inadvertently lead to anesthesia of the carpometacarpal and middle carpal joints. Conversely, injection of the middle carpal joint may lead to inadvertent desensitization of the proximal suspensory ligament area.[38]

Disease within the carpus often results from stress-induced fatigue damage that leads to osteochondral damage at consistent sites, especially in racehorses. Carpal disease can also result from acute damage that can lead to osteochondral damage at inconsistent sites or damage to the soft tissues, which typically occurs in nonracehorses and as acute traumatic incidences in all horses. Because of the consistency of stress-induced damage within racehorses, clinicians are more likely to give a reliable prognosis based on large retrospective studies of these problems. However, acute lesions in unusual sites are often difficult to completely characterize and treat, and it is difficult to give an accurate prognosis. In addition, osteoarthritis (OA) is common in the carpus in all breeds and can either result from progression of obvious damage early in life or can be insidious in onset later in life.

Osteoarthritic carpi typically manifest into progressive joint space narrowing, osteophyte and enthesophyte formation, and restricted range of motion. Often there is varus deformity due to medial collapse of the joints. Regardless of the site of pain within the carpus, horses with carpal pain typically show limb abduction while moving at both the trot and the walk and consequently have a very short gait. Although synovial effusion of the carpal joints is usually indicative of primary carpal pain, a lack of effusion can occur in cases of primary subchondral bone pain. Conversely, veterinarians must also be careful of swelling in structures other than the carpal joints such as the extensor tendon sheaths and acquired bursitis lesions such as hygromas. Effusion in either structure can be mistaken for synovial effusion of the dorsal aspect of the carpal joints.

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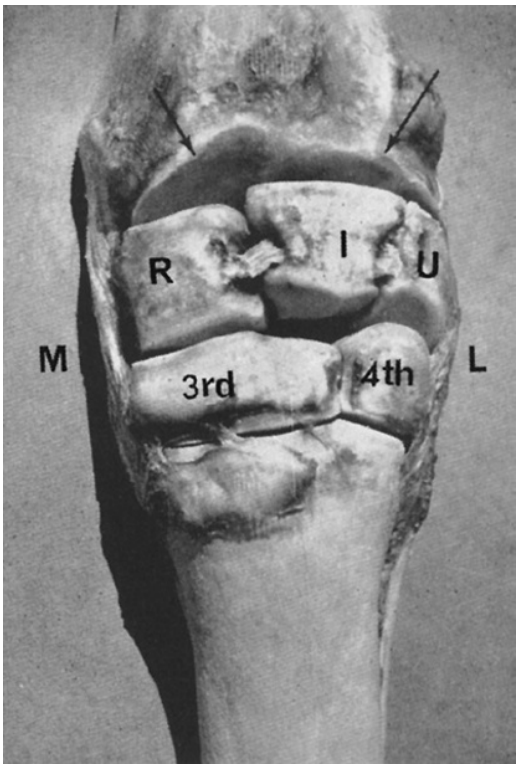


Figure 5.1. Normal left bony carpus. The joints (from proximal to distal) are the antebrachiocarpal (radiocarpal), middle carpal (intercarpal), and carpometacarpal joints. Arrows, areas where chip fractures may occur on the radius; M, medial; L, lateral; R, radial carpal bone; I, intermediate carpal bone; U, ulnar carpal bone; 3rd, third carpal bone; 4th, fourth carpal bone.

DEVELOPMENTAL ABNORMALITIES OF THE CARPUS

Developmental abnormalities of the carpus are one of the most common problems at birth in young horses. The types of developmental problems can be classified into three categories:

1. Angular limb deformities (ALD) or deviation in the frontal plane is defined by the joint and/or bone at the center of the deviation and further defined by the placement of the distal limb in reference to that center.[13] For instance, in the carpus, the distal limb is medial to the carpus in foals that have carpal varus, and the distal limb is lateral to the carpus in those that have carpal valgus. It should be noted that most foals are born slightly carpal valgus because there is greater musculature on the lateral aspect of the distal radius compared with the medial, thus providing increased tension across the carpus on the lateral side of the limb. However, with birth and growth, this usually self-resolves to a normally straight limb.
2. Flexural deformities are defined as deviations in the sagittal plane of the knee so that the joint of reference is cranial or caudal to a line drawn down the center of the limb from the lateral side.[13]
3. Rotational deformities occur when the limb distal to the point of origin rotates outward or inward from the normal axis.[13]

Angular and flexural deformities are usually pathologic in origin and often require prompt therapy. However, rotational deformities are often conformational, and the origin often occurs proximal to the carpus. In those foals in which angular and flexural deformities are deemed to be of clinical significance, prompt therapy is needed to prevent lameness and loss of future athletic performance.

A study of three breeds of French horses (Thoroughbred, trotters, and Selle Français) showed that carpal valgus was the most frequent congenital abnormality (found in 42.1% of foals) and that carpal contracture occurred in 30.8% of foals. During the study conformation became less valgus and most issues resolved by the time of weaning.[101]

Angular Limb Deformity

ALD in general are discussed in Chapter 10, but more detail is provided here on ALD of the carpus. ALD of the carpus are one of the most commonly treated problems in equine practice. However, mistakes are often made using both overconservative and overaggressive approaches to treatment. An understanding of the normal developmental patterns of the limbs is essential to avoid these mistakes.

Etiology

ALD of the carpus can result from asymmetric growth in the distal radial metaphysis, asymmetric growth of the distal radial epiphysis, incomplete ossification of the cuboidal bones or the proximal aspect of the second and fourth metacarpal bones, and/or joint laxity. ALD are typically congenital or acquired. Congenital abnormalities such as incomplete ossification of the cuboidal bones and collateral ligament laxity are not uncommon. Uterine malpositioning or stagnant movement can lead to decreased growth, which can be correlated with mare nutritional status.[77] Cuboidal bone ossification is typically complete by the time of foaling,[82] but without complete ossification, normal loading (weight-bearing) within the joints can cause progressive damage to the cartilaginous structure, predominately on the lateral side of the joints leading to progressive valgus deformity. Toxic chemicals and other possible endocrine influences have also been linked to congenital ALD.[3,39,50,83]

Some foals are born somewhat dysmature and may have joint laxity in the carpus. Constant monitoring and reassessment of these cases are necessary. Oftentimes the stress of turnout or small pen exercise is enough to strengthen those tissues and self-correct the problem. However, in these individuals, care must be taken to avoid excessive exercise, which could lead to progressive ALD. In these latter cases, progressive laxity can lead to pathologic stress levels within the carpal bones, possibly leading to carpal bone malformation. Carpal deformity should be evaluated before turnout and then again after a determined amount of time (1–2 hours). If the deformity is slightly worse after turnout, but returns to baseline throughout the day, then the amount of turnout can be adjusted to help the foal adapt and self-correct.

Foals that have acquired ALD are usually born normal, but the limb starts to deviate with time. This can

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be caused by unrecognized congenital ALD that worsens, trauma to the physis or the epiphysis, lameness on a contralateral limb, overnutrition as a foal which induces large body mass, inappropriate exercise, lack of exercise, excess exercise, or poor conformation with growth.[13] As an example, foals that have narrow chests at birth and yet straight limbs have a propensity to develop a wide chest that may lead to carpal varus in the future. In these cases it is good to examine the mare and possibly the stallion to try to determine the type of conformation that may influence the joints in the future. If trauma to the physis or epiphysis is severe enough, the growth may actually stop on that side due to damage to the physeal cartilage, sometimes leading to bone bridging across the physis.[39] Any skin abrasion over a physis should be thoroughly explored to be sure that contamination and subsequent septic physitis does not develop.

Clinical Signs and Diagnosis

The most important information that a veterinarian can give an owner about foals is that they require constant monitoring during growth. Foals can be considered to be easily deformable due to a number of factors during growth, as mentioned above, and they require at least weekly monitoring to stay ahead of rapid changes. For instance, a lameness of any cause in any limb may cause secondary ALD in other limbs. Therefore, during history taking, it is important to know how often the foal has been monitored and the changes noted by the owner since birth. The history should reveal whether these foals were normal at birth, if they were premature, the diet of the mare and foal, and when the ALD was first noticed.

The foal should be examined both statically and dynamically. It should be allowed to freely walk around so as not to induce an abnormal stance through handling (Figure 5.2). This should be done for several minutes because clinical impressions can easily change based on how the foal is standing. It is also common to examine the limb with the veterinarian standing at the foal's shoulder, looking straight down through the radius, carpus, and third metacarpus to assess ALD. In addition, because rotational deformities are common with carpal ALD, care must be taken not to overinterpret the severity. For instance, it is common to see outward rotation of the limb in addition to a carpal valgus deformity. If the foal is examined from directly in front of the foal's head, then this rotational deformity can make the ALD look worse than it really is. It is important to remember to stand directly in front of the face of the particular carpus. In the case of the previous example, the examiner must stand dorsolateral to the midline plane of the foal to accurately assess each carpus.

In some instances the author recommends that periodic photographs or videos be e-mailed for review, and in these cases it is important to remind the owner that the photographs must be taken directly in front of the face of the carpus. It is common to recommend taking several photographs starting at the very front of the foal and rotating laterally right and left in front of the knee.

During physical examination, the foal can be manually "squared up" if needed; however, that must be taken into consideration along with assessment of the foal moving around freely and standing freely. The foal should also be walked to characterize foot flight. For instance, it is not uncommon in foals with carpal varus to break over the dorsolateral aspect of the foot, in which case they may actually swing the limb out. The same is true with carpal valgus, in which the foal may swing the leg medially due to dorsomedial break-over. The conformation of the hoof can often help in determining break-over, because the site of break-over—either dorsolateral or dorsomedial—will be worn compared with the other side.

During physical examination it is important to identify the site of deformity and estimate its severity, characterizing it as mild, moderate, or severe. Some people advocate the use of a goniometer; however, classifying these into mild, moderate, and severe is sometimes just as effective. It is important to jog these foals or let them run free in a small pen to determine whether a subtle lameness is present, especially in an opposing limb, and to palpate the limbs for effusion, physeal pain, and



Figure 5.2. A foal demonstrating normal degree of carpal valgus expected after birth in the left forelimb and a moderate carpal valgus of the right forelimb. Notice that the foal is standing fairly square on its own, providing a good examination. However, the examination findings should be an assessment based on several minutes of examination and movement.

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swelling (Figure 5.3). It is also important to manipulate the limb to characterize any joint laxity. Physeal swelling in particular should be palpated for pain because septic physitis can be insidious in onset and severity.

The above physical examination techniques can only be performed if the foal is sound and standing. When the foal is recumbent, it can be observed while trying to stand. If this cannot be accomplished and if the owner has not seen the foal stand and nurse, then serum IgG concentrations in the foal should be assessed. If the foal is lying down, joint laxity must be assessed, and if there is severe ALD, it is important to assess whether this is reducible and if swelling is present anywhere on the limb. The limb should be put through a full range of motion to assess whether pain is present and to assess the full range of extension and flexion.

Diagnostic Imaging

Radiographs of the carpus should be performed to include the distal radius and proximal cannon bone for points of reference for measuring ALD. Some clinicians advocate using long film detectors to do this; however, appropriate assessment can usually be accomplished using standard detectors. It is important to remember that the growth status of the physes often cannot be accurately assessed radiographically. For instance, characterizing a physis as open or closed is difficult radiographically, since growth stops well before radiographic evidence is seen. In particular, assessment of distal radial physeal closure cannot be accurately estimated with radiographs, because it is not uncommon for the physis to remain visible even after growth has stopped.



Figure 5.3. A weanling's limb showing mild carpal varus deformity and physeal swelling on the medial aspect of the distal radius.

Both dorsopalmar and lateromedial views are necessary. On the dorsopalmar views, lines are drawn dissecting both the distal radius and the proximal third metacarpal bone; the area where the lines intersect helps to describe the location within the joint in which the deviation is occurring (Figure 5.4).[19] For instance, if the intersection occurs within the joints, then cuboidal bone malformation often is the cause of the ALD. A goniometer or digital measurement can be used on the radiograph to measure the severity of angulation. In addition, lines can be drawn through the joint surfaces and the distal radial physis, and the line or lines that deviate from perpendicular to the third metacarpal bone can be identified as the site of deviation.

Care must be taken not to overinterpret these results. It is not uncommon to have to sedate foals to acquire radiographs, and the sedated stance may not truly represent the severity of deformity. In most cases sedation can make any degree of joint laxity worsen the appearance of the radiographic findings.

In addition to measuring angles, the degree of ossification, malformation, and damage to the joint must be characterized. It is recommended that a full series of carpal radiographs be taken if initial films show any signs of damage, because subtle damage can affect the prognosis. Any foal deemed to be premature or dysmature should have a radiographic examination of both carpi and other joints such as tarsi since incomplete ossification of the cuboidal bones is not uncommon. Dorsopalmar and lateromedial views of the carpus should be obtained. A grading system has been developed called the skeletal ossification index (SOI),[1] which has been correlated with gestational age and body weight, but not with prognosis for soundness.

It is not uncommon in the physes to see metaphyseal flare, asymmetrical widening of the physes, sclerosis of the physes (which usually occurs on the concave surface), physeal widening (which usually occurs on the convex side of the deformity), epiphyseal wedging, carpal bone wedging (which primarily occurs in the third carpal bone), distal displacement of the ulnar carpal bone or the fourth metacarpal bone, and increased angulation of the head of the fourth metacarpal bone.[14,15,98] Although it is important to characterize these findings, their significance on prognosis is questionable. In addition, any osteochondral fragments, signs of physeal trauma, septic physitis, osteomyelitis, or arthritis must be taken into consideration because their presence significantly affects the prognosis. In general, most people characterize the degree of angulation as mild (5°–10°), moderate (15°–25°), or severe (more than 25°).

Treatment

Treatment of ALD of the carpus depends on the severity and type of deformity that is present. Conservative therapy is often used for most congenital causes of ALD. Sometimes, in mild cases of deformity,

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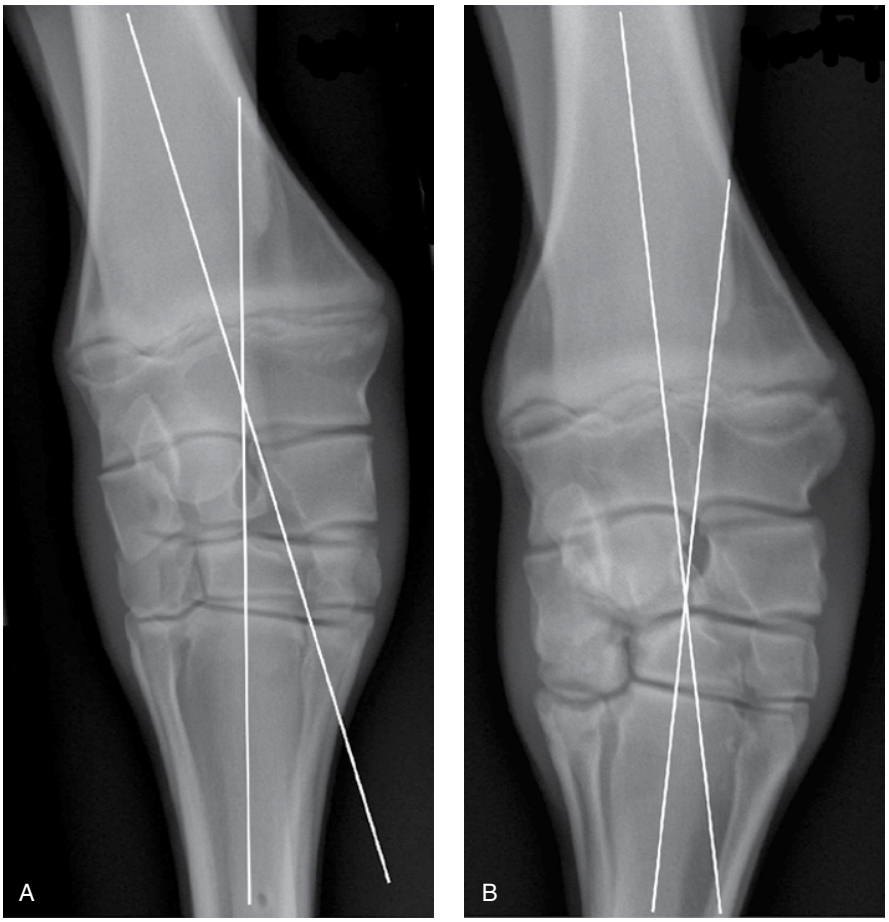


Figure 5.4. Radiographs demonstrating how lines can be used to determine the site of angulation in carpal ALD cases. (A) Distal radial epiphysis. (B) Intra-articular.

this is as simple as stall rest and proper trimming for normal break-over of the hoof. In foals with carpal valgus, the dorsomedial aspect of the foot is typically worn because the foal will break over in that location. Therefore, the dorsolateral aspect should be trimmed to achieve a normal-appearing foot. Some advocate medial hoof extensions, although there is concern that this could lead to ALD in the fetlock joint, and some advocate the use of excessive dorsolateral trimming. The author prefers to use extensions instead of excessive trimming, especially in foals that are toed out in addition to being carpal valgus. In the author's experience, prolonged use of medial or lateral extensions can lead to abnormal changes in the growth of the fetlock joint, at least until the time of distal third metacarpal physeal closure (3–4 months).

Nutritional status of the foals should also be assessed and corrected if abnormalities are detected.

If the degree of palpable joint laxity is significant, the angulation is severe, and/or there is decreased ossification of the cuboidal bones, then a tube cast or splints may be necessary to axially align the joints (Figure 5.5). A tube cast or splint is usually applied from the proximal radius distally to the level of the fetlock joint. It is important to leave the foot out so stimulation of muscle contraction of the forelimb can be continued to minimize limb weakening.

The techniques for applying tube casts and splints include a bandage alone, bandage plus PVC pipe placed on the caudal aspect of the limb, and bandage plus a cast that is either placed on the caudal aspect of the limb or a true tube cast that surrounds the limb. Care must be taken when applying splints and casts as foals are susceptible to pressure sores. Instead of PVC pipe splits, the author prefers cast material elongated over the back of the limb with similar placement to PVC pipe. Regardless of the type of application, these should be changed every 7–10 days and usually are needed for approximately 14 days. The author tries to minimize external coaptation, which weakens the limb and can cause significant sores.

Keep in mind that significant problems can result in a foal in a tube cast or in a splint, particularly slippage of the cast or the foal outgrowing the cast. Cast sores are a major problem in foals; if there is any concern about the foal's use of the limb or if heat is detected in the cast, it is best to change it at that time.[106] It is recommended that tube casts and splints be applied under general anesthesia after physically straightening the limb. The author has successfully used a customized sling and buggy system, especially in foals that have incomplete ossification of their carpal and tarsal bones.

In some cases of mild ALD, brief turnout may be helpful when the foal is thought to be strong enough.

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Figure 5.5. Front view of sleeve casts applied to both forelimbs. *Source*: Courtesy of Dr. Ted Stashak.

However, close observation is needed, especially in the first few days. It is important to observe the foal before it goes out and assess the degree of ALD. The typical recommendation is to turn the foal out for a couple of hours initially and reassess the degree of deformity when the foal comes back in. It is not uncommon to see the ALD worsen and/or the foal develop a mild flexural deformity and/or shaking in the carpal joints when it comes in from turnout. If this occurs, the foal's amount of turnout should be decreased until these signs cease. Foals usually respond quite rapidly, reaching significant or even full turnout within 7–14 days. Increased turnout may help strengthen and broaden the chest in narrow-chested foals in which carpal ALD and external limb rotation are concerns, resulting in a more normal limb conformation.

Surgical options are used when the foal either fails to respond to conservative therapy or the angulation is severe enough that it is unlikely to correct on its own. Periosteal transection has been used for decades to help correct ALD.[4,48] The rationale is that endochondral ossification is stimulated on the side in which the periosteum is transected and elevated.[4,25] In the carpus this is typically done on the concave side of the distal radial metaphysis 3 cm proximal the physis.

The efficacy of periosteal stripping has come under debate in recent years.[100,110] Although some clinicians feel that it still has merit, two studies demonstrated its lack of efficacy.[100,110] The studies show that the limb will typically correct on its own with proper foot trimming. Slone found that there was no difference between horses that underwent the procedure and those that had stall rest and appropriate hoof manipulation.[110]

Transphyseal bridging, which retards growth on the convex side of the limb, is more predictably effective than periosteal transection for correcting ALD.[40] The procedures include:

1. Placing two screws and a figure-8 wire on the convex side, in which a screw is placed proximal and distal to the physis and wire interlaced between the screws to stop the growth on that side
2. Placing a staple or plate across the physis[13]
3. Placing a transphyseal screw at an angle across the physis on the convex side

Significant growth does not slow in the distal radial physis until approximately 16 months of age; therefore, transphyseal bridges can be used out to 16–20 months of age in the carpus.[41] These bridges are most effective between 0 and 8 months of age, when the most rapid growth occurs. Bridges also can be used in severe cases younger than 6 months of age or in older cases that are nonresponsive. It has been suggested that the screw and wire technique be used in foals less than 6 months of age[128] and a transphyseal screw in those older than 6 months of age because of concern of bone bridging across the physis and overcorrection of the deformity after removal of the transphyseal screw. However, it appears that approaches are changing. The author currently will use a transphyseal screw in most cases of varus and valgus deformity. Witte et al.[128] have not seen a problem with bone bridging, and Baker et al. saw no complications when used to correct cases of carpal varus. In fact they saw no deleterious effects on sales prices or racing performances in those foals.[6] Baker et al. showed that in foals less than 2 months of age that had bilateral carpal valgus (some deemed severe up to 17°), unilateral periosteal transection had no comparable effects to the unoperated limb. This lends considerable support to the fact that at this age considerable improvement in angular deviation can occur with conservative treatment.[7]

The concern with the transphyseal screw technique is that although it is simple and quick, the effects of the screw on normal physeal physiology are not completely understood. The screw and wire technique can be used through two stab incisions and then the tissues in between undermined to place the wire to minimize scarring and potential soft tissue complications. It is not necessary to countersink the screws because that makes removal more difficult. In addition, some surgeons feel that the screws can be offset in the cranial to caudal direction to address rotational abnormalities that may originate at the carpus. In some cases, a transphyseal screw can be placed across the distal lateral radial physis in the standing yearling.[88] In nearly all cases, the screw must be removed, and this can generally be done standing or under a quick general anesthesia in more difficult foals.

In some cases a dorsomedial toe extension is preferred for foals with valgus deformity, to help further

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stimulate growth correction, although it is reserved for foals older than 3–4 months of age to avoid problems with fetlock ALD.

In addition to fixation, in severe cases in which bone bridging is thought to occur, the physis can be resected and fat graft interposed to keep the physis open.[43]

Prognosis

The prognosis is good to excellent in more than 80% of the cases.[4,14,15,40] However, Mitten et al. found that in 199 racehorses, there was no significant difference in the ability to start. Foals that had periosteal transection experienced a slight decrease in the percentage of starts, a slight decrease in the number of 2-year-old starts, and a slight decrease in the starts percentile ranking number compared with a nontreated group. Although the limbs were deemed to be straight, the investigator concluded that other factors in the carpus could have led to these reduced outcome parameters in cases treated with periosteal transection.[86] There is concern that significant ALD of the carpus could lead to subtle but permanent deformity within the carpal bones. This could theoretically lead to abnormal forces within the carpus, which is another reason to address these promptly.

Flexural Deformity

Flexural deformity, commonly known as bucked knees, is thought by some to occur in 3%–4% of foals.[13] While mild cases are typically uneventful (Figure 5.6), severe cases can result in dystocia during birth. The deformities, characterized by a disparity in length between the muscle tendon unit and bone, can be congenital or acquired. Acquired deformities occur between 1 and 6 months of age.



Figure 5.6. A foal with mild flexural deformity of the carpi that did not require treatment because it self-corrected over a few days.

Etiology

The pathogenesis of flexural deformity is unknown. Autosomal trisomy in two foals has been associated with this problem.[21] It has been hypothesized that the more common congenital occurrences are due to uterine malpositioning; poor mare nutrition; exposure of the mare to influenza, Sudan grass, locoweed, or other agents; and trauma to the limb, in which increased flexor tone and decreased extensor tone are noted.[78,79]

Acquired flexural deformity can be induced by rapid growth, overnutrition, pain in the limb, or pain in other limbs that causes overloading of that particular limb. Rapid growth is thought to induce physeal pain, and consequently some of these foals stand with their carpi flexed to relieve the pain. In addition, damage to the suspensory ligament, superficial digital flexor tendon (SDFT), or deep digital flexor tendon (DDFT), or injury to the heel or carpal pain can cause the foal to rest the limb in a flexed fashion, leading to contracture of those tissues.

It is not uncommon to see mild flexural deformity in foals that have been turned out soon after birth. In these cases it is thought that the normal exercise, especially in those foals that may be slightly dysmature, can lead to microdamage within the physes and consequently pain. This tends to improve on its own within a couple of hours of rest. In addition, rupture of the common digital extensor tendon can lead to flexural deformity; however, it is unknown whether the rupture causes the deformity or vice versa.

Clinical Signs

The degree of flexural deformity is variable and usually involves both front limbs. In mild cases it may only occur after turnout or when the foal is newborn. In these cases improvement usually occurs within days, with or without rest. In foals with significant flexural deformity, the gait is often significantly affected, and it becomes very difficult for the foal to move around. In addition, the fetlock joint may become involved in the deformity and make it difficult for the foal to rise. It is not uncommon to see skin trauma to the fetlock in these foals.

Diagnostic Imaging

Radiographs should be taken in cases that are deemed to be clinically significant. Lateromedial and dorsopalmar views are needed to assess for incomplete ossification of the bones, cuboidal bone malformation, or damage in the carpus. A complete series of radiographs should be taken if abnormalities are detected on initial views.

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Treatment

Treatment depends on the severity of the flexural deformity. In cases of acquired deformity in which rapid growth and pain is thought to play a role, the foal can be weaned to decrease the energy intake from the mare's milk or a muzzle can be applied to the foal to reduce nursing. In addition, the foal's nutrition must be reassessed, turnout can be reduced, and low levels of NSAIDs can be used to reduce the inflammation that is thought to occur in the physis. In mild cases or those that only occur with exercise, turnout can be reduced. For farm managers, it is important that they understand to observe the foals both before and after turnout to monitor whether there is improvement or worsening in the flexural deformity. This will help guide the amount of turnout each foal should have. In some mild cases, physical therapy in the form of 15 minutes of manual extension every 4–6 hours can also be used.[50]

Oxytetracycline can be administered in most cases with good results.[57,68,72] The typical protocol is 3 g oxytetracycline mixed in a 1-L bag of balanced electrolyte solution IV, once daily for 3 days. This should be given over 30 minutes to minimize any possible problems. The effects of oxytetracycline typically occur 3 days beyond the last administration.

If the deformity is severe at birth and the foal cannot stand, casts or splints can be applied immediately and changed every 24 hours. A bandage with a caudal splint is usually sufficient because the flexural deformity is in the cranial to caudal direction.[61] Again, the splint should only extend to the fetlock joint to maintain muscle strength. Foals have been shown to respond well to this.[49] Several commercially available braces can also be used. Regardless of the type of external coaptation used, close observation and monitoring are essential. Skin damage is common, and for milder cases it is often best to treat without coaptation to avoid long-term scarring.

Because of the potential of sores from casts and splints, it is not uncommon to use these for several hours a day and rotate their application. The author typically sedates or anesthetizes the foal, applies a thin bandage from the fetlock joint to the proximal radius, and molds a 4-in. cast material lengthwise from proximal to distal across the caudal aspect of the limb. Vet wrap can then be applied over this to stabilize the leg. Once this is set, it can be reused several times in daily bandage changes.

If contralateral limb lameness is present, that must be addressed to prevent and/or treat flexural deformity in the secondary limb.

Medical management of flexural deformities carries a good prognosis if the foal shows improvement within a few weeks. Carpi may buckle or shake slightly for several months; however, that appears to resolve on its own. This appears to vary with turnout and rest, which can be managed effectively.

Surgical options, while available, are of questionable efficacy. In mild cases, desmotomy of the superior check ligament and/or inferior check ligament can be done to help relax the stress in the caudal aspect of the limb. In congenital forms of flexural deformity, radiographs may indicate cuboidal bone malformation. In these cases, musculotendinous unit contracture of the SDFT, DDFT, ulnaris lateralis, flexor carpi ulnaris, and possibly the suspensory ligament can occur. Therefore, the ulnaris lateralis and flexor carpi ulnaris insertions on the accessory carpal bone may need to be severed to improve the condition.[13]

Prognosis

The prognosis is good for cases of mild flexural deformity that respond well to therapy. However, for those that fail to respond or are severe in nature, the prognosis is often poor.

MISCELLANEOUS CARPAL SWELLINGS

Extensor Carpi Radialis Tendon Damage

Etiology

Rupture or tearing of the extensor carpi radialis tendon occurs rarely in both adults and foals. Although the etiology is unknown, it has been reported in adults to occur mostly in jumpers and in horses that have exostoses on their distal radius.[23,76,115,120] This is typically traumatic in origin in foals and adults.

Clinical Signs and Diagnosis

On physical examination there is swelling of the sheath over the carpus (Figure 5.7). For those with complete rupture, overflexion of the carpus occurs as the foal or horse walks. There may or may not be extensor carpi radialis muscle atrophy, and a palpable defect is often detected within the swelling of the extensor carpi radialis tendon sheath.

Treatment

Rest is often necessary. Surgical repair of complete rupture in the acute stages in adults has been recommended, with casting for 2–4 weeks after surgery. Tenoscopy and debridement of the ruptured ends has also been advocated, along with casting for 2–4 weeks.

Prognosis

Overall, the prognosis for complete rupture of the extensor carpi radialis tendon in an adult is poor for athletic use, but guarded to good for those that have partial tears. Ruptures in foals are thought to carry a good prognosis.

Common Digital Extensor Tendon Rupture

Etiology

Usually both common digital extensor tendons are ruptured, although this can occur in a single limb. The rupture typically occurs shortly after birth. This problem has been reported to occur with other signs such as decreased endochondral ossification at other sites, decreased pectoral muscle mass, and prognathic conformation to the jaw.[84] It is overrepresented in Arabian horses, Quarter horses, and Arab-Quarter horse crosses.[84] Some have speculated that this is heritable,

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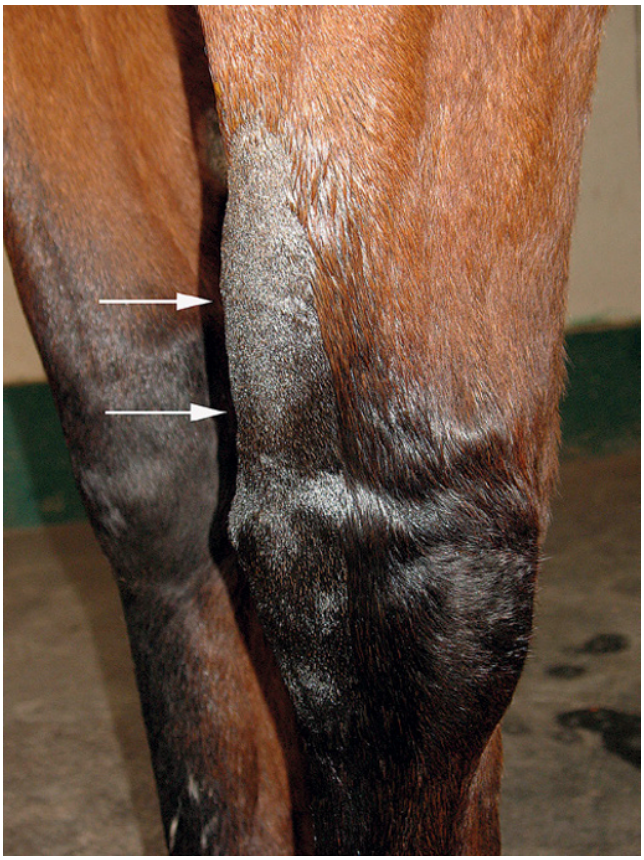


Figure 5.7. An adult horse with synovial effusion of the extensor carpi radialis tendon sheath proximal to the carpal joints (arrows). *Source*: Courtesy of Dr. Ty Wallis.

especially if other congenital defects are present. It is often uncertain as to which came first, rupture of the common digital extensor tendon or the flexural deformity. However the author has seen traumatic rupture of the common digital extensor tendons in foals that were born with severe carpal flexural deformity. Historically, these foals often demonstrate some shaking at the carpi and possible buckling. It is interesting to note that one study showed that there were histologic signs of chronic changes in the tendons in a newborn foal that had ruptured; the investigators hypothesized that the chronic changes could have occurred *in utero*.[13,132]

Clinical Signs and Diagnosis

Clinical signs include swelling on the dorsolateral aspect of the carpus, mainly in the form of effusion in the common digital extensor tendon sheath, occasional fetlock knuckling, possible palpable separation of the tendon, and variable lameness (Figure 5.8). In Meyer's report of 10 cases, 4 foals had severe flexural deformity, 3 had mild flexural deformity, and 3 had no flexural deformity. Thus, the degree of flexural deformity can be variable. These foals must be palpated because they can be easily confused with simple flexural deformities. Radiographs and ultrasound of the carpi can be performed to check for other problems and document the amount of tendon separation.



Figure 5.8. A foal showing bilateral effusion of the common digital extensor tendon sheaths due to rupture of the tendons.

Treatment and Prognosis

Treatment commonly includes stall rest with or without bandaging. Bandaging is recommended to help protect scraping of the dorsal aspect of the fetlock joint that may occur due to hyperflexion of the carpus. Primary repair of the tendons has been advocated by some clinicians,[55] although it is not necessary because the tendon ends are unlikely to heal primarily and rather adhere to the sheath. Splints or casting can be used if a persistent flexural deformity results. Fetlock joint contracture can sometimes occur, and in these cases an inferior check ligament and/or superior check ligament desmotomy can be performed.

In uncomplicated forms of rupture of the common digital extensor tendon, the prognosis is often good, although it is guarded if significant contracture is present.

DORSAL CARPAL SWELLING

Hygroma

Etiology

A hygroma is an acquired bursa on the dorsum of the carpus caused by trauma from falling, getting up and down, hitting a fence, or chronically pawing and hitting the dorsum of the carpus, such as on a feeder or water trough.[99,115,119]

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Clinical Signs and Diagnosis

Nonpainful, fluctuant, uniform soft tissue swelling occurs on the dorsal aspect of the carpus (Figure 5.9). Pressure does not induce swelling in any associated joints or tendon sheaths. Range of motion of the carpus may be reduced, but lameness is unusual. However, lameness can be severe if the hygroma is infected. Injection of radiopaque contrast agent into the hygroma confirms its extra-articular position. If a hygroma is suspected of being infected, then a fluid sample should be submitted for cytology and culture, similar to the procedures performed for septic arthritis.

Treatment

Some hygromas may resolve on their own, but in most cases drainage and injection of anti-inflammatory agents can be used; repeated injection is necessary in many horses.[115,119] Spontaneous resolution has been seen after injection of an iodine-containing contrast agent. Injection of atropine (7 mg total dose) may also help to resolve the swelling. Owners should be warned that bandaging is an essential component of treatment and that long-term chronic skin thickening may occur. Other treatments include incisional drainage, injection of irritants such as Lugol's solution, and blistering.[115,119]

Contrast radiography should be performed to ensure that the hygroma is an isolated structure. Although preoperative contrast radiographs may show no communication between a hygroma and a joint or tendon sheath, it is possible that one exists in the form of a one-way valve from the joint into the mass.[52,99] Drainage of the mass with a Penrose drain and bandaging has been used successfully for treatment of recurrent hygromas.[2] However, the author has found the effectiveness of this to be limited.

Surgical excision can be performed in horses with chronic hygroma and is best accomplished if the fluid sac is left intact and dissected from the other tissues.[113] Soft tissue and skin closure are routine, and a splint or sleeve cast can be used to prevent flexion for better healing. Prognosis for resolution of hygroma is often good, although some degree of thickening usually persists and the owner should be warned of a likely cosmetic blemish.



Figure 5.9. Chronic hygroma of the carpus. Notice the diffuse swelling isolated over the dorsum of the carpus. *Source*: Courtesy of Dr. Gary Baxter.

Synovial Hernia

A synovial hernia is a defect in a joint capsule or tendon sheath through which the synovial membrane can protrude. The condition is uncommon and rarely causes lameness, but it is a cosmetic blemish. A soft tissue mass can be palpated over a joint and often can be moved between the hernia and the underlying joint or tendon sheath. Synovial hernias can occur between the mass and either the palpable carpal joint or extensor tendon sheaths. Unlike synovial effusion, synovial hernias seem to have a larger outpouching of fluid and may disappear with joint flexion. Contrast agent injected into the hernial sac is detected in the underlying joint or tendon sheath, although a one-way valve may be present, limiting movement of contrast material. If the synovial hernia is of cosmetic concern, surgical excision can be performed, with a good prognosis for soundness provided no other joint diseases are present.[113]

Ganglion

A ganglion is a fluid-filled structure that connects to a joint or tendon sheath through a one-way tract from the joint into the mass. Unlike a synovial hernia, the mass lacks a synovial lining and often is filled with mucin. Ganglions, although common in humans, are rare in horses, and they have been reported around the stifle and the carpus.[75,99] Demonstrating a connection between a ganglion and an adjacent joint by injection of radiographic contrast agent into the mass may or may not be possible.[2]

Synovial Fistula

Synovial fistulae are communications between two synovial structures, usually a joint and tendon sheath. They have occurred between the antebrachiocarpal joint and the common digital extensor tendon, the middle carpal joint and the extensor carpi radialis tendon sheath or the common digital extensor tendon sheath, and the extensor carpi radialis tendon sheath and a carpal hygroma.[21,52,56,67] Additional joint damage is often present in association with the fistula, causing lameness referable to the area.[67] Swelling in the joint and nearby tendon sheath occurs, and fluid often moves between the structures. Radiography may reveal additional joint or tendon sheath damage, and contrast agent injected into one of the structures is visible in the other.

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Occasionally a fistula can be seen during arthroscopic surgery, but closure of the fistula requires an arthrotomy. Arthroscopic surgery for treatment of a primary problem, without repair of the fistula, has resulted in resolution of lameness without resolution of the swelling. Fistulae are typically not repaired unless a cosmetic effect is important, if the swelling itself is impeding performance, or if medical therapy fails to alleviate lameness.

INTRA-ARTICULAR FRACTURES

Although most horses with intra-articular fracture of the carpus display acute onset of clinical signs, the damage is chronic in nature, at least in racehorses, and occurs at consistent sites in the dorsal aspect of the joints. The damage is the end result of a chronic process in which stress-related subchondral bone damage occurs.[59,111] The pathogenesis of repetitive stress injury is discussed in Chapter 7. Acute fracture and fragmentation of the carpus can occur, but this is typically in unusual locations, especially in the palmar aspect of the joints, making an accurate prognosis difficult to make. Therefore, in equine athletes, and particularly racehorses, the damage occurs in predictable sites that lend themselves to more accurate prognosis with treatment.

Three types of fractures can occur within the carpal joints of the horse: osteochondral fragmentation, slab fracture, and comminuted fracture.

Osteochondral fragmentation occurs on a single articular surface and is commonly found on the radial carpal, third carpal, intermediate carpal bone, and distal radius. The fragments can be attached with fibrous tissue, loose, or free within the joint and are categorized as (1) recent and complete fragment; (2) fragment with capsule attachment; (3) chronic fragment with bone attachment, usually by fibrous tissue; or (4) fragment with extensive attachment to the bone.[13]

Regardless of the type of fragmentation, osteochondral fragmentation typically occurs at consistent locations; again, this reflects the chronic nature of the disease. Thoroughbreds and Quarter horses commonly show fragments within the radiocarpal and middle carpal joints. In Thoroughbreds, fragmentation commonly occurs off the distal aspect of the radiocarpal bone, distal lateral radius, and proximal aspect of the intermediate carpal bone. In Japan, the most common sites for fragments to occur in Thoroughbred racehorses are the distal lateral radius, proximal aspect of the third carpal bone, and distal aspect of the radiocarpal bone.[87] In racing Quarter horses, fragmentation occurs off the distal aspect of the radiocarpal bone, proximal aspect of the intermediate carpal bone, and distal lateral radius.[79] In Standardbreds, the fragmentation typically occurs in the middle carpal joint, especially on the third carpal bone (Figure 5.10).[69,93,105] It is important not to rely on these generalizations because fragmentation can occur anywhere within the carpi and the distribution may differ with different population bases.[81] Fragmentation can also occur at the palmar aspect of the antebrachial carpal and middle carpal joints.[62,127]

Slab fractures of the carpus usually occur completely through the bone (they fracture through both articular surfaces), and although they are most common in the third carpal bone, they can occur in the intermediate and radial carpal bones as well. In Thoroughbreds and Standardbreds, slab fractures commonly occur on the third carpal bone and primarily affect the radial facet. These fractures can also occur on the intermediate facet, both facets, or in a sagittal orientation.[75,105,116]

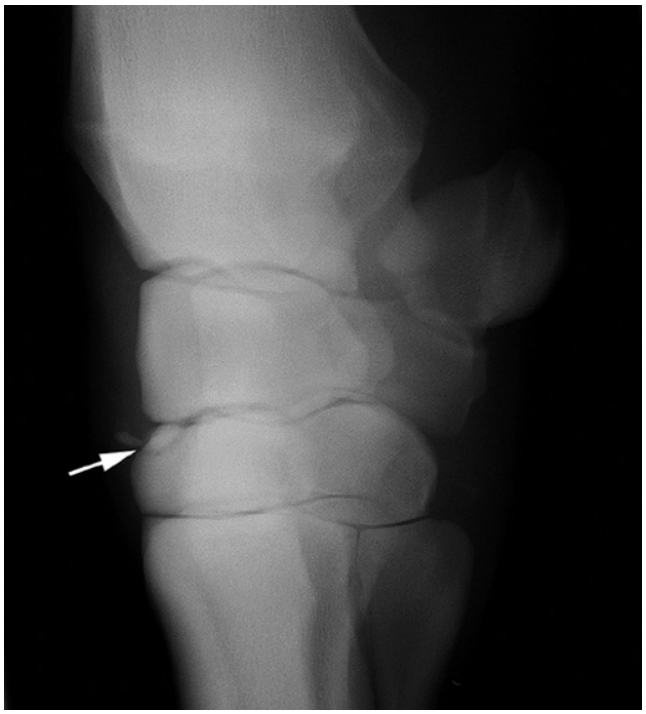


Figure 5.10. Oblique radiograph of the carpus demonstrating osteochondral fragmentation of the proximal third carpal bone (white arrow). Although this site of damage is seen more commonly in Standardbreds, this was a racing Quarter horse. *Source*: Courtesy of Dr. Gary Baxter.

Comminuted fractures of the carpus occasionally occur, and although they primarily involve the third carpal bone, they can also involve the radial carpal, intermediate carpal, and fourth carpal bones. Horses that suffer these fractures are usually axially unstable. They require emergency stabilization and usually surgery to be sound for breeding.

In addition to fragmentation and fracture of the carpus, it is not unusual to find subchondral bone lysis that leads to pain, typically in racehorses. This primarily occurs in older Standardbred racehorses due to chronic stress-induced disease within the subchondral bone.[103] This syndrome is discussed in the OA section of this chapter.

Etiology

The biomechanical forces that occur in the carpus and lead to fracture are difficult to characterize due to the large number of bones and soft tissues involved. The carpal bones are not in direct contact with their opposing surfaces during the flight phase of the gait. However, with weight-bearing and full extension, it is hypothesized that the carpal bones assume a congruent articulation with their opposing bones.[3,5] It is also hypothesized

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that this level of congruity can change with fatigue, increased speed, poor racing surface, poor trimming, possibly uncoordinated movement, and variability in racing surfaces, perhaps leading to fracture.

Subtle geometric abnormalities that may be within these joints also could predispose these horses to fracture. Considering the incidence of ALD of the carpus in young horses, it is likely that these subtle geometric abnormalities occur, causing problems at speed. This is a known occurrence in the distal third metacarpus in the fetlock joint. Pathologic studies that have been performed in an effort to determine the cause of these fractures have led to the observation that many are chronic in nature in racehorses.[111]

It is known that increased axial loading leads to increased dorsal compression between opposing carpal bones. Therefore, chronic high loads and the instantaneous maximum loads likely lead to disease.[94] Although these horses typically present with acute lameness and swelling, histopathologic observations have demonstrated a chronic disease process that not only leads to bone microdamage but attempts at bone remodeling and healing.[34,35,92,95,111,131] It has been shown repeatedly that intense bone modeling and remodeling occur at the sites at which osteochondral fracture occurs.[59] It is likely, then, that some of these fractures may in fact occur during the peak bone remodeling phase, which includes intense osteoclastic bone resorption. Not unlike dorsal metacarpal disease, some of these horses may be predisposed to fracture during these intense remodeling periods. Therefore, it is likely that chronic repetitive stress can lead to a chronic pathologic process that ultimately leads to bone failure and demonstration of clinical signs.

Soft tissue fatigue that can occur within the joints includes a decrease in soft tissue support of the joint capsule and the surrounding tendons and ligaments. This is compounded by the hypothesis that the hoof often impacts the ground before maximum extension occurs, thereby causing the joint to snap shut and leading to the assumption that these intensive, pinpoint stresses can occur at these sites[3,5] and may lead to damage. In addition, it has been observed that fatigue of the flexor tendons and muscles in the caudal aspect of the leg can lead to hyperextension, as has been observed in many horses at the end of the race. Because of the prevalence of damage that occurs dorsally, several investigators have hypothesized that a back-at-the-knee conformation can lead to increased incidence of disease.[3,103] However, Barr et al. showed that there was no correlation to the incidence of carpal damage when this conformation was evaluated.[9] Others have observed that most horses in Europe are "over at the knee" and therefore likely have a decreased incidence of this problem, although this has not be substantiated.[3]

Clinical Signs

Horses with fracture in the carpus can appear with various degrees of lameness, synovial effusion, soft tissue swelling, and carpal flexion during standing. If osteochondral damage is complete and enters the joint, synovitis typically results, which leads to clinical signs of joint effusion and pain. However, on rare occasions when subchondral bone damage alone occurs, synovitis leading to joint effusion may not be appreciated, and the horse may or may not be positive to carpal flexion. Horses with small osteochondral carpal fragments are often only subtly lame with mild amounts of effusion and soft tissue swelling.

The common characteristic of their movement is abduction of the forelimbs, which some have hypothesized is an attempt to minimize carpal flexion. Consequently, this decreases hoof height during flight. Horses with osteochondral damage are often positive to carpal flexion. Horses that have complete fracture such as slab fractures may have intense pain even with passive flexion. Horses with fracture or fragmentation of the palmar aspect of the joints are often significantly responsive to flexion, which reflects the extensive soft tissue damage that can occur in this area. In more chronic stages of osteochondral fracture, there may be physical limitations to the amount of flexion in the carpus, with or without pain.

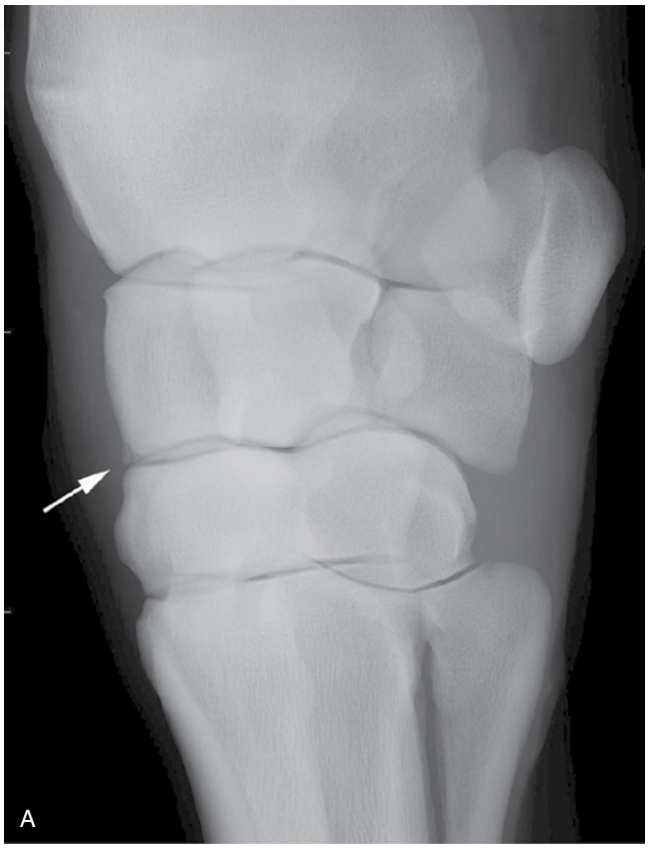
Horses with comminuted fractures of the carpus are often axially unstable, and effusion may be associated with subcutaneous swelling. If the horse can tolerate it, it is a good practice to palpate the dorsal aspect of the carpal bones while flexed, because horses with osteochondral damage often demonstrate pain on palpation of certain bones. Often, fibrous thickening of the joint capsule can be appreciated if a significant amount of joint capsule is involved in the fragmentation. However, in these cases, the presence of a hygroma or extensor sheath swelling must be ruled out.

If there are obvious signs of osteochondral fracture, it is often a good idea to perform a radiographic examination prior to performing intrasynovial analgesia because with loss of pain, the horse no longer protects the limb, possibly leading to worsened damage. If a subtle lameness is present or radiographs are inconclusive, then intra-articular analgesia is often needed to confirm the site of pain. For intra-articular analgesia of the carpal joints, 5–7 mL of anesthetic is injected into the joint and the horse checked after 10 minutes and again at 30 minutes.[58] Horses that have incomplete slab fractures that have not broken into the joint may not respond significantly to intra-articular anesthesia. The same may be true of horses that suffer from subchondral bone disease of the middle carpal joint. However, in these horses, there may be subtle preexisting lameness that prompts further diagnostic imaging.

Diagnostic Imaging

A minimum of six radiographic images are needed to fully characterize the carpal joints, especially those with small fragmentation (Figure 5.11). Most of the fragments are on the very dorsal aspect of the bones; therefore, some practitioners recommend that oblique views be taken more parallel with the lateromedial view to fully characterize the damage and detect subtle lesions. Images should be evaluated for osteochondral damage that can be surgically addressed, as well as osteoarthritic changes such as osteophyte, enthesophyte, and joint space narrowing that can affect the prognosis for any surgical outcome. Not all radiographic abnormalities can be clinically significant. Simon and Dyson showed

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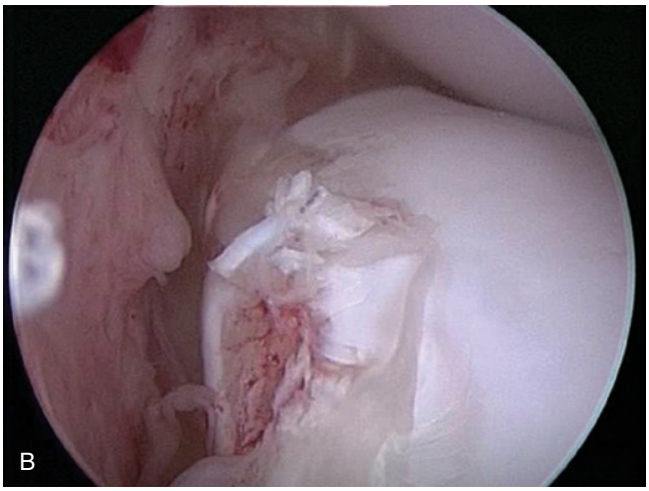


Figure 5.11. A radiographic image (A) showing a subtle fragment on the distal aspect of the radial carpal bone (arrow). The corresponding surgical image (B) is shown as well. This demonstrates how radiographs typically underestimate the severity of articular cartilage damage within the joint.

that radiographic abnormalities within the ulnar carpal bone are likely incidental findings and oftentimes may not be clinically significant.[108] At times it can be difficult to identify the clinical relevance of what may appear to be an osteophyte in the joint; however the presence of this finding along with synovial fusion and lameness often confirms an intra-articular component involved in the disease process, therefore justifying surgical exploration.

Both limbs should be radiographed, because more than 50% of all horses with osteochondral fragments have them in both carpi.[79] In addition, the third carpal skyline view is important for detecting sclerosis, lysis, and fracture and has been shown to give a good impression of third carpal bone density.[118] Some surgeons believe there is a correlation between the amount of pain and the duration and displacement of the fragment, but no objective studies have characterized this.

Mineralization in the palmar aspect of the carpal joints is often appreciated. Some clinicians have attributed this to chronic corticosteroid injection; however, there is evidence that sometimes this is due to chronic damage within the dorsal aspect of the joints that leads to maceration of the fragments and migration to the palmar aspect of the joint.[44] Sometimes significant OA and articular cartilage loss are present in these horses. In addition, in cases of comminuted osteochondral fracture of the carpus, the third carpal bone is usually involved, along with the radiocarpal and/or intermediate carpal bone. These can be difficult to characterize, and subtle changes in the angles of the radiographic images may be needed.

Slab fractures of the carpus are usually most easily seen on the standing lateromedial projection because these fractures often reduce in a flexed lateral position. However, nondisplaced fractures often require a skyline for full characterization (Figures 5.12 and 5.13). Sagittal slab fractures typically occur on the medial aspect of the radial fossa of the third carpal bone, and either a third carpal bone skyline projection or a dorsomedial to palmar lateral oblique projection is needed to see this. In addition, subchondral bone lysis within a third carpal bone may not initially be present on radiographs, which justifies the use of subsequent radiographs to diagnose this problem once the bone in that area has resorbed. Nagy and Dyson have shown that significant osteochondral remodeling occurs in the medial aspect of the carpus as demonstrated by magnetic resonance imaging (MRI). Therefore, advanced imaging techniques are sometimes needed to fully characterize the amount of bone damage at this site.[90]

Nuclear scintigraphy is beneficial, especially in racehorses, due to intense remodeling of the carpal bones. Furthermore, scintigraphy can best characterize the intense sclerosis and lysis that can result and may cause pain. A study of Standardbreds showed that increased uptake was common in the middle carpal joint, and most of this reaction was thought to be clinically significant.[33]

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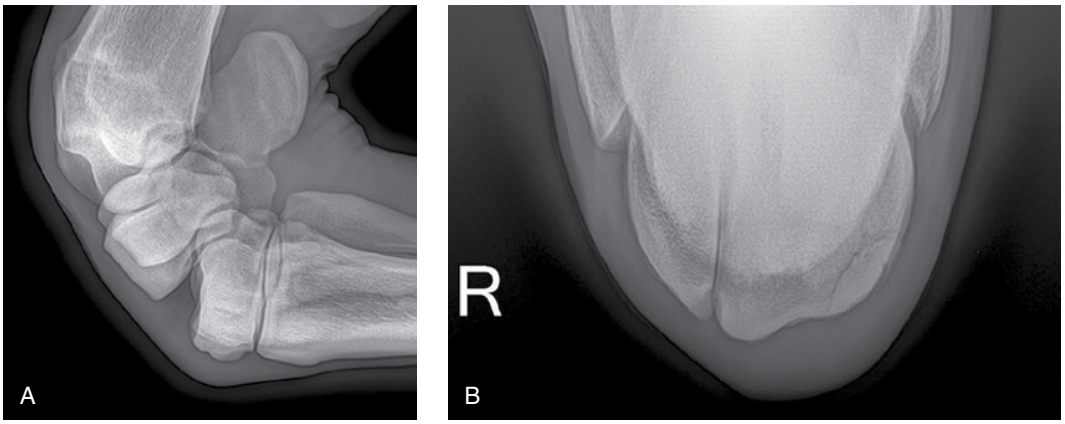


Figure 5.12. Radiographs demonstrating the need for a skyline projection to characterize a slab fracture of the third carpal bone. The fracture can be clearly seen on the skyline view (B) without any evidence of the fracture on the flexed lateral view (A). *Source*: Courtesy of Dr. Ryan Carpenter.

In the study by Simon and Dyson, lameness related to the carpal joints was associated with increased ratio of radiopharmaceutical uptake and increased radiopharmaceutical uptake grade, both of which correlated with opacity of the third carpal bone.[108] In addition, intense uptake has been observed in 2-year-old Quarter horses that were worked on a treadmill for 6 months.[59] Therefore, it is important not to overinterpret the findings because uptake can occur in horses that are undergoing active exercise.

MRI and computed tomography have also been used to characterize subtle lesions in the carpus. Computed tomography has higher resolution and can be used to detect both subtle lesions and bone mineral density abnormalities (Figure 5.14). MRI is useful for detecting soft tissue damage and bone edema.[90] Edema in the subchondral bone has been seen in joints with damage, but its cause and consequences are unknown. Ultimately, arthroscopic surgery is usually the best diagnostic tool to characterize and treat the problem in the carpus. The exception to this may be cases in which subchondral bone pain is a primary problem without the overlying articular cartilage damage.

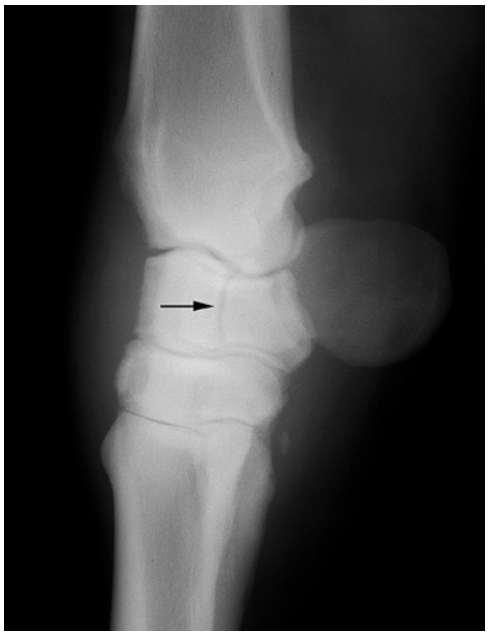


Figure 5.13. A lateral radiograph of the carpus demonstrating a slab fracture of the radial carpal bone (arrow).

Treatment

While some clinicians recommend rest for treatment of osteochondral fragmentation of the carpus, most surgeons believe that arthroscopic removal of the fragments and the resultant reduction of synovitis induced by the instability provide a more predictable outcome. Most surgeons feel that in horses that are conservatively treated, if these fragments are allowed to heal in an incongruent fashion, the fragment of bone can break away later with training, again leading to clinical signs. Arthroscopic surgery is the best method to fully characterize the disease process, treat the primary problem, and give an accurate prognosis for return to athletic use.

Degeneration of articular cartilage and bone has been graded for severity and correlated with outcome:[81]

Grade 1: Minimal fibrillation or fragmentation at the edge of the defect left by the fragment, extending no more than 5 mm from the fracture line
Grade 2: Articular cartilage degeneration extending more than 5 mm back from the defect and including up to 30% of the articular surface of that bone
Grade 3: Loss of 50% or more of the articular cartilage from the affected carpal bone
Grade 4: Significant loss of subchondral bone (usually distal radial carpal bone lesions)

After osteochondral fragments have been removed, augmentative therapy such as microfracture or various intra-articular medications can be used if the lesions are severe. There has been concern about operating horses after they have been recently injected with corticosteroids because the horses may be predisposed to postoperative synovial sepsis; however, this has not been shown to be of major concern.

Arthroscopic surgery of the carpus is typically performed to remove the fragmentation or repair the fracture. Wright

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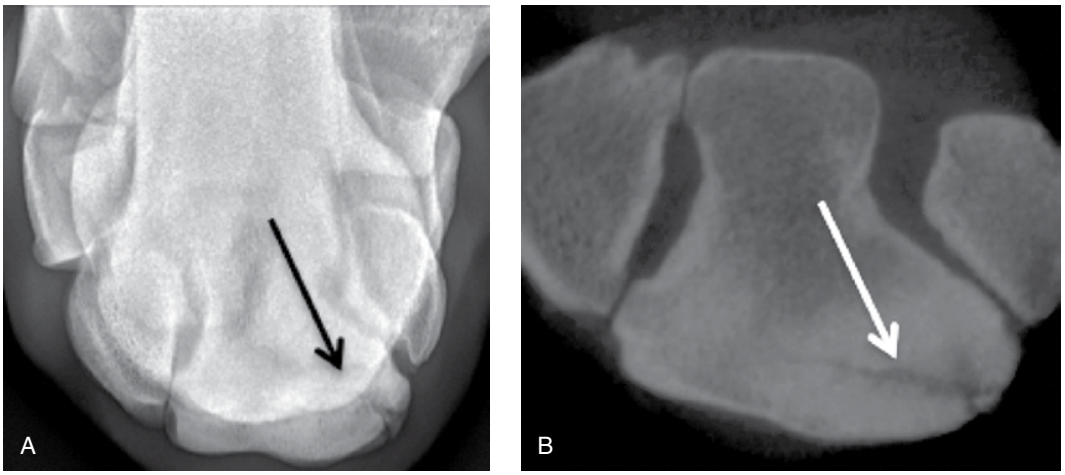


Figure 5.14. (A) Skyline radiograph of a 2-year-old Thoroughbred filly demonstrating osteochondral damage in the third carpal bone (arrow). (B) CT of the same horse that helps delineate the full extent of the fracture (arrow).

and Smith have shown the use of 2.7-mm screws to repair osteochondral fragments that are typically removed. These fragments must be of sufficient size and integrity to hold a 2.7-mm screw, and if successful, the prognosis using this repair technique is excellent.[130] For more details on arthroscopic surgery of the carpus, see McIlwraith et al.[69,81] While the majority of arthroscopic surgery is performed in the dorsal aspect of the joint, occasionally fragmentation of the palmar aspect of the joint can be removed with relatively good success.[26,62]

Slab fractures usually require internal fixation, such as lag screw fixation, to provide the best chance of achieving athletic soundness. However, thin slab fractures (less than 5 mm) can be removed because often they will not support lag screw repair. However use of smaller screws such as 2.7-mm screws may help achieve improved fracture fixation.

Although surgery may not initially be considered for horses intended for retirement or breeding, it is possible that some cases may degenerate and later become unstable or see advanced progression of OA if the fracture is not stabilized. The degree of joint surface damage, which is common with slab fractures, often dictates the prognosis of return to athletic use (Figure 5.15). Stephens et al. showed that complete displaced fractures can be repaired through internal fixation and incomplete and/or nondisplaced fractures can be treated with rest and conservative therapy.[116]

Sagittal slab fractures also are seen. In a retrospective study, 7 of 12 horses treated conservatively for sagittal fractures raced; however, most experienced surgeons believe that internal fixation is needed for the best prognosis.[36,103] Details of arthroscopic repair of slab fractures can be found in McIlwraith et al.[81] Methods to repair slab fractures vary according to surgeons. Some prefer single 4.5- or 3.5-mm cortical screws placed through the radial facet via arthroscopic guidance. Fractures through the radial and intermediate facets require two screws. A headless variable pitch screw has been used, and experimental evidence shows this to be equivalent to cortical screws for holding strength. This type of screw theoretically eliminates pain caused by a cortical screw head.[20]

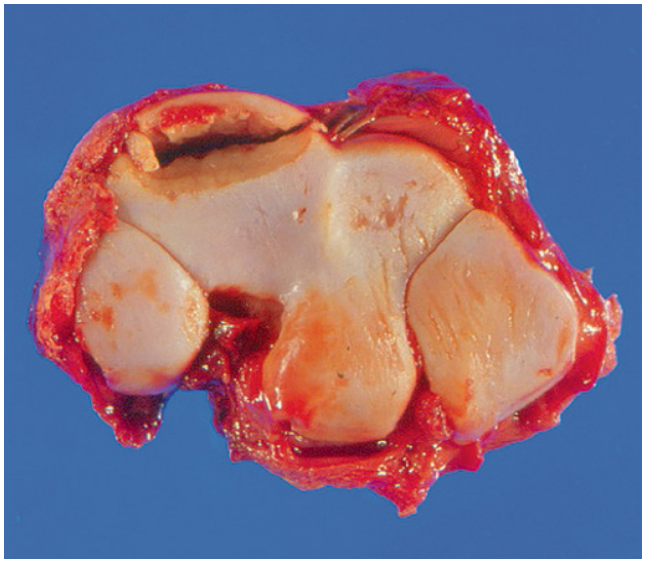


Figure 5.15. Gross image of a slab fracture of the radial facet of the third carpal bone. Notice the degeneration of articular cartilage and subchondral bone that can occur. Even with good reduction, this joint surface defect can worsen the prognosis for return to athletic function.

Comminuted fractures require internal fixation or arthrodesis to restore axial stability to the limb and give the horse a chance to become pasture sound.[16,64] The immediate stability gained from internal fixation improves the time to pain-free limb use and prevents overuse and consequent laminitis in the opposing limb. Conservative therapy with casting and/or splints results in more prolonged lameness, which can lead to cast sores within the limb and often laminitis within the opposing limb. Conservatively treated horses sometimes heal with a deviation and significant chronic pain in the limb. It is unusual for a horse to achieve athletic soundness after such an injury because the joint surface damage is often severe. Lag screw fixation can be used to stabilize individual fractures; however, in severely

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comminuted injuries, internal fixation with plates may be necessary, and partial or full arthrodesis needed.

Postoperative Care

For osteochondral fragmentation, sutures are removed at 10 days, and horses maintained in a bandage for 10 days. One common sequela to carpal bandages is the creation of sores over the accessory carpal bone and the medial distal condyle of the radius. There are several ways to prevent these, including padding on either side of the structures, creating a window in the bandage for these areas to be exposed, or using super glue or adherent drapes over the incision sites without a bandage. Most clinicians prefer bandages because the compression helps decrease postoperative swelling; however, it has been observed that super glue has worked well.

The time needed for rehabilitation depends on the amount of damage, and the intensity of postoperative rehabilitation depends on the amount of articular cartilage damage and where the damage occurred. Rehabilitation is faster for the antebrachial carpal joint than the middle carpal joint because damage in the former is less common on the weight-bearing joint surface and consequently suffers minimal stress-induced damage. In general, most surgeons recommend 2 weeks of stall rest followed by 2–4 weeks of stall rest and hand walking and then 2–4 additional weeks of turnout or swimming for problems within the antebrachial joint.[103] For mild to moderate damage within the middle carpal joint, the recommendation is 2–4 weeks of stall rest, 4 weeks of stall rest and hand walking, and 8 weeks of turnout or swimming; for severe middle carpal joint lesions or global articular cartilage damage, 4–6 months of rest are often recommended.[103] Typically horses with slab fractures require 4 weeks of stall rest, 8 weeks of stall rest and hand walking, and 2–3 months of paddock turnout. Horses should be radiographed prior to training, and rarely will the screw need to be removed.[103] For a detailed description of rehabilitation techniques, the reader is referred to Haussler and King.[46]

In addition to rehabilitation, some form of intra-articular therapy is recommended to reduce inflammation and speed healing, especially of articular cartilage. If intra-articular medication is needed, the horses can be injected at the time of suture removal or after. The author prefers interleukin-1 receptor antagonist protein (IRAP) or polysulfated glycosaminoglycan/hyaluronic acid combination (with amikacin) once weekly for 3 weeks, starting 2 weeks after surgery. Treatment with IRAP is not uncommon, especially for severe joint damage, because the growth factor content within this product can be significant and theoretically help with articular cartilage healing. Intra-articular hyaluronic acid and polysulfated glycosaminoglycan are thought to promote healing and decrease inflammation.[60] Some surgeons find intra-articular stem cell therapy to be beneficial, although a study showed minimal benefit in experimental osteochondral fragmentation. However microfracture and intra-articular stem cell therapy has been shown to have a positive effect at other sites.[80] Theoretically, the stem cells could promote release of growth factors into the joint, affecting articular cartilage healing of a defect.[60]

Some surgeons use systemic nonsteroidal anti-inflammatory medications for horses with carpal fractures, either phenylbutazone or Equioxx therapy. Local application of Surpass has also been shown to be beneficial both clinically and in experimental studies.[60] Although systemic polysulfated glycosaminoglycan and intravenous hyaluronic acid therapies generally have shown modest effects, they may help maintain horses with carpal damage. The author recommends the use of passive range of motion and swimming if significant joint capsule damage is noted at the time of surgery.[46]

Potential postoperative problems include sepsis and subcutaneous infection. Problems such as persistent effusion and osteophyte formation are usually a result of the primary disease process, although excessive debridement of the joint capsule can lead to enthesophyte formation.

Prognosis

The prognosis for osteochondral fragmentation is variable depending on the breed. For Thoroughbreds and Quarter horses, the chance of racing at the same or increased level is approximately 68%.[79,87] McIlwraith et al. found that horses with grade 1 or 2 damage were more likely to return to racing than those with grade 3 or grade 4 damage (74% vs. 54%, respectively). However, Lucus et al. found that in Standardbreds, 74% raced at least one start, although most had decreased earnings and numbers of starts.[69] For slab fractures, 65%–77% of horses raced. Martin et al. found that 68% of horses raced postsurgically, although they had significantly decreased value.[75] Stephens et al. found that 65% of Thoroughbreds raced but decreased their earnings per start and that 77% of Standardbreds raced, 100% if they had raced before.[116] The prognosis is good for riding or breeding.[65] For a more accurate summary of prognosis for arthroscopic treatment of carpal damage, the reader is referred to McIlwraith et al.[81]

ACCESSORY CARPAL BONE FRACTURE

Fracture of the accessory carpal bone can occur in any breed and primarily occurs in the frontal plane through the lateral groove of the bone (Figure 5.16). It is hypothesized that most of these fractures heal by fibrocartilaginous nonunion due to the constant pull from the flexor muscles.[10,31]

Etiology

Accessory carpal bone fractures are thought to result from external trauma; however, it is very rare to see primary skin damage over these sites. Some clinicians have hypothesized that the cause may be extreme internal forces such as asynchronous contraction of the flexor carpi ulnaris and ulnaris lateralis muscles,[5] horses landing partially flexed, leading to a bowstring effect of the flexor carpi ulnaris lateralis and flexor tendons,[71] or extreme stress induced by the bone being caught between the third metacarpal bone and the radius, because concurrent lesions have been seen in the caudal radius.[102]

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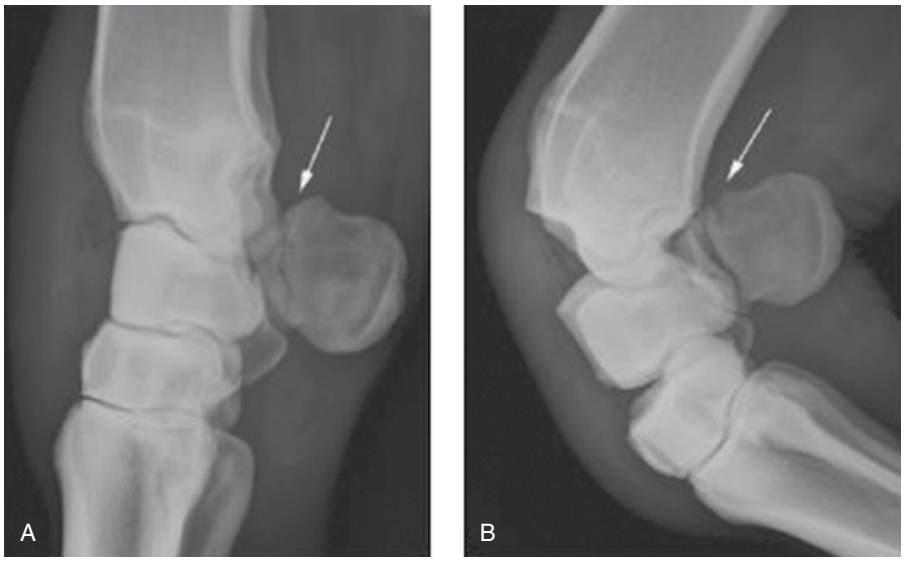


Figure 5.16. Lateromedial (A) and flexed lateromedial (B) radiographs of a fracture of the accessory carpal bone (arrows).

Clinical Signs and Diagnosis

Horses with fractured accessory carpal bone typically have acute lameness with or without dorsal carpal or carpal canal swelling. There is often palpable pain over the accessory carpal bone, crepitus is sometimes felt, and sometimes with flexion, lateral to medial instability can be palpated. Care must be taken in examining these horses because they are very painful with flexion and may stand with their carpus flexed. One frequent clinical sign is a decrease in digital pulses with flexion.[13] Radiographs are often sufficient for characterizing the fracture; however, ultrasound examination of the carpal canal can help to characterize the extent of damage. It is important that a complete set of radiographic images are obtained, especially for complete fractures of the accessory carpal bones as it has been shown that fracture fragments can displace in multiple planes and even displace within the carpal canal distally. With this in mind, it would be important to image distally to the mid metacarpus to ensure that fragments are not present in the distal aspect of the carpal canal.[85] It is important to perform a complete ultrasound examination of the carpal canal as fracture fragments can cause damage to the soft tissues of the carpal canal.[85] Ultrasound examination should be repeated during the healing process because fibrous union is common, and expansion of this fibrous response could impinge the carpal canal, leading to pain. It is thought that severely comminuted fractures could increase the chance of carpal canal involvement, but large retrospective studies are lacking.

Treatment and Prognosis

Damage to the accessory carpal bone can be treated conservatively with 3–6 months of stall rest, small fragments can be removed via arthroscopy, or lag screw fixation of larger fractures can be performed.[31] Three to six months of rest or lag screw fixation are recommended.[31] Ulnar neurectomy and arthroscopic surgery and debridement have also been recommended, but subtotal resection is no longer advocated. Small fragments involving the proximal dorsal aspect of the accessory carpal bone may involve the palmar aspect of the antebrachiocarpal joint and can be removed arthroscopically.[62] It is important to address damage to the carpal canal and remove any possible fragments that may be present and debride damaged soft tissues within that area.[85] However, because involvement of the carpal canal is often delayed until well into the healing phase, tenoscopic examination and retinacular release, if needed, also may be delayed. Periodic radiographs should follow treatment; distraction and further lysis of the fracture due to the fibrocartilaginous nonunion may be seen over time. Prognosis for healing is good.[62,85] Although return to soundness is generally good, it can be reduced if significant carpal canal involvement is evident.

CARPAL LUXATIONS

Luxation of the carpal joints is rare but can occur in any of the three joints. The medial collateral ligament is reportedly most commonly ruptured; however, the lateral collateral ligament can be affected with or without carpal bone comminution. Avulsion fractures can also occur as a result of external trauma such as foaling, jumping, falling, slipping, or from kicks.

Clinical Signs and Diagnosis

Clinically, the horses are acutely lame with swelling around the joint, and there may or may not be an ALD present, depending on the severity of damage. These luxations are rarely open; in the author's experience this occurs mostly in racehorses with catastrophic injuries to the carpus. Horses with carpal luxations may be axially unstable, and crepitus may be palpable during manual deviation of the joint (Figure 5.17). Damage to the collateral ligament can also occur on its own.[30]

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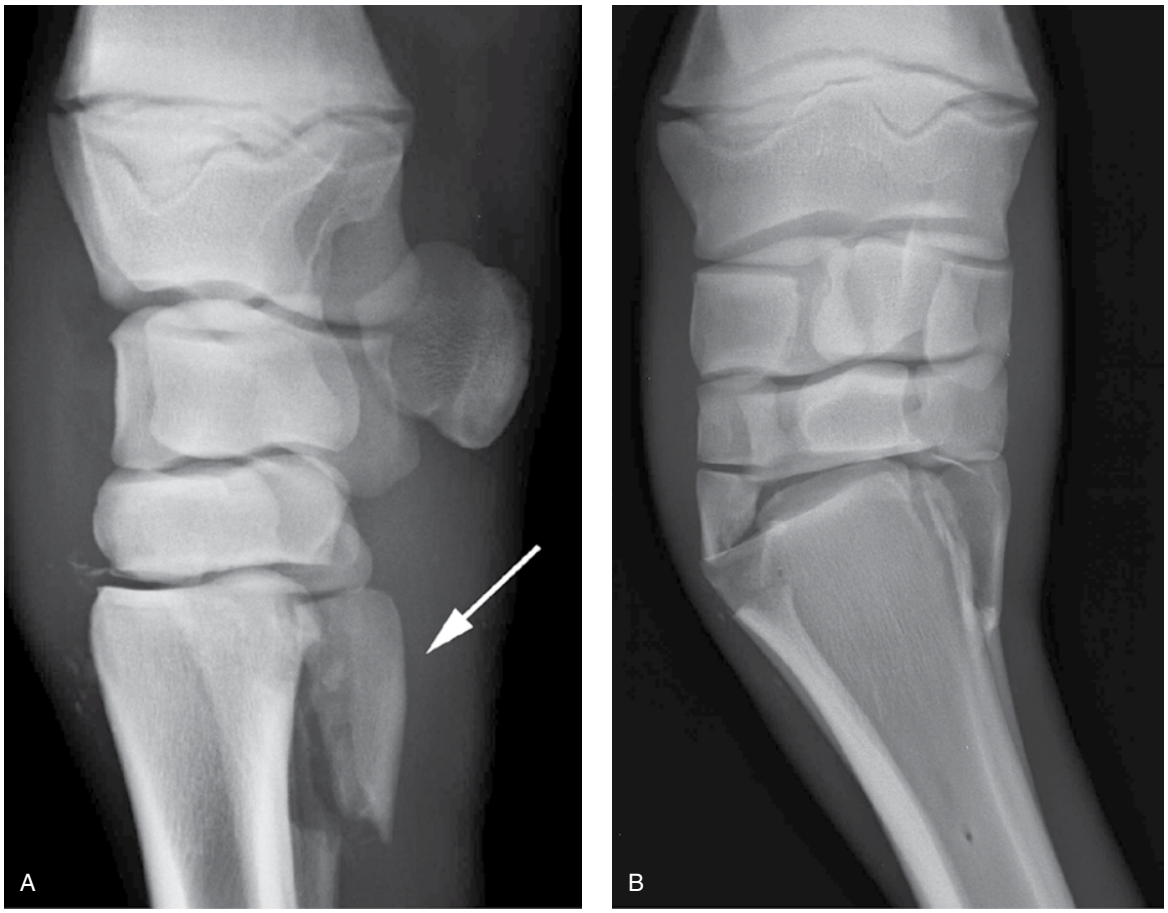


Figure 5.17. Radiographic images showing biaxial splint bone fractures (A, arrow) that led to instability and luxation of the carpus (B). *Source*: Courtesy of Dr. Gary Baxter.

Radiographs and ultrasound are often diagnostic, although in some partial tears the horse must be sedated and manual deviation of the carpus imposed to see the subluxation on radiographs (stress radiographs). In addition, dorsopalmar luxations can occur and radiographs should be assessed closely. Ultrasound is the best diagnostic method for characterizing full- and partial-thickness damage to the collateral ligaments, although with time, enthesophyte formation is usually appreciated radiographically.

Treatment and Prognosis

Treatment for complete luxations often involves placing the horse under general anesthesia to achieve reduction. Arthroscopic surgery may be helpful to debride damaged tissues if joint damage occurs. The luxation can be reduced if the carpal bones are not involved; otherwise, fractures may need to be debrided to facilitate reduction. Dorsopalmar luxations can be difficult to reduce, and sometimes manual fatiguing of the limb or surgery is needed to reduce the luxations. Once reduced, the limb can be put through rotational manipulation.

If the limb is deemed to be rotationally stable, then a tube cast can be applied from the proximal aspect of the radius to the distal third metacarpus.[13] If the limb is deemed to be rotationally unstable, a full-limb cast should be used. Foals are usually casted for approximately 4 weeks and adults for 6 weeks to allow fibrosis to occur in the collateral ligament area and joint capsule. The horse may be transferred to a bandage and/or splint for several weeks, but stall confinement for several months is necessary.

Partial or pancarpal arthrodesis may be needed to stabilize the limb for breeding and pasture soundness if multiple fractures are present (Figure 5.18).[22] However, even if the limb can be reduced adequately, chronic OA and pain may necessitate arthrodesis to control pain and prevent contralateral limb laminitis. Soft tissue lesions can be augmented with stem cells, platelet-rich plasma, or extracorporeal shockwave therapy to help stimulate healing. The prognosis is good for healing but guarded for athletic use.

SOFT TISSUE DAMAGE TO THE CARPUS

Collateral ligament injury is sometimes seen in the carpus. Although these may be difficult to diagnose acutely, improvement in ultrasound examination of these structures and the use of MRI have helped with early diagnosis. Some have described collateral ligament abnormalities in the carpus closely associated with OA;[30] so the problem is sometimes diagnosed well after OA has already been established.

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Figure 5.18. Lateral radiograph 1.5 years after the horse (Figure 5.17) underwent arthrodesis of the carpometacarpal joint using a locking plate. *Source*: Courtesy of Dr. Gary Baxter.

Injury to the intercarpal ligaments is common, especially in cases with osteochondral damage. The dorsomedial intercarpal ligament attaches to the distal radiocarpal bone in the area where osteochondral fragmentation typically occurs. Whitton et al. have shown that the dorsomedial intercarpal ligament resists dorsomedial displacement of the radiocarpal bone; therefore, it is likely that the stress at this site makes this area prone to damage.[123,125] Selway proposed that hypertrophy of this ligament could lead to disease, but Whitton et al. have shown no correlation between dorsomedial intercarpal ligament hypertrophy and osteochondral damage.[122,124,126]

The palmar intercarpal ligaments have been shown to provide considerable stability to the carpus. The medial and lateral palmar intercarpal ligaments allow abaxial translation of the carpal bones to dissipate axial forces through the carpus.[18] Whitton et al. found no correlation of tearing of the palmar intercarpal ligaments with the severity of disease other than the fact that severe tearing of these ligaments can lead to instability of the joint and hemarthrosis. Beinlich and Nixon reviewed the diagnosis and treatment of horses with avulsion fracture of the lateral palmar intercarpal ligament from the ulnar carpal bone[11,12] and found that the fragments were best demonstrated on the dorsopalmar and especially the dorsolateral palmar medial projections, which corresponded with arthroscopic findings in this group of horses. In addition, they found that arthroscopic removal of these fragments was more beneficial than rest alone, with 91% of those horses returning to their intended use.[11,12]

Enhanced diagnostic information can be achieved from MRI and CT arthrography.[45,89] On MRI, Nagy and Dyson characterized many intercarpal ligaments that cannot be identified or characterized using other imaging techniques. Gray et al. also showed that CT arthrography could be used to characterize the intercarpal ligaments and that it is an acceptable technique to diagnose lameness originating from the carpus.

OSTEOARTHRITIS

Etiology

OA is common in the carpus and can result from 1 of 2 syndromes. In young horses that are athletes, such as racehorses, stress-related bone damage—especially that which causes physical damage to the joint—can result in secondary OA; see Chapter 7 for details on stress-related bone damage. It is likely that the pathologic process that leads to this disease progresses to the point of articular cartilage damage and OA. The other form of OA occurs in horses that demonstrate insidious, progressive onset of disease regardless of their previous history. In either case, if the severity of damage can be stabilized, these horses are often clinically manageable for nonracehorse type of work. However, when the damage is progressive, it is possible that even pasture soundness is questionable.

Clinical Signs and Diagnosis

Whether the OA is due to previous injury or insidious in onset, the clinical response is similar. Horses with a previous history of disease can be monitored closely for radiographic evidence of advancing OA and treated. In insidious cases, often no problems are noticed until the limb begins to swell and lameness becomes obvious. In the latter cases, the farrier will often note that the horse begins to resent flexion and manipulation of the limb. Radiographs taken at this point often show some signs, and treatment can be helpful if started at that time.

Radiographic signs of OA may include mild osteophytes, enthesophytes, and osteochondral fragmentation early in the disease process (Figure 5.19). Subchondral bone sclerosis may be identified and mild joint space narrowing may be present. Severe changes can result from the progression of OA, leading to combinations of subchondral bone sclerosis and lysis.

Treatment and Prognosis

Some early forms of OA can be treated with rest and systemic nonsteroidal anti-inflammatory treatment. Stall confinement is rarely helpful in management of horses with carpal OA; in other species, including humans, strengthening of periarticular soft tissues is beneficial.[17] Paddock turnout seems to help these horses, although it is important to monitor their activity and limit their exposure to other horses that may stimulate excessive exercise.

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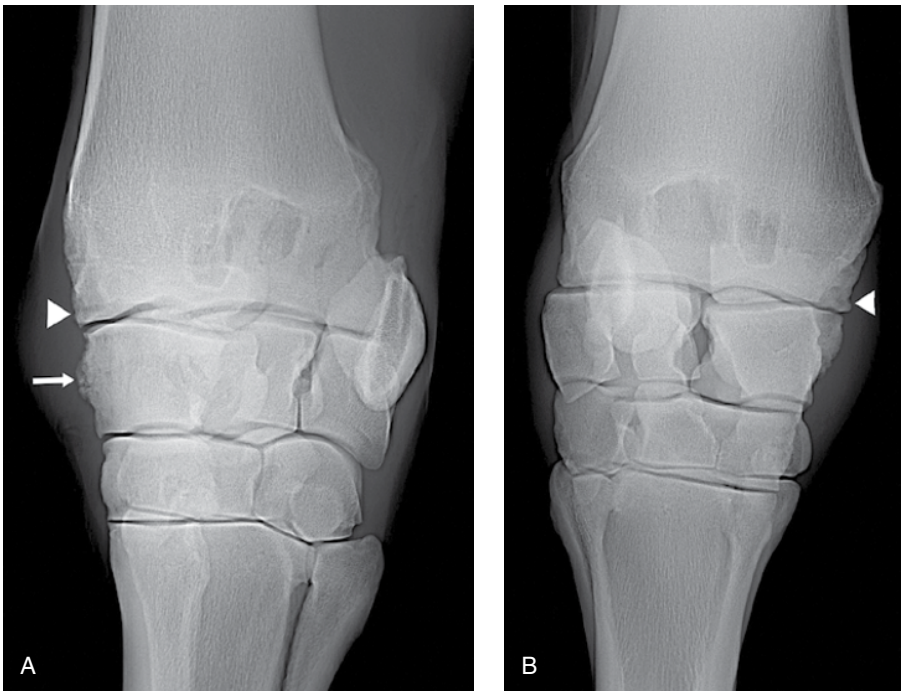


Figure 5.19. Radiographs demonstrating mild signs of osteoarthritis, including enthesophyte (A; arrow) and osteophyte (A, B; arrowheads) formation.

Surgery can be performed to remove osteochondral fragments or for augmentative therapies such as microfracture and resurfacing; however, the disease process will likely continue to progress postoperatively, and in most cases the goal of surgery is to help relieve pain and the severity of the disease.

Intra-articular medication is meant to treat the synovitis component that often leads to pain, but its efficacy for treating subchondral bone damage is questionable. Intra-articular corticosteroid and hyaluronic acid combinations may work for a short period of time but often stop working. Intra-articular hyaluronic acid and polysulfated glycosaminoglycan combinations or IRAP may be considered. In some cases, there is subjective evidence to show that intra-articular administration of stem cells may be helpful in managing horses with carpal OA. Although it is thought that only triamcinolone acetonide or betamethasone should be used in high-motion joints such as the carpus, methylprednisolone acetate may be the only thing that is effective in some cases of severe OA. Although this has been shown experimentally to result in progressive articular cartilage damage, in these cases management of pain is often considered the priority. It is sometimes difficult to determine the origin of the greatest site of pain in horses with carpal OA. In cases with severe osteophyte and enthesophyte formation and subsequent reduced range of motion, the author has occasionally used corticosteroids administered via regional limb perfusion of the carpus to help alleviate pain. Further studies are needed to evaluate the efficacy of this treatment, but in some cases it seemed to be effective.

Medial collapse of the carpal joints is not uncommon in severe cases of OA, and often dorsolateral break-over of the hoof in that limb is noticeable. This progressively worsens between trimmings; it is a subjective opinion that this can lead to worsening of pain. A mild lateral extension or a full shoe itself may help to alleviate some of this pain by slowing the dorsolateral break-over that occurs. In severe cases of OA, partial or pancarpal arthrodesis is often advocated to reduce the amount of chronic pain in the limb and prevent laminitis in the opposing limb.

CARPOMETACARPAL OSTEOARTHRITIS

OA of the carpometacarpal joint is a separate syndrome that predominantly involves mainly Quarter horses and Arabian horses.[73,96] The osteoarthritic changes manifest mostly at the articulation between the second carpal and second metacarpal bones and radiographically show osteoproliferative changes and joint space narrowing medially (Figure 5.20). Lameness is insidious in onset, but can progress rapidly. The etiology of this problem is unknown, but it has been suggested that an anatomic abnormality may exist between the second and third carpal bones.[73] Medical management of these cases can be frustrating over time because they typically become less responsive to treatment.[96] Similar to OA at other sites, medical management is worth attempting until there is concern that the lameness could contribute to opposing limb laminitis. At this point, a partial carpal arthrodesis could be performed. Partial carpal arthrodesis can be performed in one of two ways. Barber et al. evaluated a drilling technique for arthrodesis in 12 horses and found that it was considered successful in most cases.[8] However when evaluated experimentally, the same technique failed to produce consistent ankylosis in normal joints.[97] Consequently, some surgeons have advocated plate fixation to fuse the joint.

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OSTEOCHONDROSIS OF THE CARPUS

Osteochondritis dissecans has been reported in the carpal bones, although it is uncommon compared to other joints in the horse. Subchondral cystic lesions in some carpal bones are incidental and are often of questionable clinical significance. This is especially true with cysts within the second carpal bone in association with the presence of the first carpal bone and in the proximal aspect of the second metacarpal bone. However, subchondral cystic lesions in the radiocarpal bone and the distal aspect of the radius (Figure 5.21) are often clinically significant, and arthroscopic surgery and debridement are warranted if they are felt to communicate with the carpal joints. Simple drilling or transcystic screw could also be considered in these cases. Oftentimes, MRI or CT is needed to best characterize these lesions.

OSTEOCHONDROMA OF THE DISTAL RADIUS

Osteochondroma (OC) formation at the distal end of the radius is an uncommon condition causing lameness in the horse.[53,63,70,114] The growth most commonly occurs in adult horses adjacent to the physis at the caudal distal aspect of the radius.[47,112,113] Although very uncommon, the cranial aspect of the radius also can be involved.[104] Radiographically and histologically, these new bone growths appear much like those reported for hereditary multiple exostosis. However, unlike hereditary multiple exostosis, they appear as singular lesions or affect only a few other long bones, rather than numerous growing bones. Solitary OCs have been reported in two foals; the distal palmar aspect of the middle phalanx was affected in one, and in the other the calcaneus was involved.[24,32] The reader is referred to Chapter 7 for more details.

Etiology

Although a single dominant autosomal gene is responsible for the development of multiple exostosis in humans and horses,[107] the genetic implications for isolated OC remains unclear. In humans, solitary OCs are not considered to be inherited.[51] It is postulated that metaplastic cartilage foci develop in the metaphysis and distal diaphysis from abnormal growth of the periosteum. As the cartilage grows, it undergoes endochondral ossification similar to that occurring at the physis. The developing exostosis, which is continuous with the cortex of the bone and surrounded by a cartilage cap, is called an osteochondroma.

Nixon et al. have reported on a series of cases in which exostoses of the palmar aspect of the closed physis were removed arthroscopically. In these cases, although they appeared similar to OC, they were histologically different.[91]

Clinical Signs

Affected horses often present with a history of intermittent lameness that increases with exercise. An obvious



Figure 5.20. A DP radiograph demonstrating collapse of the medial aspect of the joint space and osteoproliferation typical of OA of the carpometacarpal joint. *Source*: Courtesy of Dr. Gary Baxter.

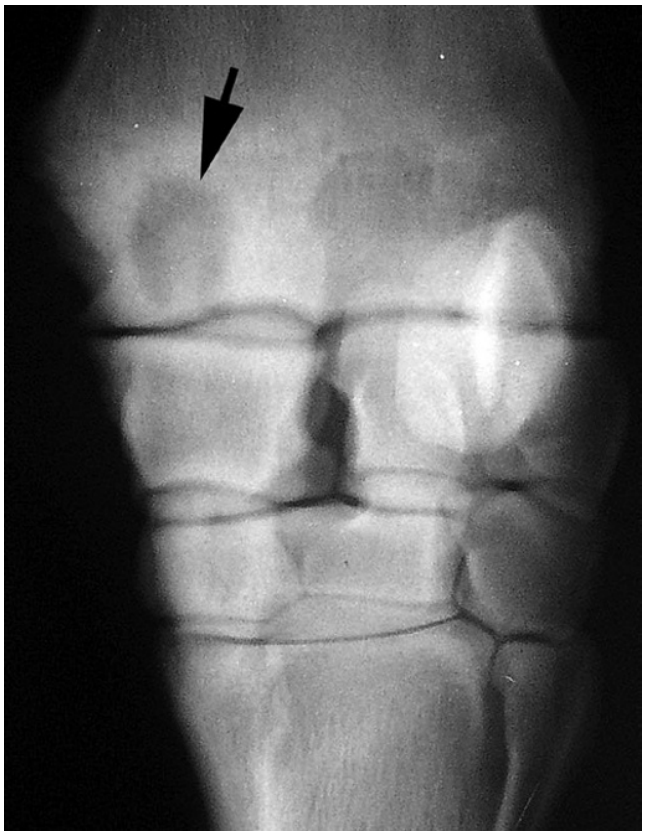


Figure 5.21. A radiograph of a subchondral cystic lesion of the distal radius (arrow). *Source*: Courtesy of Dr. Gary Baxter.

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swelling of the carpal canal sheath cranial to the ulnaris lateralis is often present and may be visible medially or laterally on the limb (Figure 5.22). In some cases the swelling can be extensive. At exercise a moderate lameness graded 1–2 out of 5 is commonly observed.[112] Palpation of the caudodistal aspect of the radius with the limb held flexed at the carpus allows the examiner to feel the bony protuberance in some cases. Deep palpation of the site is often painful, resulting in limb withdrawal from the pressure. The range of carpal flexion is usually less than normal, and considerable pain is elicited with rapid carpal flexion. A carpal flexion test usually exacerbates the lameness.

Diagnosis

Radiography is necessary to diagnose the condition and its location. In most cases the OC is located on the caudomedial aspect of the distal radius adjacent to the physis; however, smaller OCs have been observed on the caudolateral aspect of the distal radius as well. Radiographically, these lesions appear as conically shaped bony protuberances with an outer cortex and inner medullary cavity (Figure 5.23). The size of the OC and degree of ossification are variable. Ultrasonography can also be used to determine the presence of deep digital flexor tendinitis.[121] Considering in racehorses associated flexor tendon damage is present in almost all cases, ultrasonographic examination is essential.[129] Intrathecal anesthesia is occasionally needed to confirm the clinical significance of the lesions.

Treatment

Although the instillation of corticosteroids into the tendon sheath temporarily resolves the tenosynovitis, the clinical signs usually reoccur.[121] Presently surgical excision of the OC is the treatment of choice and it is curative in most cases. Although excision of the OC via an open lateral or medial approach has been described,[47,63,70] the removal using an arthroscopic technique is currently recommended.[112,113,129] Both lateral and medial endoscopic approaches have been described; the author prefers the lateral approach. The exact technique can be found in other references.[81]

Prognosis

The prognosis for surgical excision of solitary OCs is good for return to performance. In one report, follow-up on two horses after 1 and 2 years found that the horses were free of lameness and no distention of the sheath was apparent.[112] In another report, follow-up 4 months after surgery found the horse to be free of lameness and no swelling in the carpal canal.[113] No lameness has been observed in two other cases at 4- and 12-year follow-up, and the bone excision sites remained nonreactive radiographically. Nixon et al. found that all 10 horses in their series of exostoses returned to intended use, with only 1 horse requiring additional medical therapy.[91] In yet another study in 22 Thoroughbred racehorses, all horses went back to work after surgical removal and debridement of soft tissue damage.[129]



Figure 5.22. Carpal canal swelling visible (arrow) and palpable on the lateral aspect of the limb.



Figure 5.23. Lateral radiograph of the carpus demonstrating an osteochondroma on the caudal aspect of the distal radius (arrow).

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DESMITIS OF THE ACCESSORY LIGAMENT (RADIAL OR SUPERIOR CHECK LIGAMENT) OF THE SUPERFICIAL DIGITAL FLEXOR TENDON

Etiology

The accessory ligament of the superficial digital flexor tendon (AL-SDFT) is a strong fibrous band that originates from the distal caudomedial surface of the radius and joins the SDFT at that level.[42] Until recently sprain to this structure has been poorly defined, and many conditions associated with the caudal aspect of the radius and carpus were attributed to this condition including (1) lowering of the angulation of the accessory carpal bone, (2) enthesophyte formation at the distal caudomedial aspect of the radius, (3) cranial displacement of the proximal end of the radius, and (4) alterations in the antebrachiocarpal (radiocarpal) joint capsule on its dorsal surface.[42,74]

Sprain of the AL-SDFT has been more clearly defined with the improvements in ultrasound resolution and the advent of MRI.[27,28] The condition occurs most commonly in young adult horses with a high level of physical activity. In a report on 23 horses with abnormal ultrasonographic findings in the AL-SDFT, 11 of 23 were racehorses, 9 of 11 were sport horses, and the remaining were pleasure or instruction horses.[28] Presumably, extreme hyperextension of the fetlock in conjunction with hyperextension of the carpus can cause a sprain of the AL-SDFT.[66,74]

Clinical Signs

Affected horses have a history of starting races and workouts quite well but are reluctant to really sprint out. Rarely do they attain their previous performance levels.[42,74] A visible swelling of the carpal sheath is observed in some cases. At a walk, a gait impediment characterized by a lateral floating placement of the foot just before it contacts the ground may be observed. The toe and heel are placed on the ground at the same time, much like a horse walking down an incline. In a review of 30 cases, this gait alteration was most commonly observed with radiographic lesions on the caudomedial distal aspect of the radius associated with the origin of the AL-SDFT.[42] In acute cases the carpal sheath is frequently distended and variably painful to digital palpation.[42] Simultaneous trauma to the SDFT may also occur, resulting in a painful swelling.[54]

Diagnosis

In acute cases, radiographs of the caudal aspect of the distal end of the radius are usually negative. However, in chronic cases, radiographs of the distal radius may reveal enthesophytes associated with the attachment of the AL-SDFT. Interestingly, in a study in which radiographs were taken on horses with abnormal ultrasonographic findings in AL-SDFT, only 4 of 18 had an abnormal profile at the caudal distal aspect of the radius, and 14 of 18 were considered normal.[27] This underscores the value of ultrasound examination in the diagnosis of desmitis of the AL-SDFT.

Ultrasound findings associated with injury of the AL-SDFT include thickening, hypoechoic defects, and alteration in fiber pattern alignment.[27] Injury to the AL-SDFT can also be accompanied by abnormal findings involving one or more of the structures in the carpal canal such as synovial effusion within the carpal sheath, tendinitis of the SDFT, distension and thickening of the retinaculum flexorum, tenosynovitis of the sheath of the tendon of the flexor carpi radialis muscle, and/or injury to the proximal attachment of the suspensory ligament (third interosseous muscle). Characterization of this additional damage is important for determining therapy and prognosis.

Treatment

In the acute cases exhibiting carpal sheath distension, needle drainage and the injection of a corticosteroid and hyaluronic acid are recommended.[27,28] Rest in a stall for 4–6 weeks is recommended with hand-walking exercise beginning at the third week. Systemic nonsteroidal anti-inflammatory drugs may be indicated for associated problems and are administered as needed. Pressure/support wraps are applied and maintained for 2–3 weeks. Following stall rest the horse should be confined to a small run for another 6 weeks. Recheck ultrasound examination should be done at this time to assess healing. Generally, affected horses can return to performance in 4–6 months.[28] Arthroscopic examination of the carpal canal can be performed to aid in characterization of the damage and treatment if necessary, and tenoscopic release of the carpal flexor retinaculum can be performed if needed.

Prognosis

The prognosis appears to depend on the severity of the injury, the intended use of the horse, and whether the SDFT is involved. In one study, 8 of 13 sport horses were able to return to their previous level of activity, whereas only 7 of 16 racehorses returned to racing at their previous level.[29] In another report, of eight horses suffering from injury to the AL-SDFT alone, without tendinitis of the SDFT, six returned to their previous level of performance within 4–6 months.[28] Tenoscopic release of the carpal flexor retinaculum has been useful for resolving lameness in two horses.[117]

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THE ANTEBRACHIUM, ELBOW, AND HUMERUS

Jeremy Hubert

FRACTURES OF THE RADIUS

In an original retrospective study of radial fracture configurations, comminuted fractures were most common (21 of 47), followed by oblique fractures (12 of 47), transverse fractures (7 of 47), and physeal fractures (7 of 47). Open fractures usually involve the medial surface of the antebrachium, where there is minimal soft tissue coverage (Figures 5.24–5.26)[72] A more recent study of 54 cases of radial fractures reported that 24% were open and comminuted, 26% were incomplete or fissure fractures, and 50% were deemed repairable as oblique (10), transverse (7), Salter–Harris type II (5), spiral (3), and combined fractures of the radius and ulna.[78] Incomplete or fissure fractures are relatively common fractures of the radius; the majority of which occurred along the longitudinal plane.[20] Other uncommon radial fractures include stress fractures and smaller articular fractures involving either the proximal or distal articulation.[52] Radial stress fractures compose a small percentage of reported stress fractures. In a recent study of stress fractures in a racing Thoroughbred population, only 4.3% of stress fractures involved the radius.[53]



Figure 5.24. A complete oblique fracture of the radius extending from the mid-diaphysis to the metaphysis. *Source*: Courtesy of Dr. Andrew Lewis.

Etiology

Radial fractures usually result from a high-impact blunt trauma such as a kick from another horse. A controlled postmortem study to evaluate fracture configurations of the radius after a simulated kick on the medial aspect of the radius resulted in the majority of the bones sustaining a fissure fracture (incomplete fracture) that ran longitudinally along the diaphysis. The next most common configuration was an oblique fracture, sometimes with a fissure component, followed by a butterfly fragment fracture.[30] The medial aspect of the bone was chosen in the postmortem study to emulate the more common finding of a kick to the medial side; a smaller retrospective study of radial fractures revealed the



Figure 5.25. A cranial to caudal view of a distal diaphyseal comminuted fracture of the radius. *Source*: Courtesy of Dr. Martin Waselau.

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majority occurred from a kick to the medial aspect of the bone (Figure 5.26).[52] Age may play a role in fracture configuration; comminuted or butterfly fragment fractures are usually noted in older horses (older than 2 years), whereas simple oblique fractures occurred in younger horses. Transverse fractures were exclusive to foals less than 6 months of age (Figure 5.27).[72]

Clinical Signs

Horses with complete displaced fractures present with a non-weight-bearing lameness, varying degrees of swelling in the antebrachium, and instability associated with the fracture site. Crepitation may be felt and pain elicited when the distal limb is manipulated. Wounds and penetration of the skin from the fracture on the distal medial side of the antebrachium are common.

Horses that present with complete or incomplete nondisplaced fractures are usually very lame but are willing to bear some weight on the limb. Instability and crepitation are not present; however, there is usually some swelling of the antebrachium, and pressure applied to the region is often painful.[7] Wounds at the site of traumatic impact are often present on the medial distal aspect of the antebrachium (Figure 5.26).

Stress fractures are difficult to identify on physical signs alone. Horses generally present with a history of an acute onset of lameness that subsides with rest and reoccurs with exercise. Horses are often mildly lame at examination and shoulder flexion may worsen the lameness. These fractures can also occur bilaterally.[52]

Diagnosis

The presence of a displaced radial fracture is confirmed on radiographic examination. Several views are often needed to evaluate the configuration and extent of the fracture. Computerized tomography (CT) may be useful to help determine the ideal internal repair method if a complex comminuted fracture is to be assessed completely.

Acute nondisplaced fractures may be difficult to diagnose initially on radiographic exam. If a nondisplaced fracture is suspected, the horse should be cross-tied in a stall to prevent it from lying down, and repeat radiographs should be taken in 2–4 days. This time period usually allows recognition of the fracture lines. Alternatively, nuclear scintigraphy can be used to assist in the diagnosis.

Stress fractures are usually best diagnosed by nuclear scintigraphy where a focal increased uptake of the radionuclide in the midshaft of the radius is usually found (Figure 5.28).[52] Confirmation with radiography can be performed at a later date. Evidence of focal endosteal or periosteal reaction indicates a healing stress fracture that will correlate with the focal area of increased uptake. Alternatively, quantitative scintigraphy can be performed.[85] This enables the clinician to assess the degree of injury if there are no initial radiographic findings as well as later during the convalescent period when the quantitative findings can be used to accurately determine a date to return to training or work.



Figure 5.26. Wound on the medial aspect of the antebrachium from a kick injury. This horse had a concurrent distal radial fracture. Radiographs of the radius should always be taken in horses with these injuries.



Figure 5.27. A transverse complete midshaft fracture of the radius of a foal. *Source*: Courtesy of Dr. Martin Waselau.

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Figure 5.28. A nuclear scintigraphic examination of increased uptake of radioactive isotope in the distal radius, indicating a stress fracture of the metaphysis. *Source*: Courtesy of Dr. Dan Burba.

Treatment

First Aid

Immediate immobilization of a displaced radial fracture is very important (see musculoskeletal emergencies in Chapter 12). Appropriate attention to any open wound to address contamination and to reduce further soft tissue damage is necessary. With closed displaced fractures, application of appropriate external coaptation can prevent the fracture from becoming open. A heavy layered cotton bandage is firmly applied from the hoof to as far proximal on the antebrachium as possible. Two splints, one placed lateral and one placed caudal to the limb, with the lateral splint extending from the ground to the withers and the caudal splint extending from the ground to the olecranon process, is recommended.[10,93] Nonelastic adhesive tape or 1–2 rolls of fiberglass cast material are used to affix the splints to the bandage. The portion of the lateral splint that extends proximal to the bandage should contact the brachium when the limb is in a weight-bearing position.[90] This proximal application of the lateral splint provides counterpressure to prevent the limb from abducting with weight-bearing. This approach generally stabilizes the limb enough to allow the patient to be transported for treatment without running the risk of further injury to the fracture site.

Nondisplaced Fractures

Fractures that are nondisplaced or incomplete with minimal displacement are often candidates for conservative treatment. This involves stall confinement for up to 3–4 months.[7,20] It may be necessary to attach the horse to a wire to keep the horse from laying down to initially prevent catastrophic breakdown of the fracture. Horses should be closely watched for tolerance of being tied. NSAIDs are initially administered; these can be weaned off over time. Short hand walks can be initiated after 90 days. A total of at least 6 months before a return to intended use should be considered.

Displaced Fractures

Internal fixation of complete displaced fractures of the radius is the preferred method of treatment. The use of transfixation casts is generally considered for open contaminated fractures in which the chances of successful plate fixation are poor because of the risk of infection or when there are economic constraints. The application of a full-limb cast alone for displaced fractures of the radius is not acceptable in most cases because the humeroradial joint cannot be adequately immobilized. If a cast is applied, it frequently results in increased rotational forces on and movement at the fracture site. In addition, the more proximal the fracture, the greater the chances that the cast will cause a pendulum effect to the distal limb. This causes more tissue damage and results in a failure of the fracture to heal.[27] However, cast application alone has been used to successfully treat some distal radial fractures when there are economic constraints.

A combination of a full-limb cast and Thomas splint has also been recommended for some uncomplicated midshaft radius fractures in young horses.[27,67] Bandage splints have been used to treat a few horses with complete fractures of the distal radius. In one report, two horses with complete fractures (one nondisplaced, one displaced) involving the distal half of the radius were treated successfully with a modified Robert-Jones bandage with the addition of light strong splints.[68] Healing occurred in 14–16 weeks and both horses were able to return to usefulness.

A transfixation pin cast using positive profile, centrally threaded, one-fourth-inch stainless steel pins placed transcortically in a sagittal plane at 30° to each other is best suited for fractures in the distal third of the radius (Figure 5.29).

A single-plate application on the cranial or craniolateral surface is only recommended in foals less than 250 kg that have a transverse mid-diaphyseal fracture with an intact caudal cortex (Figure 5.30).[90] In foals, it is preferential not to bridge the physes and to not engage the ulna because it could result in elbow dysplasia and arthritis due to the differential growth rate of the ulna and radius.[79] Internal fixation in adult horses for displaced fractures generally has an unfavorable prognosis. In larger foals and adults, two plates are used: one is placed as a tension band on the cranial surface, and the other is placed as a neutralization plate on the lateral or medial surface 90° to the other plate (Figure 5.31).[5] The use of a 5-mm locking compression plate (LCP) or a dynamic condylar plate (DCP) as one of the plates is recommended.[47,90] In a report of 54 radial fractures, double plating was used in all adult horses. A variety of plates were used including DCP, limited contact dynamic compression plate (LC-DCP), dynamic condylar screw (DCS), or the LCP. Although there was no correlation as to what would be the best plate, these authors believed that the use of LCP would ultimately provide a better outcome based on the improved stability shown in

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Figure 5.29. Transfixation pins applied to the proximal radius prior to applying a full-limb cast to treat a comminuted distal radial fracture.

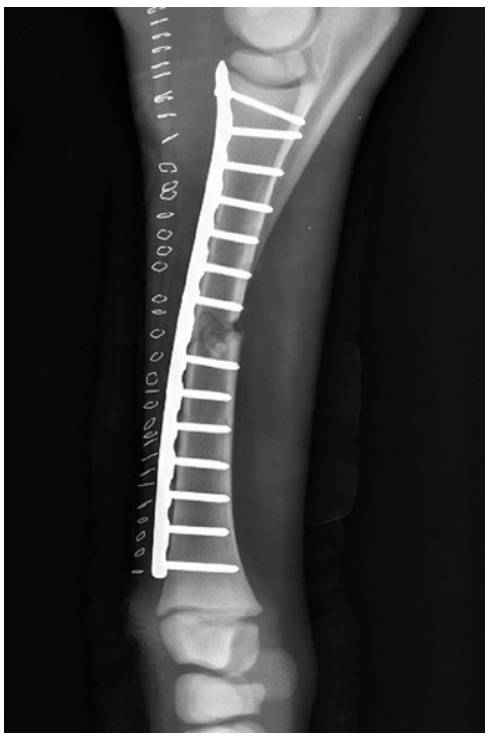


Figure 5.30. Single DCP application on the cranial surface of the radius to repair a transverse mid-diaphyseal fracture in a foal weighing less than 250 kg.



Figure 5.31. Two 5-mm locking compression plates placed on the dorsal and lateral aspects of an obliquely fractured radius. *Source*: Courtesy of Dr. Dan Burba.

biomechanical testing. Plate luting with antimicrobial-impregnated PMMA and bone graft application is advisable[90] although in a recent study there was no correlation with grafting, antibiotic-impregnated PMMA, intravenous regional limb perfusion, and survival to discharge, but there was a correlation with surgical time.[78]

Physeal Fractures

Salter–Harris type I and II fractures of the proximal physis are encountered although infrequently (Figure 5.32). They may be accompanied by a fracture of the ulna, and a degree of radial nerve trauma may be encountered. Minimally displaced fractures may be treated conservatively in select foals (Figure 5.32). Repair generally involves a single-plate fixation of the ulna with the screws at the level of the proximal physis and lower, engaging the radius. A second plate can be placed laterally with the proximal screw in the physis, although this is usually unnecessary.[90] In smaller foals, the repair can be performed by transphyseal screws, and a wire fixation instead of a lateral plate can be placed alone. There is minimal growth from the proximal physis due to the damage, and luxation of the elbow joint is unlikely. Type III fractures that are nondisplaced are rested, whereas displaced type III fractures are repaired with screws placed in lag fashion in combination with a tension band application.[72,90]

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Figure 5.32. A Salter–Harris type II fracture of the proximal radius (arrow). This foal was treated conservatively.

Prognosis

Nondisplaced complete and incomplete fractures appear to have a good prognosis with conservative treatment.[7,20,68,78] Stress fractures of the radius are expected to have a good prognosis if adequately rested, similar to reported results of tibial and humeral stress fractures.[67]

The prognosis for displaced radial fractures depends on the age and weight of the horse, its temperament, and the fracture type. Although there are a few reports of successful treatment of displaced radial fractures with internal fixation in mature horses, most adult horses have an unfavorable prognosis for survival regardless of the treatment selected.[72,78,90] In one report on 47 cases of radial fractures, successful treatment (82%) only occurred in horses younger than 2 years of age with an overall rate of 64% survival to hospital discharge.[72] In the more recent report of 54 cases, the prognosis for survival to discharge for young horses with open reduction and internal fixation (ORIF) techniques was good but the converse was true for older horses with negative correlations associated with surgical site infection (SSI) and increased surgical time (>168 minutes).[78] In one retrospective study of horses weighing more than 300 kg presenting with radial fracture, 9 of 15 were treated with internal fixation, and only 2 horses were discharged from the hospital to resume their former activities.[5] Reasons for nonsurvival included primary implant failure, infection causing implant failure, and support limb laminitis. Comminuted fractures were associated with the highest incidence of fatal complications. In the more recent study, 27 horses underwent ORIF and 15 survived to hospital discharge (56%), of which only three were adult horses. Failure in the surgically managed adult horses was a result of SSI and resultant construct failure in 13.[78]

Displaced closed simple fractures of the radius in foals have a favorable prognosis. Physeal and transverse mid-diaphyseal fractures have an excellent prognosis. In one report, 2 of 2 foals with Salter–Harris type I and II proximal physeal fractures and 6 of 7 foals with mid-diaphyseal transverse fractures were treated successfully. Treatment of proximal oblique fractures in 3 of 4 foals also had an excellent outcome.[72]

THE ELBOW

Fractures of the Ulna

Ulna fractures are a relatively common fracture in younger horses. In a retrospective study of 49 ulnar fractures, 79% occurred in horses less than 2 years of age.[92]

Ulnar fractures are classified as types I through VI (Figure 5.33). Types I and II are Salter–Harris types, fracturing through the apophysis of the olecranon tuberosity in horses that are usually less than a year old. Type I is more common in neonates, whereas type II is more common in older foals. The incidence of these fractures is reduced as this physis closes, starting around 15 months of age. Types III through VI involve the diaphysis of the olecranon (Figure 5.34). In adults the fractures often involve the articular surface and are displaced due to contracture of the triceps apparatus.[19,21,24,92] Displacement is relative to the level of fracture on the ulna; fractures distal to the level of the elbow joint have less distraction due to the ligaments between the radius and ulna.

Etiology

Direct impact or trauma is the most common cause; however, in foals, type I and II fractures can occur from excessive tensile load of the triceps apparatus. Kick injuries appear to be a common cause of ulna fractures in adults and may be associated with wounds on the lateral aspect of the elbow.

Clinical Signs

Fractures of the ulna usually result in an acute non-weight-bearing lameness with a classic dropped elbow appearance (with the carpus flexed and the horse not bearing any weight on the limb). Nondisplaced fractures may initially show the dropped elbow appearance but will show progressive weight-bearing of the limb. Varying degrees of soft tissue swelling, crepitation, and skin wounds can be apparent. Limb manipulation is resisted, and efforts to get the horse to load the leg, even with a splint in place, are unsuccessful until the triceps apparatus has been repaired.

Diagnosis

Diagnosis is based upon the appearance of the horse, limb palpation, and radiographs. Differential diagnoses may include radial or humeral fractures, radial nerve

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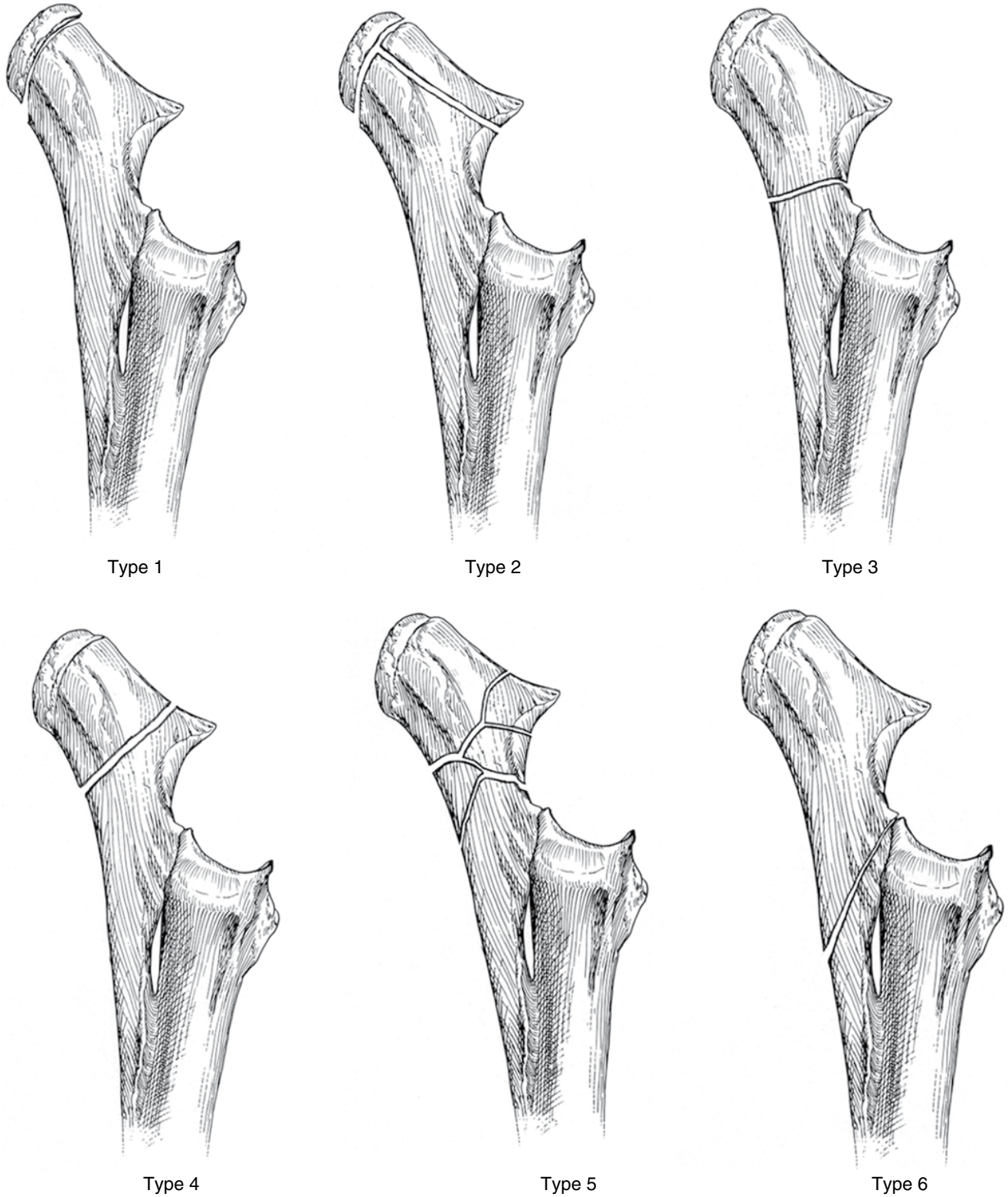


Figure 5.33. Classification of equine ulnar fractures.

paresis, or joint sepsis until a radiographic study has been performed. This should include lateral and craniocaudal views to provide the configuration of the fracture. In cases of a type I fracture in foals, it may be necessary to take a flexed view to provide distraction of the fragment to identify the fracture correctly.[69]

Treatment

Fracture cases that include nondisplaced, nonarticular ulna fractures or severely comminuted are candidates for conservative treatment (Figure 5.35). Horses with articular fractures with minimal displacement

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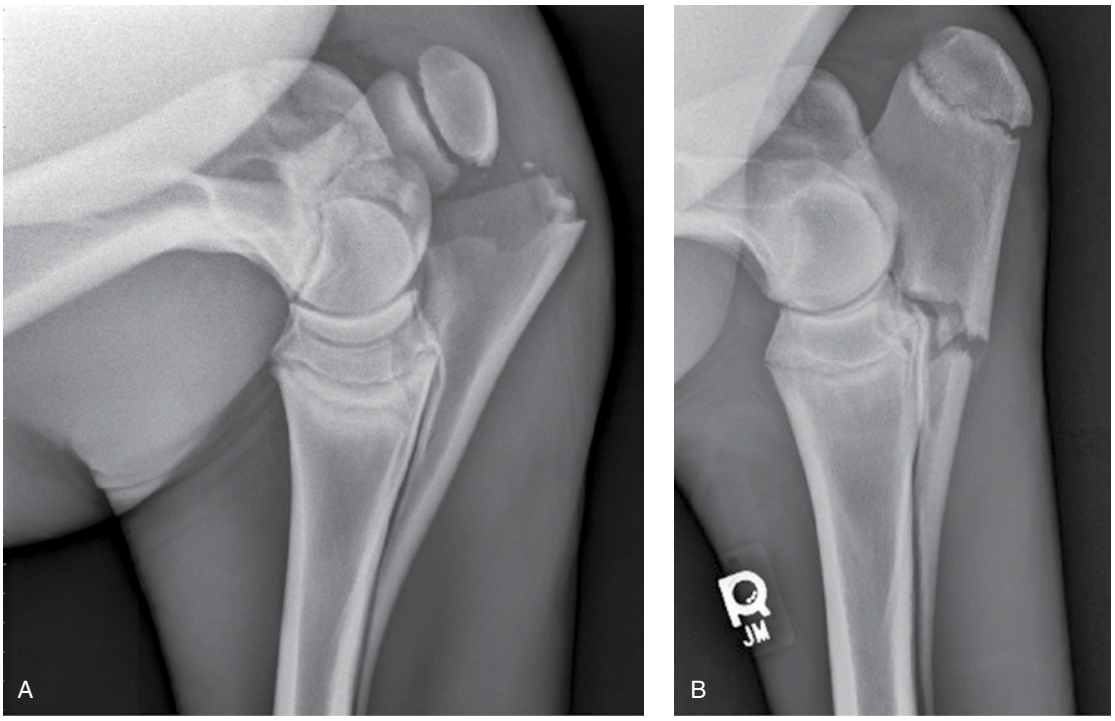


Figure 5.34. (A) Type III ulnar fracture. *Source*: Courtesy of Dr. Lori Gaschen. (B) Type VI ulnar fracture. *Source*: Courtesy of Dr. Martin Waselau.

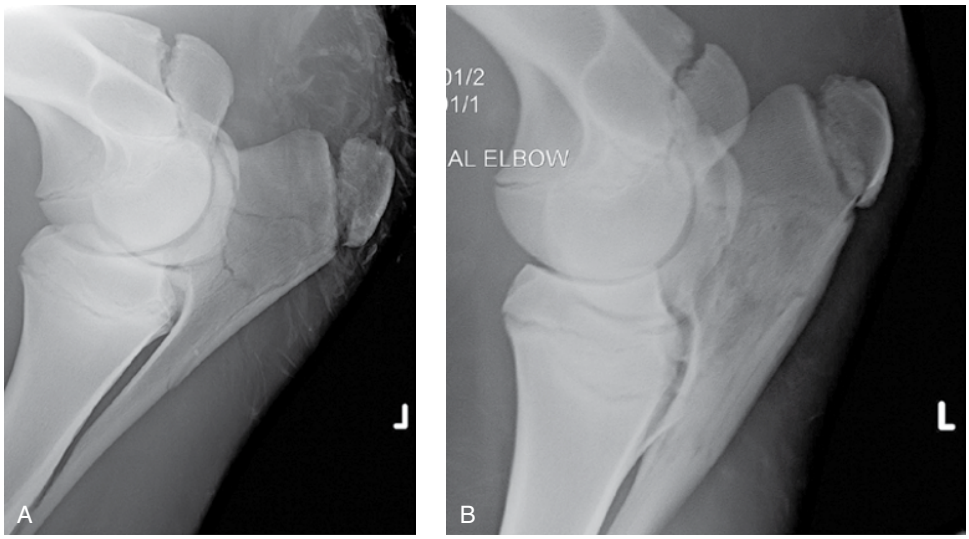


Figure 5.35. Comminuted ulna fracture in a young foal (A) that healed following conservative treatment (B).

also may be treated conservatively; however, better results are obtained with internal fixation to maintain joint congruity. Conservative treatment includes bandaging the limb with a caudal splint incorporated to the level of the proximal antebrachium (Figure 12.8). Complete stall rest is necessary, and a bandage and splint are maintained for at least 6 weeks or until a substantial improvement in the lameness is noted. NSAIDs are administered in the early stages. Repeated radiographic studies can be performed to ensure fracture healing and that progressive displacement is not occurring.[92]

Internal fixation of ulna fractures is performed in two basic manners. In young horses (younger than 6 months), the repair may be performed using a combination of screws, pins, and tension band wires, or plating (Figures 5.36 and 5.37). Distracted type I and II fractures require contouring the plate over the top of the olecranon. In younger foals a hook plate can be applied, or in even younger foals a tension band device with a pin or screw can be placed through the proximal epiphysis of the olecranon and a tension band wire applied and twisted about a screw placed caudally and distally in the ulna.[14,63]

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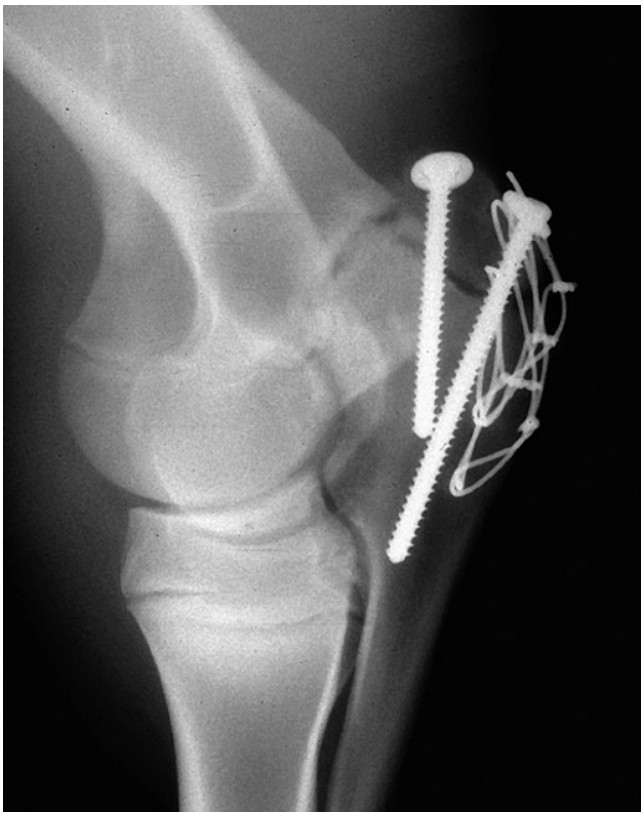


Figure 5.36. Two 4.5-mm cortical screws with washers and figure-8 wires used to create a tension band for a type I ulnar fracture. *Source*: Courtesy of Dr. Gary Baxter.



Figure 5.37. Type III ulnar fracture repaired with a narrow DCP applied to the caudal aspect of the ulna. The fracture was in an older horse; therefore, the distal screws penetrated the radius. *Source*: Courtesy of Dr. Katie Seabaugh.

In older horses, a caudal application of a plate using the tension band principle is necessary.[12,18,19,57] A controlled study of different repair types in adult cadaver limbs showed that DCP application was more biomechanically sound than pins and wires.[33] Plates are applied to the caudal aspect of the olecranon and ulna to counteract the tension of the triceps brachii muscle (Figure 5.37). The plate may be contoured over the top of the olecranon if the fracture is proximally located. Depending upon the size of the horse, a narrow or wide DCP can be applied; a broad DCP is applied in horses greater than 500 kg.[65] More recently the LCP has been used in repairs of the olecranon and may be indicated if possible.[37,47] In most adult horses the 4.5-mm-narrow LCP is likely to be adequate.

Prognosis

The prognosis for conservative management of nondisplaced fractures and nonarticular fracture types I and IV of the ulna is considered good.[92] Nondisplaced articular fractures, especially the type VI fracture, also respond well to conservative treatment. One retrospective study reported 70% of affected horses becoming sound.[92]

The prognosis for internal fixation of displaced types III, IV, V, and VI is also considered to be good.[19,21,24] Internal fixation of type I and II fractures in younger foals has a good prognosis when a hook plate is used, with 7 of 10 foals becoming athletes.[63] Type II fractures in young horses also do very well when repaired with DCP, with 16 out of 24 horses returning to athleticism.[81] In another study of pins and tension band wiring, 13 of 22 horses were reported to be athletically sound, and 18 fractures were considered to be repaired successfully.[61] In a recent prospective study using LCP in 16 horses with 18 fractures, complete fracture healing was achieved in 13 horses. Twelve fractures were type II and six type IV. Post-discharge complications of implant infection and lameness were resolved with implant removal.[37]

Luxation and Subluxation of the Elbow Joint

Subluxation or luxation of the radiohumeral joint with or without a concomitant fracture has been reported, although it is an uncommon injury.[17,42,71,73,82] Subluxation commonly occurs with rupture or partial tear of the medial collateral ligament.[76] Complete luxations often occur in conjunction with a fracture[46,82], but luxation without fracture has also been reported (Figure 5.38).[17,42]

Etiology

Severe, forceful limb abduction such as getting a leg caught in a fence is often associated with subluxations of the elbow, whereas severe falls have been reported to cause complete luxations.[17]

Clinical Signs

Swelling in the axilla of the affected side and a non-weight-bearing lameness are the commonly reported

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Figure 5.38. Luxation of the elbow joint with the humerus displaced medially. No fracture was present. *Source*: Reprinted with permission from Rubio-Martinez et al.[71]

signs for a subluxation. Palpation of the medial aspect of the joint and manipulation elicit a painful response, and limb instability may be noticeable. Signs associated with partial rupture of the medial collateral ligament may be less obvious because there is less inherent instability. Swelling may still be noticeable on the medial aspect of the joint. Joint effusion is usually noticeable laterally along with a grade 2–3 of 5 lameness. Flexion or abduction exacerbates the lameness.[76]

A complete luxation usually causes severe swelling and discomfort, with horses unable to bear weight nor extend their limb. This will not resolve until correct alignment and stabilization is established.[76]

Diagnosis

A definitive diagnosis is made with radiographic examination. Subluxation may require stressed views, and chronic cases may reveal evidence of osteoarthritis and an enthesiopathy of the medial collateral ligament. Stressed views are performed by manipulating the limb to exacerbate the subluxation, in this case by stabilizing the humerus and applying pressure to the radius in a medial or lateral direction.

Treatment

Closed reduction and conservative management has been achieved in select cases,[17,42] but more commonly, internal fixation is required because conservative management fails. Conservative management usually results in continued instability and resultant osteoarthritis.[42] Attempts to repair the collateral ligaments in complete luxations have been attempted.[71] Prosthetic collateral ligaments have been made by placing screws in the medial and lateral epicondyles of the distal humerus and the proximal tuberosities of the radius near the attachment of the collateral ligaments and wrapping surgical wire around them in a figure-8 pattern to emulate the ligament.[71]

Luxations in conjunction with an ulna fracture have been reported with no attempt to repair the collateral ligaments. The fracture was repaired with a broad DCP placed on the caudal surface of the ulna. Stability was attributed to the interdigitation of the three bones of the elbow joint.[82] With any repair attempt, significant distraction is necessary to aid the replacement of the luxation; in one case a small (1 cm) piece of the anconeal process was removed to assist in reduction.[71] Removal of the anconeal process should not affect prognosis.[2,39]

Prognosis

The prognosis for subluxation of the elbow appears to be guarded to unfavorable to return to performance, depending on the degree of injury to the medial collateral ligament.[76] Generally, a partial tear injury is expected to have a better prognosis, and some of these horses may be able to return to light work.

The prognosis for complete luxation is generally considered guarded[71,73,76]; however, there are at least two reports of repairs, and the horses were considered sound 6 and 14 months after surgery; the former horse had a concomitant ulna fracture.[17,82]

Subchondral Defects and Subchondral Cystic Lesions (SCLs) of the Elbow

Subchondral defects and subchondral cystic lesions (SCLs) of the elbow joint are uncommon, although they have been observed in a wide variety of horse breeds of all ages.[8,36,95] SCLs typically communicate with the joint, and they most commonly occur on the medial side, involving either the proximal medial radius or distal medial condyle of the humerus (Figure 5.39).[36] Although uncommon, SCLs can occur bilaterally.

Etiology

Proposed causes for subchondral defects and SCLs include osteochondrosis at a weight-bearing location in the joint and trauma. Trauma is perceived to occur by a concussive event, causing damage to the cartilage and subchondral bone plate, and the resultant pressure and presence of synovial fluid developing a cystic lesion.

Clinical Signs

Horses often present with a history of an acute onset of lameness that may wax and wane with use.[8,36] Palpation of the caudal aspect of the elbow joint capsule may reveal fluid distension and some thickening. Flexion and extension of the elbow region often elicits a painful response, and a flexion test usually exacerbates the

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lameness.[36] The size of the SCL or its location does not appear to influence the degree of lameness. Intrasynovial anesthesia eliminates the lameness in most cases.[8,36]

Diagnosis

Radiographs are required to make the diagnosis. In one study, 4 of 7 of the SCLs were located in the proximal medial radius, and 3 of 7 were located in the distal medial condyle of the humerus.[36] In another study, SCLs were located primarily in the proximal medial radius (5 of 6), and 1 of 6 was located in the distal medial condyle of the humerus.[8]

Treatment

Subchondral defects are generally treated conservatively, whereas SCLs can be treated either conservatively or surgically.[8,36,95] Both have been treated successfully with prolonged rest and NSAID administration.[36,95] Initially, rest with concurrent intrasynovial treatment of triamcinolone and hyaluronic acid may be indicated. If the lesions do not appear to be resolving or the lameness does not improve, surgical intervention may be advised. In one study of six horses with SCLs in which conservative (3 of 6) and surgical (3 of 6) treatments were compared, surgical extra-articular enucleation of the cyst provided better long-term success (determined by return to athletic function) with less evidence of osteoarthritis than conservative therapy.[8]

While extra-articular access of the cyst is the recommended surgical approach, a complete treatment via arthroscopic approach to this joint is limited due to the cyst location. Lesions located in the proximal radius are generally not amenable to an arthroscopic approach; however, lesions located in the medial or lateral humeral condyles may be accessed more easily arthroscopically. Often the lesions are visualized with the arthroscope, yet adequate debridement is difficult (Figure 5.39).[59] For a medially located lesion, the surgery is performed with the horse positioned in lateral recumbency with the affected limb down. Care is taken to identify but not disturb the neurovascular plexus, a known complication of this approach, and the humeroradial joint identified with needle placement. The relative location of the cyst is then identified radiographically; extra-articular osteostixis and/or arthroscopically guided intralesional or extra-articular steroid injection can be performed.

Prognosis

The prognosis appears to be good for conservative treatment of SCLs as long as there is no radiographic evidence of osteoarthritis. Supporting the conservative approach for the treatment of SCLs is one report in which 6 of 7 horses treated with rest and the administration of anti-inflammatory agents returned to their intended use.[36] Five of the six horses returned to intense athletic performance, four flat racing, and one barrel racing. The recommendations from this report were that nonsurgical treatment should be considered in horses without radiographic signs of osteoarthritis and that surgery could be considered as an option if the horse remains lame at the 90-day follow-up examination.

The outcome of surgery on three horses with SCLs resulted in 2 of the 3 returning to their intended use. The other horse sustained a comminuted fracture of the radius during recovery from anesthesia.[8] More recently, there is evidence that if accessible, injection of the cyst itself with triamcinolone has shown favorable results in other joints,[40,88] and there is one report of a successful elbow cyst injection.[29] Injection of the cyst with corticosteroids either via arthroscopic guidance or in an extra-articular manner is recommended if possible.

Bursitis of the Elbow (Olecranon Bursitis)

Historically shoe boil or capped elbow is regarded as a problem in draft breeds, but there are reports in a variety of horse breeds.[35,74] It may occur on one or both elbows and is a characteristic movable swelling over the point of the olecranon tuberosity, usually developing from trauma. The trauma results in a transudative fluid accumulating in the subcutaneous tissue, which becomes

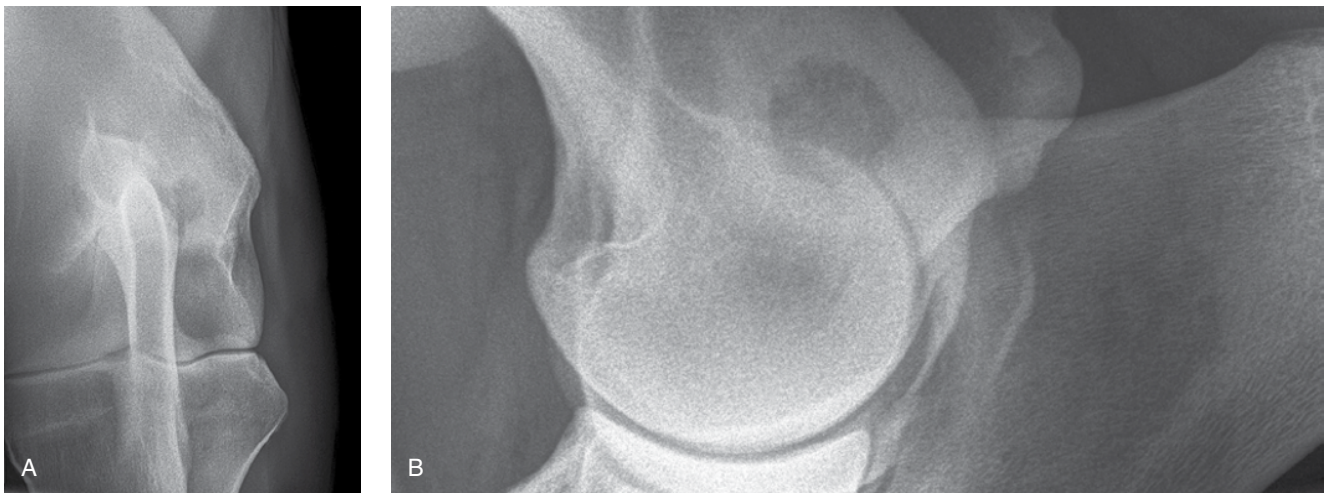


Figure 5.39. Subchondral cystic lesion located on the axial aspect of the lateral epicondyle of the radius demonstrated on cranial to caudal (A) and lateral (B) radiographic views. This cyst was observable arthroscopically. *Source*: Courtesy of Dr. Alanna Zantingh.

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encapsulated by fibrous tissue. Ultimately a synovial like membrane develops, producing fluid that is similar to joint fluid although it differs in viscosity and mucin clot.

Subsequently the chronic bursitis is characterized by the accumulation of bursal fluid and thickening of the bursal wall by fibrous tissue; fibrous bands and septa may develop in the bursal cavity, and the subcutaneous tissues around the bursa continue to thicken.[35] Bursal enlargement usually develops as a painless swelling that does not typically interfere with function unless it becomes greatly enlarged. In most cases, the acquired bursa is only a cosmetic blemish unless it becomes infected. An infected bursa is painful, causes lameness, and may break open to drain. The small true bursa that underlies the insertion of the triceps brachii muscle is rarely involved.

Etiology

Acquired bursitis is commonly caused by trauma from the shoe of the affected limb hitting the point of the elbow during motion or more commonly when the horse is lying down. American Saddlebreds and Standardbreds may hit their elbows during exercise. The bursae may become infected by a puncture or iatrogenically.[35]

Clinical Signs

The condition is characterized by a prominent, often freely movable swelling over the point of the elbow, which may contain fluid or may be composed primarily of fibrous tissue in the chronic stages. Lameness is usually not present unless the bursa is greatly enlarged or infected. Infected bursae feel warm, and firm pressure causes pain (Figure 5.40).



Figure 5.40. A chronic, infected, acquired capped elbow of the left forelimb. *Source*: Courtesy of Dr. Gary Baxter.

Diagnosis

The diagnosis can usually be made on physical findings alone. However, if the bursa appears infected, radiographs should be taken to rule out trauma or infection involving the olecranon process. If a draining tract is present, contrast material may be injected into the tract to identify its depth and course. Ultrasound can also be used to determine whether deeper structures are involved.

Treatment

In the acute stage, the condition may resolve by preventing further trauma to the region with a shoe boil roll or boot. The fluid can adjunctively be removed aseptically and corticosteroids injected after removal.[34,35] The lesion can be injected more than once. If the initiating cause is removed and the lesion is treated before extensive fibrosis occurs, it may resolve. However, in one report, 7 out of 10 horses were treated with intrabursal steroids, and none resolved.[35] Surgical intervention after lesion draining and injection is possible.[43]

There are reports of other substances such as strong 7% iodine being injected into affected bursae with varying success. Orgotein has been used, resulting in reduction but not resolution, as have short-lived radionuclides such as dysprosium-165.[35]

Surgical intervention either by placing drains or en bloc resection appears to have the greatest success.[34,35] Open drainage and application of Penrose drains and pressure bandages over periods of up to 8 weeks was successful in three horses.[87] En bloc resection is the treatment of choice for large and mature acquired olecranon bursae. Preferentially, the procedure is performed standing. Local anesthesia is administered and a curved skin incision is made over the lateral aspect. A plane of dissection is made to remove the mass in its entirety. Excess skin is removed as needed, and the skin is then closed in a tension-reducing manner such as widely placed vertical mattress sutures. A stent bandage is placed to protect the suture line and provide some compression. The horse can be maintained in cross-ties or an overhead wire until satisfactory healing is attained, usually 2–3 weeks.[35]

Prognosis

Conservative treatment of an acquired bursa to achieve acceptable cosmetic outcome is guarded. En bloc resection is regarded as a superior way to manage the lesion with good cosmesis.[34,35] In one report 5 out 6 horses resolved with primary healing after surgery.[75]

FRACTURES OF THE HUMERUS

Fractures of the humerus are relatively uncommon in horses, possibly because of the short, thick configuration of this bone and the prominent surrounding heavy musculature. The fracture can occur in horses of any age, breed, or sex, but most often affects foals less than 1 year of age, racing or race training Thoroughbreds, and horses that are used for jumping or steeplechase events.[14,55,60,96] In a study evaluating 54 horses with humeral fractures, the mean age for fracture occurrence

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was 3 years, and 28 of 54 (52%) were less than 6 months of age.[14]

Humeral fractures are classified as incomplete or complete, open or closed, simple or comminuted, and nondisplaced or displaced. Most are complete, closed, and displaced. Comminution occurs occasionally. Humeral fractures can also be classified by location: proximal humeral head (epiphysis and metaphysis in foals), greater tubercle, deltoid tuberosity, mid-diaphyseal, distal metaphyseal and epiphyseal, and distal condylar and epicondylar (Figures 5.41 and 5.42).[14,25,60,66,83,94] Most fractures are reported to involve the middle third of the diaphysis, either oblique or transverse or spiral configuration and almost never open (Figure 5.42).[11,56] A study on 54 horses with humeral fractures reported that 9 of 54 (17%) involved the physis (1 proximal and 8 distal), 8 of 54 (15%) occurred in the proximal metaphysis (4 oblique and 4 transverse), 11 of 54 (20.4%) were distal metaphyseal fractures, 26 of 54 (48%) were diaphyseal (2 short oblique, 4 transverse, 20 long oblique or spiral), and 1 of 54 was open.[14] In another study done on 22 horses with humeral fractures, 14 of 22 fractures were spiral in configuration.[96] Uncommonly foals will sustain multiple fractures involving the elbow region.[6,62] Fractures of the greater tubercle can involve either the caudal or cranial aspect[83,94] and occasionally are comminuted.[60]

Because of the large muscular attachments to the humerus, there is often considerable overriding with displaced fractures (the distal fragment being displaced



Figure 5.41. Proximal metaphyseal transverse humeral fracture. *Source*: Courtesy of Dr. Martin Waselau.



Figure 5.42. (A) An oblique lateral view of a spiral nonarticular fracture of the midhumerus. (B) Lateral view of a healing spiral nonarticular fracture of the midhumerus treated conservatively for 5 months. *Source*: Courtesy of Dr. Gary Baxter.

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caudad and the proximal fragment being displaced craniad). However, it is uncommon for proximal complete humeral fractures to become displaced because of the stability provided by the surrounding muscles (supraspinatus, infraspinatus, subscapularis, and deltoid), biceps tendinous insertions, and periarticular capsular attachments of the shoulder.[66] Incomplete stress fractures occur in two typical locations: the proximal caudal lateral cortex and the distal cranial medial cortex.[56]

The radial nerve courses in the musculospiral groove of the humerus and may be traumatized to varying degrees as a result of complete displaced diaphyseal or metaphyseal humeral fractures.[11,55] The damage may range from a minor neuropraxia to a complete severance of the nerve. Because of the profound effect on prognosis, it is important to evaluate the degree of nerve dysfunction early in the convalescent period.

Etiology

Humeral fractures frequently occur in foals, in weanlings secondary to falls or other impact injuries, and in racing breeds as either catastrophic failure during race falls or failure as a result of accumulated stress and microfracture.[11,56,60,96] In a study of 54 horses with humeral fractures, falls on hard surfaces or related to racing were responsible for fractures in 11 horses, kicks in 2 horses, post-anesthetic recovery in 2 horses, collisions with fences or another horse in 3 horses, and car collision in 1 horse.[14] Trauma is the cause of most fractures of the deltoid tuberosity or greater tubercle.[60]



Figure 5.43. Radial nerve paralysis, evidenced by the classic dropped elbow stance. This stance can also be seen in horses with ulna and humeral fractures, but accompanying soft tissue swelling is usually present. *Source*: Courtesy of Dr. Gary Baxter.

In an *in vitro* model, the configuration of the fracture was predictable and depended on the direction from which the insult originated. When the force was applied in a craniocaudal direction, the humerus fractured transversally; when the force was applied in lateral to medial direction, the humerus fractured obliquely.[56]

Horses sustaining stress fractures are at an increased risk to develop a complete fracture if they are not managed properly. A study of 34 Thoroughbred racehorses with humeral stress fractures found an increased risk of complete fracture in horses that returned to racing after a short 2-month lay-up period.[13] In one study that did not have any horses develop complete fractures during rehabilitation, the mean time to return to racing for horses with humeral fractures was 7.5 months.[67] Radiographic and scintigraphic reexamination allows for more accurate assessment of recovery.[85]

Clinical Signs

Horses with nondisplaced or minimally displaced proximal fractures (Salter–Harris type I epiphyseal, greater tubercle, or deltoid tuberosity) and nondisplaced midshaft fractures often present with a history of a severe lameness that improves over a 24- to 48-hour period. Moderate swelling may be present at the site of injury for a proximal fracture or over the lateral muscles for a midshaft fracture or even distal if a few days have passed. Pain is often present with pressure applied over the fracture and on limb manipulation. Radiography often provides a definitive diagnosis.

Incomplete fractures and stress fractures resulting in lameness can be most difficult to diagnose.[52,56,67,80,91] In some cases lameness and mild swelling may be adequate to lead to a tentative diagnosis of a fracture, but the definitive diagnosis often requires nuclear imaging.[67]

Horses with complete displaced fractures often present with a history of an acute onset of a severe nonweight-bearing lameness. Marked to moderate swelling of the muscles overlying the region is often seen, and the elbow is usually dropped (Figure 5.43). The dropped elbow may be due to the overriding of the fracture segments resulting in functional limb shortening or from the varying degrees of damage to the radial nerve.[14] Limb manipulation usually causes increased pain and an increased range of motion when the limb is adducted and abducted. Crepitation is often difficult to appreciate in heavily muscled horses, with the muffling effect of the swollen musculature. Limited manipulation should be done because it may result in further trauma to the radial nerve. Radiographs will identify the fracture, but evaluation of radial nerve damage is more difficult. Electromyography (EMG) of the antebrachial extensor muscles can be used after 2 weeks to evaluate radial nerve damage. In one study 4 of 40 horses treated for fractures of the humerus were destroyed because of loss of radial nerve function.[14]

Fractures of the distal epiphysis, condyles, and epicondylar region are very uncommon.[26,62,89] When they do occur, they often present with a history of marked lameness of a short duration. Swelling associated with the elbow region may be apparent, including joint effusion if the fracture is articular.

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Diagnosis

Radiography is used in most cases to confirm the fracture and define the configuration. A slightly oblique medial to lateral projection with the limb held in extension usually provides the information needed for epiphyseal and shaft fractures. In most cases the study can be done in the standing sedated horse. Foals can be restrained in lateral recumbency with the affected limb down to obtain a diagnostic lateral projection. Craniocaudal views of the distal humerus and oblique views of the proximal humerus can be obtained in most standing sedated horses and foals. Craniocaudal views of the entire proximal humerus are more difficult to obtain, and general anesthesia may be required.

Multiple radiographic views may be needed to identify fractures of the greater tubercle and deltoid tuberosity. Obtaining a cranioproximal to craniodistal oblique projection of the proximal portion of the humerus in a standing horse often highlights the long oblique fractures of the greater tubercle that are not evident on a mediolateral view.[60] In one study of deltoid tuberosity fractures, the authors noted a cranial 45° medial to caudolateral oblique view assisted in diagnosis.[28] In this study 86% of the fractures were identifiable with ultrasonography alone.

Horses with stress fractures often have a grade 3/5 lameness in the affected limb that improves quickly after the initial injury. Manipulation of the elbow and shoulder often exacerbates the lameness, yet diagnostic anesthesia is not useful.[67] A definitive diagnosis of stress injuries requires nuclear scintigraphic as well as radiographic examination. The caudodistal cortex or the caudoproximal cortices are most commonly affected, but fractures of the cranioproximal and craniodistal cortices also may occur.[4,52,67,80] Radiography may identify callus formation along the affected cortex in chronic cases. For more information on humeral stress fractures, refer to the section on Thoroughbred racehorses in Chapter 9.

Treatment

Currently three options are considered when managing a horse with a humeral fracture: nonsurgical (conservative) management with prolonged stall rest, surgical reduction followed by stabilization, and euthanasia.[11,14,55,66,80,84] Of the two treatment approaches, nonsurgical management appears to provide the best outcome compared with surgical approaches for most nonarticular incomplete or complete humeral fractures, no matter what the age of the horse (Figure 5.42).[14,96]

In one report, 7 of 10 horses treated nonsurgically were able to be ridden 5–12 months after the diagnosis was made, and only 1 of 3 surgically treated (2 rush pins, 1 lag screws) cases was considered sound for riding.[96] The ages of the horses that were treated nonsurgically ranged from 2 to 60 months (mean 21 months). In another study, conservative treatment resulted in 9 of 17 cases being considered successful; 4 became athletically sound and 5 were pasture sound.[14] The horses that were treated nonsurgically ranged in age from 1 week to 15 years (mean 3 years). Euthanasia still remains a commonly selected option. In a retrospective study done on 54 horses with humeral fractures, more horses were euthanized (44.4%) than treated with surgical (24%) or conservative (32%) management.[14] In another retrospective study, 9 of 22 humeral fracture cases were euthanized.[96]

Nonsurgical Management

Nonsurgical management has been used successfully to treat stress fractures; incomplete or complete nondisplaced or minimally displaced nonarticular fractures; minimally displaced Salter–Harris type I and II fractures of the proximal and distal physis; deltoid tuberosity fractures; complete displaced proximal and distal transverse and oblique fractures; and complete displaced diaphyseal transverse, short, and spiral oblique fractures.[14,28,56,66,96] In two reports a better outcome was obtained in foals compared with adults.[14,96] Long transverse and oblique fractures can be reasonably stable when the caudodistal aspect of the proximal fragment rests within the epicondylar groove. Shorter oblique fractures have less stability and usually require internal fixation. Contraction of the heavy musculature surrounding the humerus also helps stabilize the fracture while it heals. Of 19 cases of fracture of the deltoid tuberosity, local wound care alone resulted in a return to athletic function without lameness in 13 of the 14 available to follow-up.[28]

Nonsurgical management consists of 3–6 months' stall rest, with periodic radiographic or nuclear imaging reevaluation of the fracture for healing.[66,80] Initially, incomplete or complete nondisplaced fractures can be maintained on an overhead wire to minimize the chance of developing a complete fracture.[56,66,80]

Complications are not common but may include contralateral-limb laminitis and angular limb deformity, radial nerve damage, and nonunion.[14,96] No adverse effects attributable to shortening of the humerus, such as a shortened stride or a straightened elbow joint, were reported in successfully treated horses in these studies.

Surgical Management

Unfortunately, the current techniques of dynamic compression plating, intramedullary fixations, and cerclage bands or wires do not have sufficient strength to provide adequate stability in adult horses.[11,55,56]

Intramedullary pinning, bone plating, and ASIF or interlocking intramedullary nailing or rush pins have been used to successfully treat foals and ponies with humeral fractures.[14,15,23,66,91] Bone plates have been applied to repair nonarticular humeral fractures in foals and yearlings.[14,61,86] At present most reports describe the use of DCP or LC-DCP; there are few if any reports of the use of LCP for humeral repairs. The interlocking intramedullary pin has been used successfully in foals weighing up to 220 kg.[91] In larger foals a cranial plate is also applied to augment the reduction.

Unstable and extensive greater tubercle fractures of the proximal humerus may require removal or internal fixation with lag screws.[1,60,94] Extensive fractures of the deltoid tuberosity, although uncommon, also can involve the greater tubercle of the proximal humerus. Surgical intervention is required to reestablish cortical continuity of the bicipital groove and reestablish the tensional

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support of the tendinous insertions of the deltoid, brachiocephalicus, supraspinatus, and occasionally the infraspinatus muscles.[66] Generally all that is needed are 2–3 4.5- or 5.5-mm cortical or 6.5-mm cancellous screws placed in lag fashion that engage the transcortex.[23] This has been performed with success as a standing procedure.[54] In heavy horses a narrow DCP may be used to provide a tension band effect.[66]

Fractures of the condyles or epicondyles of the distal humerus occur infrequently in horses, but small articular fragments involving the condyles should be removed arthroscopically.[62,64] If the fragment is nonarticular, conservative therapy is considered.[26] More extensive fractures of the condyles or epicondylar region may require internal fixation with lag screws.

Prognosis

The prognosis for stress fractures, nondisplaced complete or incomplete fractures, or minimally displaced complete nonarticular fractures managed conservatively appears very good.[14,80,96]

The prognosis for complete displaced nonarticular fractures is guarded but appears better for horses managed conservatively. In one study in which conservative management was used in 17 horses, 9 (53.9%) were considered successful, 4 horses became athletically sound, and 5 horses became pasture sound; younger horses may do better.[14] In this same study, 13 horses were treated surgically with a variety of internal fixation methods; 3 of 13 horses survived and became athletically sound. The successful outcomes were all surgically treated with stacked pinning and all were young foals. In another study, conservative management was used in 10 horses, 7 of which were able to be ridden 5–12 months after the diagnosis was made.[96] The horses' ages in the successful group ranged from 2 to 60 months. Of the 3 horses treated surgically, 1 became pasture sound 10 months after surgery. The conclusion was that the best candidates for nonsurgical treatment are nonarticular humeral fractures with minimal displacement that involve the midshaft and are spiral and oblique with minimal overriding of the ends of the bone.

For fractures involving the greater tubercle and deltoid tuberosity, the prognosis appears good for conservative management of nondisplaced and surgically treated displaced greater tubercle and deltoid tuberosity fractures.[14,22,60] A recent report of 19 cases of treated conservatively resulted in 13 horses returning to athletic function with no lameness.[28] In one report, 9 out of 10 horses with greater tubercle fractures treated surgically returned to athletic use. Of these, 7 had the fragment removed and 2 had internal fixation.[60] There are few reports of epicondylar fractures, but in one case of a fracture of the epicondyle and supracondylar crest, the horse returned to athletic use after the fracture fragments were removed.[62]

NEOPLASTIC LESIONS OF THE HUMERUS

There have been several reports in the recent literature of both primary and metastatic bone tumors in the proximal humerus of the horse.[16,38,44] These lesions remind the clinician to consider neoplasia as a differential diagnosis as a cause of lameness.

Primary osteosarcoma has been diagnosed and described in an adult Quarter horse mare.[44] Chronic significant and progressive lameness was noted; discomfort was isolated by palpation to the scapulohumeral joint. Arthrocentesis was normal and diagnostic analgesia was uneventful. Radiographs revealed an aggressive bone lesion in the proximal humerus. Euthanasia was elected due to the poor prognosis and lack of improvement despite therapy. Histological examination provided a definitive diagnosis. Osteosarcoma is rarely reported in the horse and when so more frequently in the head.[49]

A primary hemangiosarcoma was diagnosed in an 11-year-old Clydesdale gelding.[16] Local palpation of the proximal humerus revealed discomfort and diagnostic analgesia was unhelpful. In this case nuclear scintigraphy was performed, resulting in significant uptake of isotope in the proximal humerus. Radiographs revealed a radiolucent area with areas of sclerosis and some irregular new bone formation. Bone biopsies were performed but considered non-diagnostic. A definitive diagnosis was made after histopathological examination of the tissue subsequent to euthanasia. Hemangiosarcoma has been reported in the horse;[41] however, in this report only one of 11 cases was reported to be involving bone in a limb.

Metastatic lesions in the bone from intestinal adenocarcinomas are unusual and reported in a horse with lameness associated with sequestrum formation and a pathologic fracture.[38] A tentative diagnosis of osteomyelitis had been provided, but postmortem histopathology provided a definitive diagnosis of metastatic adenocarcinoma with primary lesions in the colon.

PARALYSIS OF THE RADIAL NERVE

Paralysis of the radial nerve as a primary cause of lameness is an uncommon condition that results in the inability to extend the elbow, carpus, and digit. Similar dysfunction may also be observed with other conditions involving the upper forelimb; therefore, differentiation can be difficult, particularly shortly after injury occurs.

The radial nerve, often the largest branch of the brachial plexus, derives its origin chiefly from the eighth cervical and first thoracic nerve roots of the plexus. In 10% of horses the seventh cervical nerve root contributes to the radial nerve.[32] The radial nerve innervates the extensor muscles of the elbow, carpus, and digit and supplies the lateral flexor of the carpus (ulnaris lateralis). Paralysis of the radial nerve inactivates these muscles.

Etiology

In most cases, paralysis of the radial nerve is due to trauma of the shoulder region caused by hyperextension of the forelimb or extreme abduction of the shoulder.[70] Fractures of the humerus, the seventh cervical, and first thoracic vertebrae can all result in radial nerve paralysis.[50] Tumors, abscesses, and enlarged axillary lymph nodes that occur in the cranial thoracic region along the course of the nerve and tumors of the brachial plexus and radial nerve themselves may also result in radial

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nerve paralysis.[58] There have been isolated reports of radial nerve paralysis included in a generalized distal axonopathy.[31]

Prolonged lateral recumbency while under general anesthesia on an operating table or while on the ground may also produce a radial-paralysis-like syndrome in the forelimb.[48,51] Episodes of ischemia are likely to cause neuropractic conduction changes and permanent nerve changes if prolonged.[51]

Clinical Signs

The signs vary depending upon the extent or degree and location of paralysis. When the portion of the radial nerve supplying the extensors of the digit is affected, the signs are characteristic. In the acute phase the horse is unable to bear weight because of the inability to extend the elbow, carpal, and phalangeal joints. If the limb is placed under the horse and fixed in place, it can bear weight passively.[70] While the horse is standing, the shoulder is extended and the elbow is dropped (dropped elbow appearance) and extended while the carpus and digits are flexed. The muscles of the elbow and the extensors of the carpus and digit appear relaxed, and the limb appears longer than normal. The "dropped elbow" appearance is not specific for radial nerve paralysis; this appearance can be seen with many other conditions associated with the elbow, humerus, and shoulder regions (Figure 5.43). Occasionally, radial nerve paralysis is accompanied by paralysis of the entire brachial plexus. In this case, the limb shows paralysis of the flexor and extensor muscles and is unable to bear weight.

With complete radial nerve paresis, the horse is generally reluctant to move, and at a walk the limb is dragged forward passively by the action of the proximal pectoral, biceps brachii, and coracobrachialis muscles, with the dorsal surface of the hoof in contact with the ground. Due to the physical distress associated with radial nerve paralysis, some horses may sweat profusely and have an elevated pulse and respiration.[70] Milder cases of radial nerve paralysis present with varying degrees of non-weight-bearing lameness and a dropped elbow with mild flexion of the carpal, fetlock, and phalangeal joints. At a walk the lameness is often characterized by a lowered foot flight arch and a shortened cranial phase of the stride.[15]

Diagnosis

Many cases of radial paralysis are due to external trauma; therefore, the scapula, humerus, radius, and olecranon process should be examined radiographically.[58] Radial nerve paralysis must be further differentiated from rupture of the medial collateral ligament of the elbow, elbow arthritis, and myopathy of the biceps brachii, triceps brachii, anconeus, and extensor carpi radialis muscles.[70]

It previously was believed that cutaneous sensation to the craniolateral aspect of the antebrachium is lost with complete loss of nerve conduction of the radial nerve or brachial plexus and that if the sensation is present, it indicates the lesion selectively affects the motor fibers.[70] However, studies done in horses indicate there are no areas of skin innervated only by either the radial or the axillary nerves—the ulnar, median, and musculocutaneous nerves can be related to such areas.[9]

Electrophysiological studies can be helpful in characterizing the extent of nerve damage and can be used to determine selective denervation of the extensor muscles. However, because it takes approximately 7 days for the axon distal to the site of the damage to degenerate, EMG done before this time has limited usefulness.[3] Alternatively, faradic stimulation done 7 days after clinical signs develop can be used to differentiate between neuropraxia and axonotmesis or neurotmesis.[70] Radiography, ultrasonography, and laboratory analysis of muscle enzymes may be needed in some cases to make an accurate diagnosis of radial nerve paralysis.

Treatment

Treatment consists of systemic and local anti-inflammatory therapy, stall rest, application of a bandage splint, and controlled exercise. A PVC bandage splint applied to the caudal aspect of the limb from the proximal antebrachium to the fetlock will maintain the limb in extension, permitting the horse to bear weight comfortably. The splint is maintained until the horse can place the limb in extension without support. Controlled exercise can be started when clinical signs (neurologic function) begin to improve. The amount of exercise is dictated by the horses' capabilities. Electromyographic studies can be done at 4- to 6-week intervals to access return of nerve function.

Prognosis

EMG has been found to have a good prognostic value in assessing the extent of the injury in a small number of cases.[15] In most cases a guarded to poor prognosis is given,[15,45] but compression and entrapment injuries often lead to partial or complete recovery.[77] Recovery may take a few weeks in pure neuropraxia to several months or years in axonotmesis.[45,58] Reinnervation is, however, unlikely with more severe nerve damage.[15]

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THE SHOULDER AND SCAPULA

JEREMY HUBERT

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INFLAMMATION OF THE INTERTUBERCULAR BURSA (BICIPITAL BURSITIS)

Inflammation of the intertubercular (bicipital) bursa as a primary cause of lameness is uncommon, even though the condition can occur in horses of any age, breed, or sex.[11,42,48] In 2 reports the condition was responsible for lameness in 1 of 54 and 1 of 41 horses suspected of having shoulder lameness.[40,64]

The bicipital bursa is located between the bilobed tendon of the origin of biceps brachii muscle and the tubercles at the cranioproximal aspect of the humerus. The synovial membrane of the bursa extends around the axial and abaxial limits of the tendon and onto the margins of its cranial surface. Although uncommon, communication can exist between the shoulder joint and the bicipital bursa.

Etiology

Trauma to the cranial surface of the shoulder region is believed to be the most common cause of a primary bursitis.[11,40,48,97] Other suggested causes include stretching or tearing of the bursa or biceps tendon during the cranial phase of the stride with the limb in full extension or by a fall or slip that results in flexion of the shoulder with extension of the elbow.[80,95] Inflammation of this bursa can also be caused by dislocation of the biceps brachii tendon that may be associated with congenital hypoplasia of the minor tubercle.[25,51,66] Proximal humeral subchondral bone cysts adjacent to the intertubercular groove of the bicipital bursa may cause local inflammation,[59] and such cysts have been reported to be infected.[45]

Infection either from an open or penetrating wound or from hematogenous spread to the bursa has also been reported.[40,79,97] Cases of septic bursitis concurrent with septic tendinitis and arthritis of the scapulohumeral joint or bursitis with associated tendinitis and humeral osteitis have been reported.[43,44]

Clinical Signs

A history of trauma to the shoulder region is the most common factor; the signs of lameness usually have an acute onset that is most obvious during the stance and swing phases of the stride. On physical examination swelling over the cranial aspect of the shoulder region may be evident with or without a wound being present. Generalized shoulder and pectoral muscle atrophy may be seen in more chronic cases.[40,48,97] Pressure applied over the biceps tendon and bursal region and manipulation of the shoulder region in flexion and extension usually result in a prominent painful response (Figure 5.44). The responses to pressure and limb manipulation should be compared with those found on the contralateral unaffected limb.[48]

The lameness is often obvious. At exercise, a lameness of 3–4 out of 5 is generally seen (greater if septic), with the swing and stance phases being altered. The lameness is often characterized by a shortened cranial phase of the stride, a decrease in the height of the foot flight arc, reduced carpal flexion, and a fixed shoulder appearance during movement. The horse is often reluctant to bear full weight on the affected limb while standing.[48]

Diagnosis

Radiographs of the shoulder and ultrasound examination are performed if the physical examination localizes the pain causing lameness to the bursal region. Manipulation of the elbow into hyperextension may stress the biceps brachii tendon, helping to localize the lesion.[70] However, if the localizing signs are not obvious, bursitis is confirmed by centesis and local anesthesia of the bursa. A proximal approach to the bursa is considered to be the most successful for bursal centesis.[88] This technique is further improved by extending the scapulohumeral joint. The radius is held in a plane parallel to the bearing surface, and a 9-cm 18-g spinal needle inserted parallel to the ground into the lateral portion of the intertubercular groove. This procedure was more likely to access the bursa in the standing horse (Figure 5.45).[24] However, ultrasound-guided centesis has been demonstrated to be superior for reliably accessing the bursa.[85]

Radiographs of the shoulder region are taken to identify any osseous changes in the tubercles or bicipital groove or bursa and to rule out the possibility of fractures of the supraglenoid tuberosity or proximal humerus or ossification of the biceps tendon



Figure 5.44. As the shoulder is flexed, tension is created in the tendon of the biceps brachii muscle. If the horse has bicipital bursitis or ossification of the tendon, a painful response is elicited.

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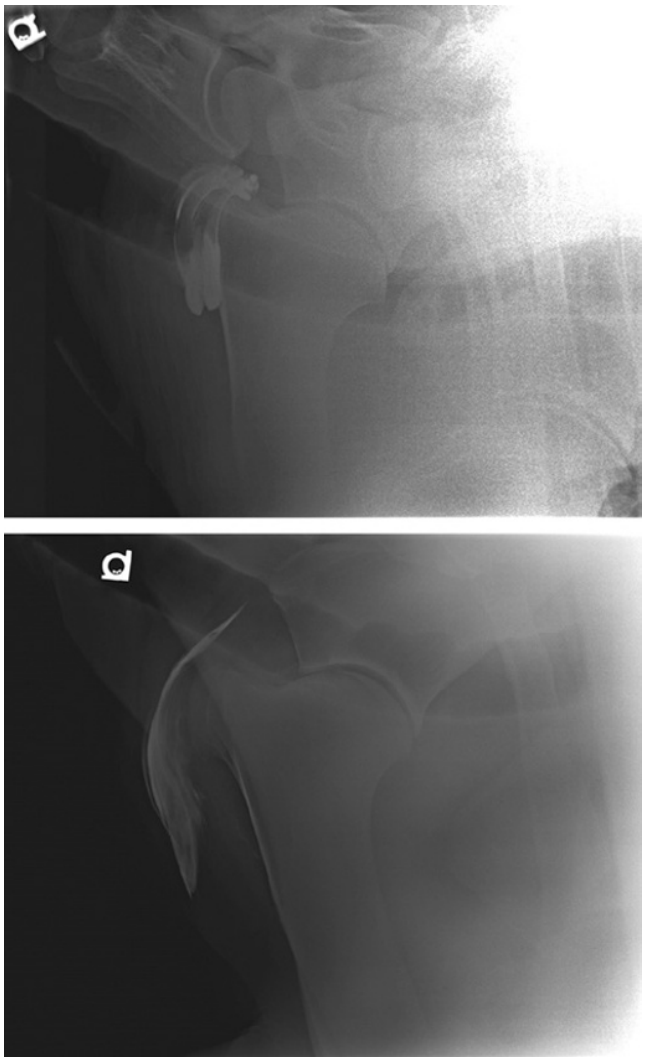


Figure 5.45. Radiopaque contrast medium contained within the bicipital bursa. *Source*: Reprinted with permission from Cole et al.[24].

(Figures 5.46 and 5.47).[11,40,48,75,97] Generally, only mediolateral, craniomedial to caudolateral oblique, and flexed cranioproximal to craniodistal (skyline) radiographs are taken in adult horses.[48,58] A cranioproximal to craniodistal oblique view is useful when the tubercles are thought to be involved.[78] Radiographs may appear normal if the disease only involves the bursa. If a wound or draining tract is present, centesis with contrast material assesses communication with the bursa.

Ultrasound examination of the biceps tendon, bursa, and bicipital groove can be very informative, even when radiographs appear normal (Figure 5.48).[17,49,77,78] Ultrasound changes associated with bursitis have included edema or hemorrhage in the biceps tendon or bursa, disruption of the tendon architecture with peritendinous thickening, an irregular surface of the bicipital groove, and hyperechoic material in the bicipital bursa.[26,97] Infection must be ruled out if hyperechoic material is seen in the bursa.[97] Nuclear medicine may be useful in some cases to detect early infected or actively remodeling bone adjacent to the bursal region when ultrasound and radiography appear normal.[48,78]

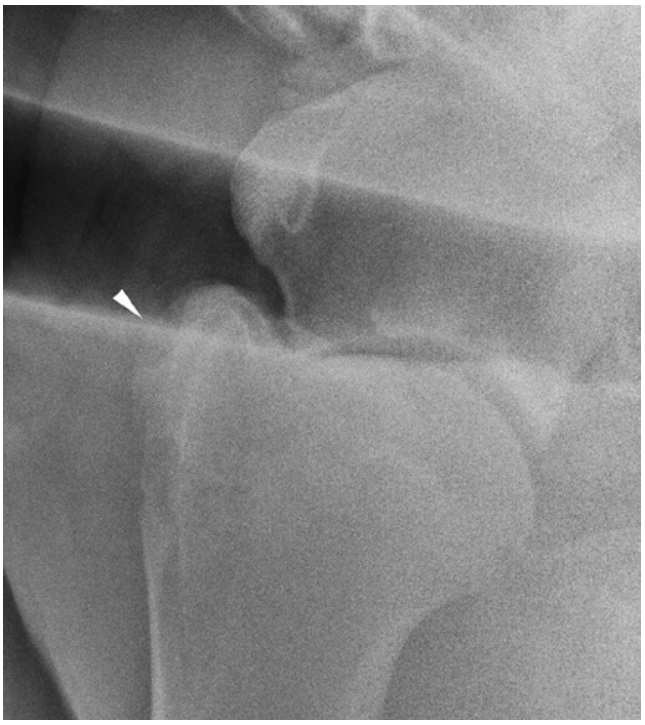


Figure 5.46. Osseous changes on the tubercles of the cranioproximal humerus (arrowhead). Infiltration of lidocaine into the bursa resolved the lameness in this case.

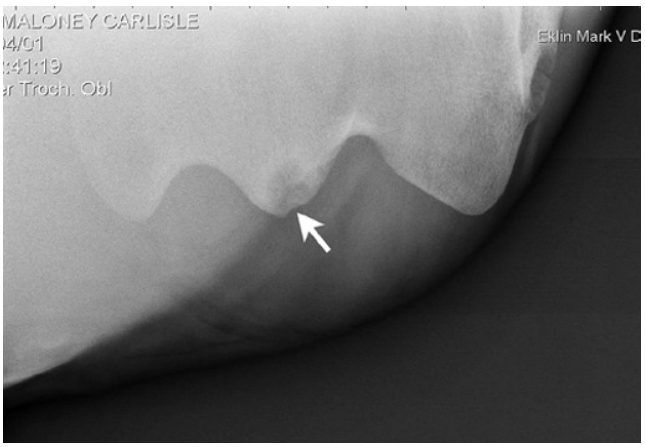


Figure 5.47. An osseous cyst located in the intermediate tubercle in the cranioproximal humerus. *Source*: Courtesy of Dr. Colin Mitchell.

Treatment

Conservative Treatment

Noninfectious bursitis without radiographic evidence of a fracture or pathology on the cranioproximal aspect of the humerus may respond favorably to rest, parenteral and/or topical administration of nonsteroidal anti-inflammatory drugs (NSAIDs), and in some cases intrasynovial injection of the bursa with corticoids and hyaluronic acid (HA).[40,42,59,81]

The duration of rest generally depends on the nature of the injury, the patient's clinical response to rest, and whether or not the biceps tendon was damaged. NSAIDs

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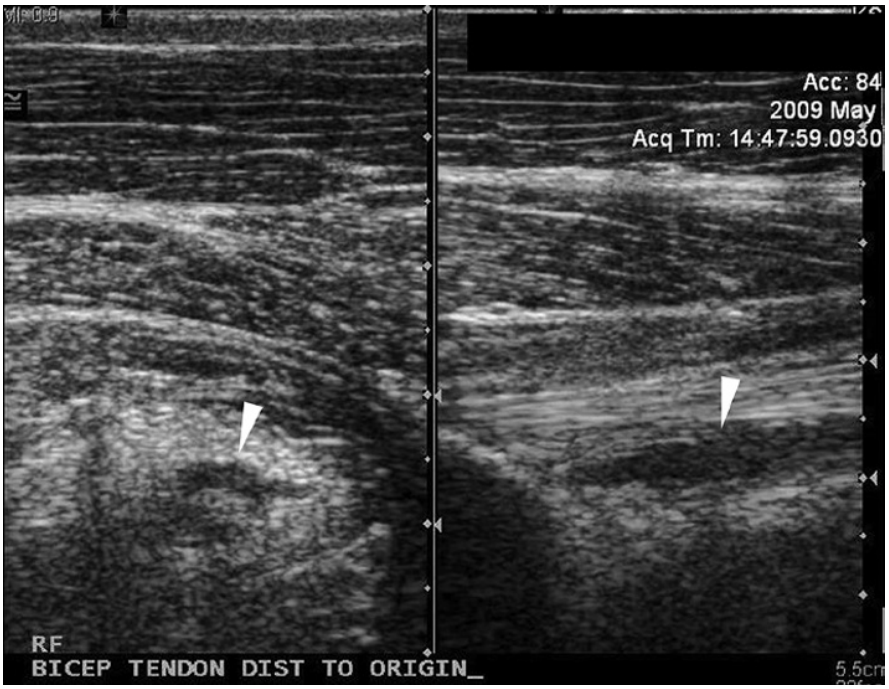


Figure 5.48. Core lesion of the medial branch of the biceps tendon just distal to the origin of the biceps brachii muscle (arrowheads).

are generally administered for 10–14 days and hand-walking exercise is begun after that. Manipulating the limb passively through a range of motion may also be helpful.[40,42,48,81] When the tendon has been injured, rest periods for up to 3 months followed by paddock rest for another 3 months may be required to allow healing of the tendon.

When the tendon has ossified, extracorporeal shock-wave therapy (ESWT) may be indicated. In humans, calcifying tendinitis is commonly treated with ESWT[46,61,82] and has been used with fair success in horses with calcifying tenopathies of the deep digital flexor tendon.[84] Additional therapy could include stem cells, IRAP, and PRP therapy, depending upon the lesion. ESWT applied to tendon lesions is still controversial; however, anecdotal reports appear to be encouraging.[18] In these horses, controlled exercise was begun as early as possible, generally after 2–3 weeks of confinement to improve range of motion and healing. The patient's clinical response and ultrasound findings should be used to determine progress in healing.

Surgical Treatment

Bursitis that results from a displaced fracture, from osseous changes associated with the proximocranial aspect of the humerus, or from sepsis generally requires surgery to resolve the problem. Incisional as well as endoscopic approaches to the intertubercular bursa have been used and described.[4,11,40,48,94] If a fracture is present, the fragment is removed, and the bed debrided and smoothed.[48] Controlled exercise as described for the conservative approach is begun at the time of suture removal in most cases.

In cases in which infection appears to be the cause, centesis of the bursa should be done to collect fluid for culture and cytology. Next, the bursa should be drained and then debrided and lavaged copiously under arthroscopic guidance followed by therapies aimed at treating infected synovial cavities[94] Controlled exercise and moving the affected limb through a passive range of motion are advocated to reduce restrictive adhesion formation.[11]

In cases where an osseous cyst is detected within the intertubercular groove of the proximal humerus, arthroscopic surgical debridement is recommended.[7,45]

Prognosis

Acute cases of nonseptic bursitis when a fracture is not the cause often respond favorably to conservative treatment.[58] Conservative therapy for more chronic cases of nonseptic bursitis appears less satisfactory.[11,40,48,97] In one report of 3 horses with chronic nonseptic bicipital bursitis that were treated surgically, all became pasture sound.[48]

Horses with chronic septic bursitis cases treated conservatively have a poor prognosis for return to performance, and surgery is usually recommended. Surgical intervention with debridement, lavage, and appropriate antimicrobial therapy and rest gives a favorable prognosis.[40,43–45,64,79,97]

A small case series described five cases of osseous cyst-like lesions in the lateral intertubercular groove of the proximal humerus that were treated successfully with corticosteroid and HA.[59] Likewise a report of endoscopic or open debridement of such lesions resulted in a favorable outcome.[7,45]

INFLAMMATION OF THE INFRASPINATUS BURSA

The infraspinatus bursa is located between the tendon of the infraspinatus muscle and the caudal eminence of the greater tubercle of the proximal humerus. The bursa

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is not visible in most horses except when inflamed or septic.[80] This is an uncommonly reported cause of lameness.

Etiology

Severe adduction of the forelimb and/or the possibility of direct trauma to this region is considered to be the cause (Figure 5.49).[80,99]

Clinical Signs

The involved forelimb may be held in an abducted position, presumably in an attempt to reduce the pressure on the infraspinatus bursa. During exercise, a moderate lameness is present with an obvious decreased cranial stride.[80,99] Adduction of the limb reportedly elicits a painful response and results in increased signs of lameness at exercise (Figure 5.50).[80] If the bursa is septic, the lameness exhibited is usually severe.[99]

Diagnosis

Ultrasonographic evaluation and comparison with the opposite limb may be required, along with ultrasound-guided centesis[85] and local analgesia, for a definitive diagnosis.[99]

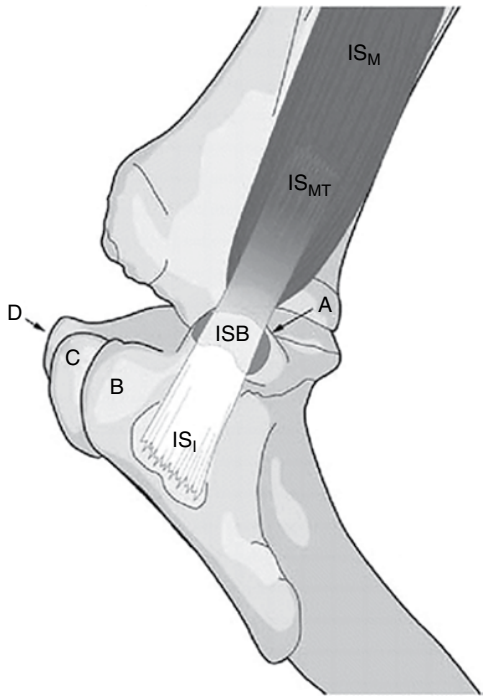


Figure 5.49. This line drawing illustrates the location of the infraspinatus bursa underlying the infraspinatus tendon. A, caudal eminence of greater tubercle (GT); B, cranial eminence of the GT; C, intermediate tubercle; D, lesser tubercle; ISM, infraspinatus muscle; ISMT, musculotendinous junction of the infraspinatus muscle; ISI, infraspinatus tendon insertion; ISB, infraspinatus bursa. *Source*: Reprinted with permission from Whitcomb et al.[99].

Treatment

In the acute stage, synoviocentesis and the administration of corticosteroids into the bursa are recommended. With sufficient stall rest (6 weeks or more) and the parenteral administration of NSAIDs, a good end result can be expected. Varying degrees of lameness may remain with chronic cases.[80] Septic involvement of the bursa requires endoscopic debridement and flushing. The initial distension of the bursa may require distension with fluid by ultrasonographic guidance to place the needle. The bursa is small and movement of instruments is restricted by the overlying muscle and infraspinatus tendon.[99]

Prognosis

In one report 3 horses returned to soundness after surgical treatment of septic infraspinatus bursitis.[99]

OSTEOCHONDROSIS (OC) OF THE SCAPULOHUMERAL (SH) JOINT OR SHOULDER

Osteochondrosis (OC) of the scapulohumeral joint or shoulder is most frequently diagnosed in weanlings and yearlings 6–12 months of age, but it has been reported in foals younger than 5 months and in horses up to 8 years old.[29,38,52,54,65,67,72–74] The incidence of the disease varies; one study reported diagnosing OC of the shoulder in 54 joints of 38 young horses radiographed for shoulder problems,[74] and in another study OC and subchondral bone cysts were observed in 6 of 29 diagnosed shoulder problems.[38] The condition is considered the most debilitating form of osteochondritis dissecans (OCD), and when it is diagnosed in older yearlings, chronic manifestation of secondary degenerative joint changes is usually present.[74]

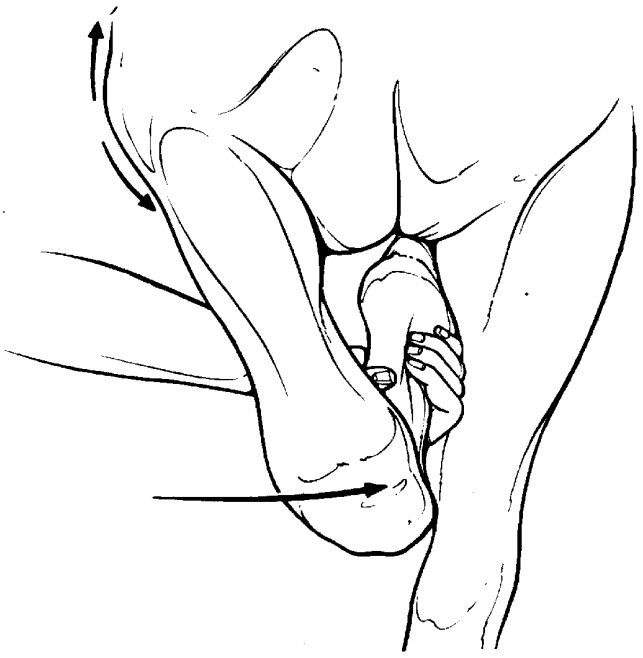


Figure 5.50. Adduction of the limb may result in a painful response in horses with infraspinatus bursitis.

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The primary cartilage lesion is located in the glenoid, humeral head, or both (most common), and the disease often affects a major part of the joint surface.[74] Regardless of the site of the lesion, secondary degenerative changes in the joint are prominent features of this disease. An exception is the solitary subchondral cystic lesions that are occasionally seen in the glenoid cavity.[38,54]

Etiology

Overnutrition, imbalanced nutrition, and a genetic predisposition to rapid growth at some stage of the development still appear to be reasonable hypotheses for most young affected horses. The secondary degenerative changes within the joint are exacerbated by the resultant instability.[74]

Occasionally, subchondral bone cysts are seen in the glenoid without cartilage changes in the humeral head, alterations of the contour of the articulations of the shoulder, or signs of secondary osteoarthritis.[38,54] Thus, it has been suggested that subchondral bone cysts may not be a manifestation of OC in the scapulohumeral (SH) joint.[40] A traumatic etiology is often suspected in such cases of subchondral bone cysts.[54]

Clinical Signs

Most horses present with a history of mild to moderate intermittent forelimb lameness with insidious onset. Atrophy of the muscles associated with the shoulder region is a common finding in chronic cases. A smaller foot with a higher heel and excessive toe wear is also commonly observed in the foot of the affected limb. Direct firm pressure with the thumb just cranial to the tendon of the infraspinatus muscle over the cranial lateral aspect of the shoulder joint may elicit a painful response, particularly in younger horses (Figure 5.51).

Exercise usually results in a moderate to severe lameness that is characterized by a shortened cranial (extension) phase of the stride and a delay in limb protraction. Stumbling may occur in some cases due to inadequate foot clearance. A prominent shoulder lift, reduced carpal flexion, and limb circumduction are often seen in the most severely affected horses. Manipulation of the affected limb in extension and flexion and abduction may cause pain and often increases the signs of lameness. Intrasynovial anesthesia is used to localize the lameness to the shoulder region; 10–15 mL of a local anesthetic should improve or eliminate the lameness in most cases.[38]

Diagnosis

Radiographs are necessary to definitively diagnose the OC lesion. The most common radiographic findings include:

1. Flattening and indentation of the caudal aspect of the humeral head
2. Alterations in the contour of the glenoid cavity with a subchondral cystic radiolucency
3. Osteophytes at the caudal and cranial aspect of the glenoid cavity (Figure 5.52)
4. Subchondral bone sclerosis



Figure 5.51. Thumb pressure applied just cranial to the infraspinatus tendon may elicit a painful response in horses with shoulder osteochondrosis.

5. Remodeling of the humeral head and glenoid cavity (Figure 5.52)[67]

Less common findings include osteophytes and subchondral bone cysts associated with the humeral head.[74] Intra-articular free bodies are uncommon, but when present they settle in the cranial and caudal cul-de-sacs of the joint.[72] Centrally located glenoid sclerosis and small glenoid cysts can easily be overlooked.[32]

The normal ultrasonographic anatomy of the SH joint has been described, and humeral head OC has been diagnosed using ultrasonography; however, the evaluation of the glenoid is difficult.[92] Nuclear scintigraphy may be used to identify subtle lesions.[32,54]

Arthroscopy may be useful to make a definitive diagnosis in cases in which the lameness is localized to the SH joint with intrasynovial anesthesia but a lesion is not identified on radiography or scintigraphy. The conclusions drawn in one retrospective study of 15 horses with subtle osteochondral lesions in the SH joint suggested that a combination of the physical examination, radiology, scintigraphy, and arthroscopy may be necessary to diagnose subtle osteochondral lesions of the SH joint.[32]

Treatment

Rest and confinement may be considered for horses with mild to moderate radiographic changes that are not intended for athletic performance.[52,67,74] Of the 17 horses treated with rest, the treatment was considered moderately successful in 7 cases.[74] In another case series of 3 horses with isolated subchondral cysts in the glenoid, HA was injected into the affected joint with

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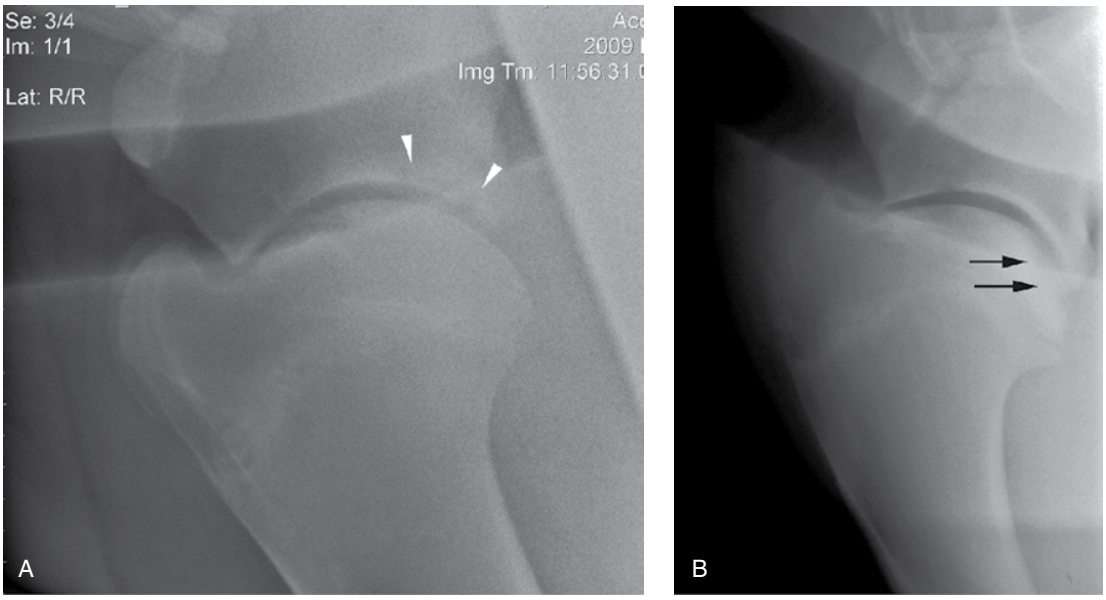


Figure 5.52. (A) An OC lesion of the caudal aspect of the glenoid cavity (arrowheads). This horse was grade 3 out of 5 lame. (B) An OC lesion on the caudal humeral head (arrows). This horse was lame at the walk and had significant muscle atrophy.

moderate success.[38] In a more recent report of 14 horses treated conservatively, 7 became sound with conservative treatment.[52]

Surgical cases should be selected carefully because of the generalized pathologic changes that are present in many cases. However, surgery benefits some horses even when secondary changes are present.[10,29] With very severe degenerative changes, the prognosis is poor and surgery is not recommended.

While arthroscopic surgery is the preferred approach for treating these lesions,[10,71] the technique is not easy, and it is particularly difficult in adult horses. In most instances the cartilaginous changes extend beyond the limits of the subchondral abnormalities seen on radiographic examination (Figure 5.53). Problems associated with arthroscopic surgery in the shoulder usually involve inadequate access to medially located lesions and debridement of large lesions.

Prognosis

Generally the prognosis with rest or surgery is considered guarded, but this is somewhat dependent on the horse's intended use and the severity of the OC lesion. Horses with mild to moderate OC have the best prognosis, and those with severe lesions usually remain lame. In one report on the outcome of arthroscopic surgery on 11 horses, 7 became athletically sound,[10] and follow-up radiographic examinations on those 7 indicated remodeling and improved contour of the glenoid and humeral head. In another study, long-term follow-up on 35 horses with OC of the shoulder treated arthroscopically found that 16 of 35 were successful.[40] A more recent study of 32 horses with shoulder OC (16 with bilateral lesions) also reported a poor outcome. In this study, the lesions were graded mild, moderate, or severe using a system that took into account lesion size and lesion severity.[52] However, there was no difference in conservative versus arthroscopic treatment.



Figure 5.53. Postmortem view of the large OCD lesion of the caudal humeral head depicted radiographically in Figure 5.52B. Arthroscopic access to medially located lesions or debridement of large lesions such as this can be difficult and often results in a poor prognosis. *Source*: Courtesy of Dr. Gary Baxter.

OSTEOARTHRITIS (OA) OF THE SCAPULOHUMERAL JOINT

OA of the SH joint is a relatively uncommon condition that can affect a variety of breeds, ages, and uses of horses.[32,40] Reports found a higher incidence of OA in Shetland ponies, miniature horses, and Falabella ponies, with a possible congenital cause.[19,22,53,76]

Although the recognition and treatment of conditions associated with SH joint have improved, the diagnosis of specific problems related to this joint remain a challenge.[32] As a result, some may require extensive diagnostic imaging, including arthroscopy, to make a definitive diagnosis. Unfortunately, the delay in recognition of the

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cause may increase the likelihood of the development of secondary OA.[72,93]

Etiology

OA of the SH joint can have multiple causes. In younger horses a developmental role due to OC may be the cause, whereas in older horses trauma may be causative.[32] A radiographic study comparing Shetland ponies diagnosed with OA, skeletally mature Shetland ponies without a history of lameness, and skeletally mature horses found that ponies had a flattening (dysplasia) of the glenoid contour, which was thought to make them more susceptible to the development of OA.[19] A recent study showed that 6 of 20 ponies had radiographic evidence of dysplasia with diagnosed shoulder OA.[22] Other case reports of SH joint OA have indicated that the glenoid cavity is relatively dysplastic in Shetland ponies.[53] Other causes of OA include intra-articular fracture and injury to the joint capsule resulting in synovitis and capsulitis.

Clinical Signs

A history of trauma is relatively common. In one report, 8 of 15 horses that had subtle OC lesions in the SHJ had a history of a traumatic insult.[32] Where mild swelling may be apparent over the shoulder region in the acute case, muscle atrophy is variable and may not reflect chronicity or severity of the lameness.[38,41] Disuse atrophy of the extensor carpi radialis muscle may be apparent in the affected limb in some cases, and an upright narrow foot may also be observed in the affected limb.[32] In contrast, in another study, only a small proportion (fewer than 5%) of the cases with shoulder problems had a smaller foot on the affected limb, although a severely lame horse may have abnormal toe wear.[38,41] Deep thumb pressure applied just cranial to the tendon of the infraspinatus muscle may elicit a painful response in young and light muscled mature horses. Upper limb manipulation (flexion, extension, adduction, and abduction) may also result in a painful response.[41]

The signs of lameness are typical of shoulder lameness, and the degree depends on the severity of the problem. At a trot, the height of the foot flight and the flexion of the carpus are decreased during the swing phase of the stride compared to the contralateral limb. The cranial phase of the stride of the lame limb is often shortened, and as the lame limb is advanced, a prominent lifting of the head and neck occurs on the affected side.[41] Additionally, the horse appears to "fix" the SH joint on the affected side. The lameness grade in one study ranged from subtle and intermittent to 4 on a scale of 5.[32]

Diagnosis

Diagnostic anesthesia can assist in localizing the problem to the shoulder region.[32,38,41,85] Radiography, ultrasound, nuclear medicine, and/or arthroscopy may be required to make a definitive diagnosis of the problem.[32,38,78] Radiography, although important in evaluating abnormalities of the shoulder joint, often underestimates the full extent of the changes involving the glenoid cavity and the humeral head.[38,40] Radiographic changes identified in one report in horses with subtle lesions in the SH joint included glenoid sclerosis, focal glenoid lysis, glenoid cysts, and alteration in the humeral head contour.[32] In another report a flattening of the glenoid without changes in the contour of the humeral head was seen in Shetland ponies.[19] Other radiographic changes associated with OA include osteophyte and enthesophyte formation (Figures 5.54 and 5.55). Contrast arthrography may improve the chances of identifying a SH joint lesion.[41,72]

The normal ultrasonographic anatomy of the shoulder region has been described.[100] Although the detection of humeral head OC has been reported, its major value appears to be in identifying injury to the soft tissue support structures surrounding the SH joint.[38,78,100] Evaluation of the glenoid cavity with ultrasound also appears limited.[32] Ultrasonography has been used to diagnose the condition in Shetland ponies when radiographs were non-diagnostic and only revealed dysplasia or flattening of the glenoid cavity. Joint distension and capsulitis provide the diagnosis.[53]

Nuclear medicine may be useful to diagnose a subtle shoulder lameness with focal intense uptake of the radioisotope, most commonly in the humeral head.[31,32]

Treatment

Conservative treatment may be indicated in horses that have no radiographic lesions and that respond favorably to intrasynovial anesthesia. Treatment involves rest, controlled exercise, anti-inflammatory drugs, and relevant intra-articular therapy including corticosteroids, HA, and IRAP. Rest periods may be as short as a few weeks to 3 months.

Horses that do not respond to the conservative treatment or have radiographic or scintigraphic changes and

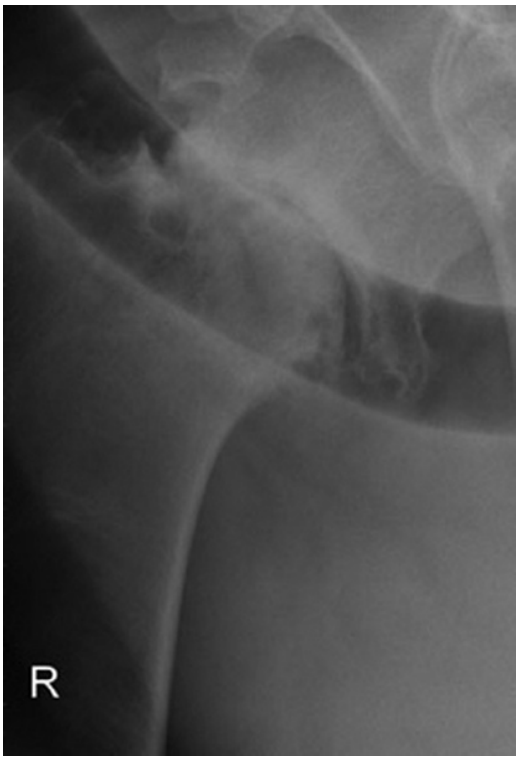


Figure 5.54. Severe OA of the scapulohumeral joint. *Source*: Reprinted with permission from Redding and Pease.[78].

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Figure 5.55. Radiographs of a subluxated left shoulder in a miniature horse that was lame at the walk. The joint had significant radiographic signs of OA. *Source*: Courtesy of Dr. Gary Baxter.

have responded favorably to intrasynovial anesthesia are candidates for arthroscopy.[32] Localized cartilage damage is debrided down to healthy subchondral bone, and osteochondral fragments are removed if they are loose.

There are reports of performing scapulohumeral arthrodesis in miniature horses to reduce morbidity in those with severe shoulder dysfunction.[6,89] A narrow DCP was placed across the cranial surface of the scapula, and the humerus with screws placed in lag fashion across the joint through the plate.[89] This procedure improved the horses' lameness by several grades, improving morbidity.

Prognosis

The prognosis appears favorable for horses without radiographic apparent lesions that respond favorably to intra-articular anesthesia and are treated conservatively with rest and intra-articular therapy.[40] Those with obvious radiographic evidence of OA generally do not respond well.

The prognosis for horses treated by arthroscopy appears good for subtle cartilage lesion. In the case of SH joint OA due to shoulder dysplasia in ponies, the prognosis does not appear as favorable. In a report of 20 ponies, 6 were euthanized for continuing severe lameness, and the other 14 remained lame.[22] In two other case reports, ponies remained lame despite concurrent conservative treatments.[53,76]

LUXATION OF THE SCAPULOHUMERAL (SHOULDER) JOINT

Luxation or subluxation of the SH joint is uncommon in horses. In a survey of 128 cases of suspected shoulder lameness in horses, only 2 cases had shoulder luxation.[38]



Figure 5.56. Cranial-caudal radiograph of the same miniature horse in Figure 5.55. The humerus and scapula were positioned more laterally than normal. *Source*: Courtesy of Dr. Gary Baxter.

Ponies appear to be at a greater risk for this condition.[8,23,40,60,101] The head of the humerus can displace in several directions, including cranial,[63,101] craniolateral, lateral, craniomedial, and medial (Figure 5.56).[23,40,50] When the humerus becomes luxated, it displaces proximad due to the contraction of the muscles surrounding the SH joint, and the direction of the luxation appears to depend on the direction of the inciting injury.

Of the cases presented in which the direction of the luxation was documented, lateral luxation with proximal displacement was most common.[40,60,63] Because the equine SH joint is stabilized by a ball-and-socket articulation, the tendons from surrounding muscles, and the glenohumeral ligaments within the joint capsule, it is not surprising that luxation of the joint results in considerable damage to these structures.[23,40] In one report arthroscopic examination following closed reduction of a luxated SH joint of 2-weeks' duration revealed bony and soft tissue debris, a cartilage defect, and a tear in the joint capsule.[63]

Etiology

Luxation of the SH joint generally occurs following excessive (forceful) trauma to the shoulder region such

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as may occur from a fall, while a horse is attempting to jump a fence, or as a result of a horse pulling and twisting a flexed limb while the foot is caught.[23,60,63,101] A report documented SH joint luxation in a septicemic foal with multiple joint laxity,[50] another cause was a rough recovery from general anesthesia,[69,103] and luxation of the SH joint has occurred following removal of a large supraglenoid tubercle (SGT) fragment.[12,16]

Clinical Signs

A history of trauma with an acute onset of severe lameness is common.[3,12,16,60,101] At presentation, horses typically exhibit a non-weight-bearing lameness. An abnormal stance may also be apparent with the elbow and carpus held semiflexed and the distal limb adducted or abducted, depending on the direction of the luxation. The distal limb is adducted when the humerus is luxated laterally, craniolaterally, or cranially or is slightly abducted if the humerus is luxated medially. A variable amount of swelling is present in the shoulder region, depending on the length of time since injury.

Muscle atrophy may be most prominent in chronic cases. One report found prominent muscle atrophy of the infraspinatus and supraspinatus muscles 2 weeks following luxation of the SH joint.[63] Distortion of the normal anatomic landmarks at the shoulder region may be apparent if swelling is not excessive, which may aid in determining the direction of the luxation. With lateral or cranial luxation, the greater tubercle and head of the humerus may be most prominent, and with medial displacement, the lateral lip of the glenoid cavity can be palpable.[63] Affected horses generally violently oppose upper limb manipulation.[23,60]

Diagnosis

Radiographs should be taken to confirm the diagnosis and rule out fracture. Generally an adequate study can be obtained in the standing horse, and the mediolateral view is thought to be the most informative.[38] The addition of the craniocaudal oblique projection will allow a better assessment of the direction of the luxation.[39] Stressed views may assist in diagnosis. Ultrasound examination may also be used to assess the extent of injury to the soft tissues supporting the SH joint.

Treatment

Ideally, the luxation should be corrected as soon as possible. General anesthesia is required in most cases, although there is one report of correction of a SH joint luxation using sedation in a 5-day-old foal with multiple joint laxity.[50] In foals, pulling the affected limb into extension while an assistant pushes or pulls the humeral head back into position will generally suffice. The foal may have to be stabilized with counterpressure applied the chest or axilla.

In the mature horse, the patient can be placed in lateral or dorsal recumbency. In lateral recumbency the body can be anchored to a fixed object, and a tension-creating device that is attached to the radius or pastern region to apply traction is used while the operator forces the shoulder back into position. If the horse is placed in dorsal recumbency, a hoist can be used to apply traction and the body weight is sufficient to provide counterweight.[69,101] An audible click may be heard when the humeral head reduces, after which it should be possible to freely manipulate the joint.[23,60] In acute cases, reduction may be accomplished very quickly. In more chronic cases, gradually increasing traction may be required with limb manipulation before reduction is achieved.[63] Arthroscopic examination of the SH joint following closed reduction may improve the outcome particularly if there is evidence of bony debris within the joint on radiography.[63] As such a radiograph should be taken of the SH joint after the reduction is complete.

Recovery from anesthesia should be assisted in all cases. Following recovery the horse should have strict stall rest for 2 months to allow healing of the joint capsule and surrounding soft tissue structures.[23] Anti-inflammatories and intra-articular therapies are indicated. Reoccurrence of the luxation does not appear to be a problem in the horse.[101]

Repair of a case of chronic subluxation of the SH joint in a minishetty stallion by arthrodesis has been reported.[8] The chronicity and bony changes acquired provided a recommendation for arthrodesis. Arthrodesis of the shoulder can usually be performed in miniature horses with a single broad or narrow LCP (Figure 5.57).

Prognosis

The prognosis for SH joint luxation is considered good for return to soundness following closed reduction and an adequate rest period, in cases in which there is not a complicating fracture. In one study, all 6

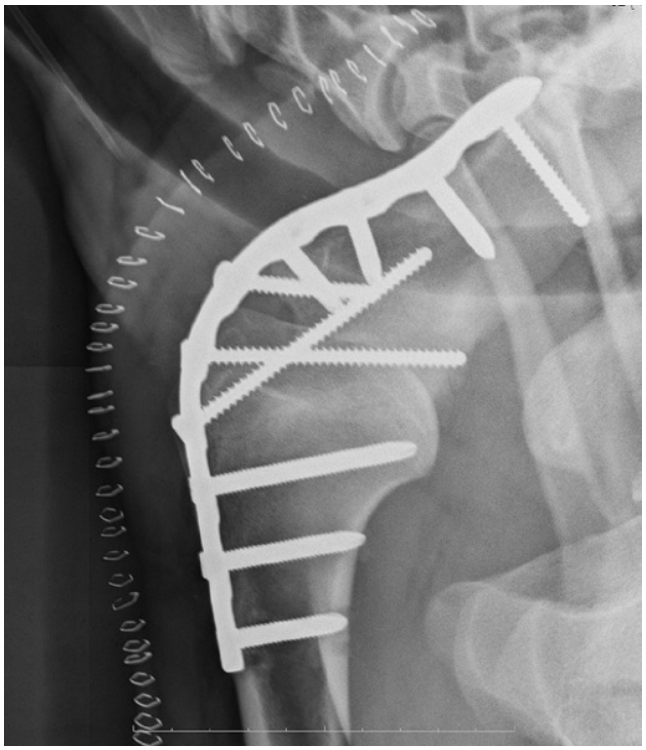


Figure 5.57. Treatment of a chronic shoulder luxation in a miniature horse with an LCP and transarticular screws.

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cases reported with SH joint luxation treated by closed reduction returned to soundness.[12,16,23,40,60,63,101,103] The prognosis for larger horses may also be good. One report of shoulder luxation in a Thoroughbred filly has been described.[63] The filly was sound for light work 8 months after closed reduction and arthroscopic examination of the joint. Long-term successful outcome was achieved in a racing Thoroughbred filly that luxated the joint during recovery from anesthesia.[69] It has been suggested that the prognosis may be improved and more accurately predicted if arthroscopy is used following closed reduction to remove any subsequent bone fragments.[23] Arthrodesis of the SH joint in a minishetty stallion with chronic subluxation resulted in a good outcome with minimal lameness at a trot 6 weeks post surgery.[8]

SUPRASCAPULAR NERVE INJURY (SWEENY)

Suprascapular nerve injury resulting in atrophy of the supraspinatus and infraspinatus muscles and shoulder joint instability can affect any age or breed of horse.[14,34] The condition was originally reported as most commonly affecting draft breeds and was believed to be associated with repeated trauma to the shoulder region from poorly fitted harness collars. With the decline in draft breeds, the condition is most commonly seen in horses as result of trauma to the shoulder region. The term "sweeny" has been defined as atrophy of the shoulder muscles in horses and is a commonly used synonym for suprascapular nerve paralysis.[14]

Etiology

Trauma to the suprascapular nerve as it passes over the cranial thin border of the scapula is believed to be the cause.[33,34,62] The suprascapular nerve originates from the sixth and seventh cervical spinal segments and passes via the brachial plexus to innervate the supraspinatus and infraspinatus muscles that overlie the scapula.[36] As the nerve reflects around the cranial edge of the scapula, it passes beneath a small but strong tendinous band. In this position the nerve appears to be most susceptible to direct trauma and compression against the underlying bone.[33–35] In horses without clinical evidence of muscle atrophy, there is also histologic evidence of chronic demyelination and remyelination at the point where the suprascapular nerve reflects over the cranial edge of the scapula. These findings suggest there may be chronic nerve compression caused by constriction of the small tendinous band resulting in subclinical neuropathy. It has been suggested that the chronic neuronal injury may make the suprascapular nerve more susceptible to acute trauma and the development of clinical signs or that spontaneous development of the condition, without a traumatic insult, may be possible.[33–35]

Clinical Signs

A history of trauma to the shoulder region is relatively common. The clinical signs vary depending on the extent of the nerve damage and the duration of the condition prior to examination. Shortly after the injury, horses often exhibit severe pain and are reluctant to bear weight on the affected limb.[35] As the pain subsides and the horse begins to bear weight, a pronounced lateral instability (excursion) of the shoulder joint (shoulder slip) during weight-bearing is observed. This sign is usually seen within 24 hours of injury and is most apparent as the horse is walked slowly toward the examiner.[35] The instability is the result of loss of the stabilizing function of the supraspinatus and infraspinatus muscles, which serve as the major lateral support for the shoulder.[62] It has been suggested that this outward excursion of the scapula during weight-bearing may cause intermittent stretching of the suprascapular nerve leading to continued trauma and perpetuation of the paralysis.[1] The acute lameness may be followed by complete recovery over a 2- to 3-month period, or denervation atrophy of the supraspinatus and infraspinatus muscles may become evident. Following injury to the nerve, muscle atrophy usually becomes apparent as early as 10–14 days after injury.[33] Once atrophy begins, the scapular spine becomes more prominent due to the loss of the muscles cranial and caudal to it (Figure 5.58).

Diagnosis

A presumptive diagnosis of suprascapular nerve injury can be made from the clinical signs and the history of trauma to the region. Radiographs of the region should be obtained to rule out a fracture or OA of the shoulder joint. Electromyographic evaluation (EMG) of the supraspinatus and infraspinatus muscles confirms selective suprascapular nerve injury.[40] For the EMG studies to be most meaningful, they should be performed a minimum of 7 days after injury. EMG findings of denervation of muscles supplied by other branches of the brachial plexus should prompt further neurologic evaluation to rule out injuries to the brachial plexus, spinal cord disease at the sixth and seventh cervical segments, and other diseases such as equine protozoal myelitis.[14,33]



Figure 5.58. Prominent atrophy of the supraspinatus muscle is evident in this horse with suprascapular nerve injury.

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Treatment

Both conservative and surgical treatments have been described for management of horses with suprascapular nerve injury.[5,15,35,68,87,102] Initial treatment in all cases is directed toward reducing inflammation in the region of the suprascapular nerve. Stall rest with the administration of NSAIDs and the topical application of cold hydrotherapy or ice packs and topical anti-inflammatories may produce a resolution of the clinical signs.

Conservative Treatment

When conservative treatment is selected, stall rest is continued until shoulder joint stability returns, followed by confinement to a pasture for an additional 2–4 months. In one study the mean time for resolution of the gait abnormality was 7.4 months, and 7 of 8 horses evaluated had a complete resolution of the joint instability.[35] The conclusions from this study were that stall rest appeared to be a viable alternative to surgery for the treatment of suprascapular nerve injury. It can be speculated that surgical decompression may result in a faster return to athletic function.

Injection of a corticosteroid into the shoulder joint may also be beneficial. In one report, 6 horses with suspected suprascapular nerve injury were treated successfully with intra-articular corticosteriods.[68] However, it is uncertain whether these horses had a true neuropraxia of the suprascapular nerve or scapulohumeral joint inflammation that caused disuse muscle atrophy, or both. Anti-inflammatory therapy is recommended. Therapeutic ultrasound has also been suggested as a treatment.[15]

Surgical Treatment

Surgical decompression of the nerve can be considered in patients that continue to exhibit signs of suprascapular nerve dysfunction for 10–12 weeks. This time frame is based on the rate of peripheral nerve regeneration (1 mm/day) and the distance from the site of nerve injury on the cranial border of the scapula to the infraspinatus muscle (7–8 cm). Using these parameters nerve function should return in 10–12 weeks.[87] In horses that show no improvement after 12 weeks or more of rest, entrapment of the suprascapular nerve by scar tissue between the overlying ligament and the cranial border of the scapula is likely. Although the time frame for reversing neurogenic muscle atrophy in large animals is not known, in humans, neurogenic muscle atrophy becomes dramatic after 4 months with loss of up to 80% of muscle fiber volume. It is generally accepted that functional reinnervation is unlikely after 12 months.[56,102] After that, irreversible changes within the muscle occur, and it is replaced by fibrous tissue.

Surgical decompression involves removing a small piece of bone from the cranial border of the scapula underlying the nerve (Figure 5.59).[87] By removing the

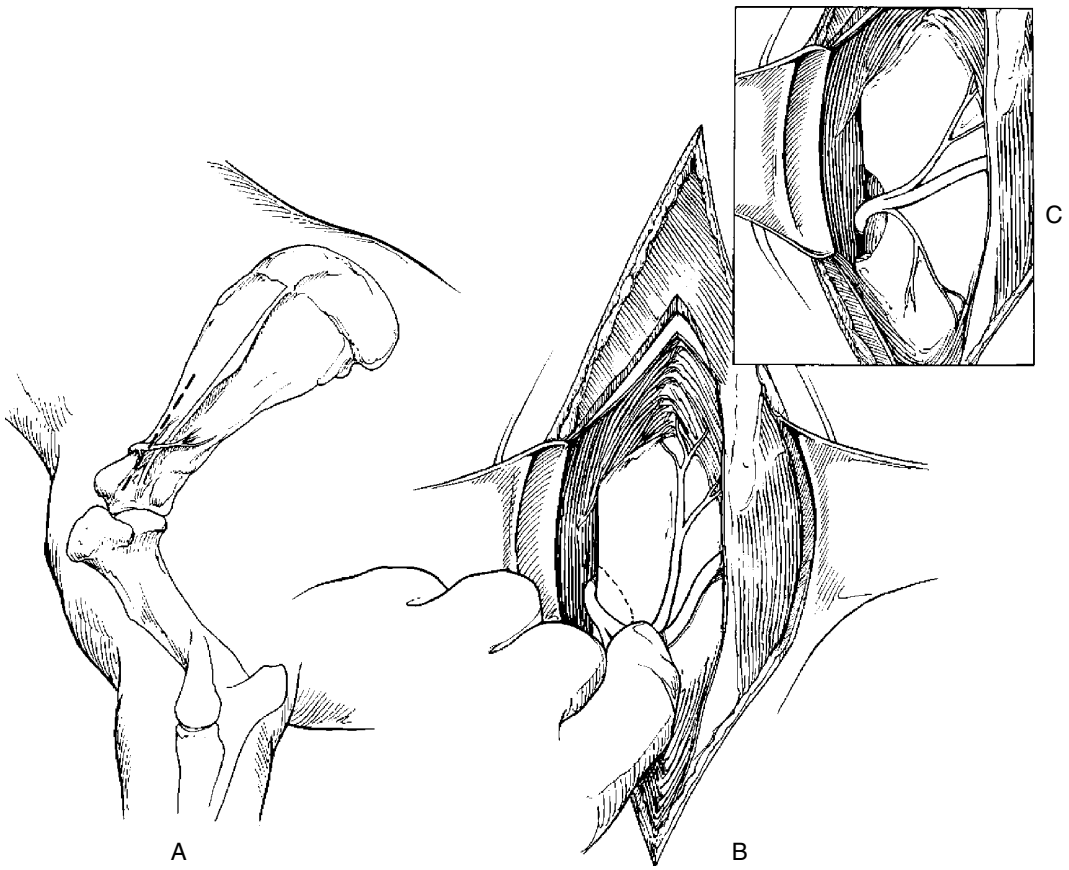


Figure 5.59. (A) Location of the skin incision (dotted line), centered over the suprascapular nerve and cranial to scapular spine. (B) The suprascapular nerve is identified, and a small crescent-shaped piece of bone is removed from the cranial border of the scapula (dotted line). (C) The bone has been removed and tendinous band resected.

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notch of bone, it is thought that the nerve is decompressed, allowing for reinnervation. Complications of the surgery include the possibility of a glenoid fracture arising from the site of the notched bone.

Prognosis

The prognosis for return to soundness in horses with suprascapular nerve injury is favorable for both conservative and surgical treatments. In one report 7 of 8 horses with suprascapular nerve injury treated conservatively were able to return to their intended use.[35] All eight initially had pronounced shoulder joint instability during weight-bearing, and joint stability returned within 3–12 months. The conclusion was that rest alone carries a good prognosis for recovery of a normal gait and return to performance; however, the recovery period may be prolonged.

A high percentage of surgically treated horses are expected to return to performance. In one report 18 of 20 horses with suprascapular nerve injury returned to soundness.[86] An important and severe complication associated with the surgery is the postoperative occurrence of fracture of the SGT and complete subluxation of the SH during anesthetic recovery.[87] Horses that develop severe atrophy of the supraspinatus and infraspinatus muscles prior to surgery may not regain normal muscle mass, and some atrophy will remain in the proximal one-third of the scapula.

FRACTURES OF THE SCAPULA

Fractures of the scapula are uncommon in horses. This is in part due to the scapula's proximal location, its close proximity to the ribs, and the large protective muscle mass overlying the region.[3] Although scapular fractures occur in all ages and breeds of horses, young (less than 4 years) intact males appear more prone to sustain the fracture.[3] Fractures of the scapula in racing Thoroughbreds and Quarter horses have been extensively described. The incidence is low at 0.39 and 0.98/1000 starters, respectively.[96] In another recent study evaluating fatal musculoskeletal injuries in racing Quarter horses, scapular fractures accounted for 8% of the injuries.[83]

Fractures of the scapula can involve the spine, SGT, body, neck, and glenoid cavity. Of these, fracture of the SGT is most common. Although simple scapular fractures are most common, comminuted fractures also occur. Bone sequestra may also develop following fractures of the scapular spine, particularly when a penetrating wound results in a comminuted fracture.[9,37] Stress fractures of the scapula have been recently described in Thoroughbred racehorses.[28] A high incidence of suprascapular nerve injury occurs following fractures of the neck of the scapula. Injury to the subscapularis nerve and brachial plexus is a rare complication associated with fractures of the scapula.

Etiology

Scapular fractures are usually caused by direct trauma or following falls during racing or jumping.[30,37] Scapular fractures resulting from racing or race falls at high speeds tend to be comminuted.[96] Penetrating wounds to the lateral shoulder region can also cause fractures of the spine of the scapula.[30,37] The California racing population reported 73 Thoroughbreds and 28 Quarter horses sustained scapular fractures while racing or training. There was a predominance of male horses and right shoulders, and the injury occurred early in their careers. Quarter horses appeared to sustain the injury more frequently.[96]

Clinical Signs

Horses frequently present with a history of trauma and signs of lameness ranging from mild to non-weight-bearing.[3,9,40] Horses with an acute fracture of the scapular spine usually exhibit focal swelling, will bear weight, and usually exhibit a mild to moderate lameness at exercise (Figure 5.60). Deep palpation over the fracture site usually elicits a painful response.

Evidence of penetrating wounds may be present. Chronic cases of scapular spine fractures may present with a draining tract or a history of the development of a recurrent draining tract as a result of a penetrating wound and sequestration of bone fragments.[9,37] Horses with fractures of the body or neck of the scapula are



Figure 5.60. Prominent swelling (arrow) located over the lateral right shoulder region of a young horse with a complete fracture of the body of the scapula. The foal was lame at the walk and carried the limb further axially than normal. *Source*: Courtesy of Dr. Gary Baxter.

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usually reluctant to bear weight initially, have difficulty advancing the affected limb, and often have swelling over the fracture site. Deep palpation and limb manipulation usually elicit a painful response.[3,20] Fractures of the scapular neck may also result in secondary suprascapular nerve injury and signs of paresis in some cases. Chronic cases of more than 3 weeks' duration usually exhibit varying degrees of atrophy of the muscles in the shoulder region and proximal antebrachium.[40] As time passes, swelling may become more apparent in the lateral shoulder region as a result of muscle atrophy, healing soft tissues, and callus formation. A swinging and support limb lameness is observed at a trot. Horses sustaining these fractures should also be assessed for other problems such as fractured ribs and intrathoracic trauma.[20]

Diagnosis

Radiographs of the region are needed for a definitive diagnosis. Medial to lateral, ventrodorsal, and oblique cranial to caudal radiographic projections reveal most fractures of the scapula. The study can be done while the horse is standing in most cases. Some fractures through the body of the scapula can be difficult to image because of the superimposition of the ribs and vertebrae over the scapula; in these cases the limb must be protracted to obtain a diagnostic film.[40] If a fracture is suspected but not observed, the horse should be confined to a stall and radiographed again 10–14 days after injury.[37] A scintigraphic exam can be performed prior to this to assist in the diagnosis. Some large, heavily muscled horses in severe pain may require general anesthesia to obtain diagnostic radiographs. Stress fractures of the scapula are diagnosed by nuclear scintigraphic exam and ultrasonography after lower limb analgesia has been performed to rule out a lower limb issue in racehorses that are acutely lame after racing.[28] Suprascapular nerve injury can be difficult to detect by physical exam alone in the non-weight-bearing limb. Electromyography (EMG) can be helpful in assessing nerve damage at least 7 days after the injury has occurred.[20]

Treatment

Conservative Treatment

Fractures of the scapular spine generally do not require surgical intervention, and most heal by bone union. On the other hand, fractures following a penetrating wound may develop bone sequestra; in these cases surgery is recommended. Some minimally displaced nonarticular fractures of the scapular body and neck or stress fractures may also be treated conservatively with a good result.[13,28,40] Providing stall rest for several months, taping the shoulder to the body wall to prevent abduction of the limb, and slinging cooperative animals have been used successfully in some cases without articular involvement.[28,37,40]

Surgical Treatment

Transverse fractures of the body and proximal neck can be surgically treated with internal fixation in young animals.[20,47,90] Stabilization is achieved with two dynamic compression (DCP) bone plates applied cranial and caudal to the scapular spine in the angle formed by the spine and the body.[20,47,90] Longitudinal fractures have been repaired using several narrow DCP placed across the fracture line staggered along the scapula.[55]

Severely comminuted fractures or fractures of the distal neck and those extending into the glenoid are very difficult to treat surgically with internal fixation. The configuration of the fracture and its close proximity to the shoulder joint do not permit application of orthopedic implants. Euthanasia is recommended in horses with extensive joint involvement and/or severe joint instability.[13]

Prognosis

The prognosis is good for fractures involving the scapular spine with or without surgery for removal of a sequestrum, and most horses can return to performance after an adequate rest period. Young horses with nonarticular simple or minimally comminuted fractures of the body and proximal neck have a good prognosis for soundness after application of internal fixation. At the 1-year follow-up of 2 horses that underwent internal fixation, complete healing and return to full function were noted.[20,47] A longitudinal fracture of the scapula in a young horse resulted in soundness at 6 months after internal fixation.[55] In adults, nondisplaced fractures of the body or neck may heal satisfactorily with conservative therapy.[40] Horses with complete fractures in the distal neck, articular fractures, and severely comminuted fractures all have a poor prognosis for return to performance.[13,37]

FRACTURES OF THE SUPRAGLENOID TUBERCLE (TUBEROSITY)

Fractures of the SGT can occur in a variety of breeds of horses and horses with a variety of uses.[15,16,27,75] They are relatively common, accounting for about 30%–50% of reported scapula fractures.[37,57] Fractures of the SGT are often simple, intra-articular, and usually affect horses less than 2 years of age.[16,57,75] In contrast, one report on STG fractures in 9 horses found 4 with comminuted fractures and 6 of the 9 were in horses 3 years or older (range 3–13 years).[37]

Anatomically, the SGT serves as the proximal attachment for the biceps brachii muscle and two glenohumeral ligaments that support the SH joint. The coracoid process, which is the medial projection of the tubercle, serves as an attachment for the coracobrachialis muscle. The SGT and the coracoid process develop from a single center of ossification and fuse with the cranial portion of the glenoid cavity and the main body of the scapula at about 10–12 months of age.[91] Because the fracture plane often courses along this growth plate, it has been suggested that this fracture may result from a separation of the physis of the SGT in young horses.[57]

Etiology

Fracture of the SGT is most frequently associated with trauma to the cranial shoulder region. Because of its superficial location, it appears that the SGT is most susceptible to injury. In two reports most horses with

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SGT fracture had a history of falling or direct trauma to the shoulder.[37,75] Overflexion of the shoulder leading to increased tension on the biceps brachii and coracobrachialis tendons that attach to the SGT has also been proposed as a mechanism for this fracture.[2] Fracture of the SGT has occurred in several horses with suprascapular nerve paralysis following surgical removal of a piece of bone from the cranial border of the scapula.[15,16]

Clinical Signs

A history of trauma resulting in severe lameness that improves rapidly is common.[16,37,75] This rapid improvement may be the reason that this fracture is not initially diagnosed in some instances and that recognition of the problem only occurs after the horse remains lame longer than expected or when muscle atrophy becomes prominent.[37,75]

In acute cases swelling is usually apparent over the point of the shoulder, and palpation generally elicits a painful response. Crepitation may also be appreciated in some cases. At a walk, the horse typically retains the ability to extend the SH joint, but the cranial phase of the stride is markedly shortened. A lameness score of 3–4 out of 5 is typical in the acute phase. As time passes, swelling over the point of the shoulder diminishes, and palpation may reveal a firm, nonpainful swelling over the point of the shoulder.[37] Movement of the tubercle can be perceived in some cases. Varying degrees of muscle atrophy are usually apparent in chronic cases.[37] Some horses may also exhibit signs of suprascapular or radial nerve paralysis. Intra-articular anesthesia of the SH joint may not improve the lameness.[37]

Diagnosis

Radiography is required to make a definitive diagnosis of the fracture (Figure 5.61). Generally, the fracture is simple or comminuted and intra-articular.[15,37,75] Calcification of the biceps tendon may also be associated with fracture of the SGT.[37] Electromyographic studies are sometimes needed to rule out neurogenic atrophy of the affected muscles.

Treatment

Several options can be considered for management of SGT fractures. The selection of the management approach depends on the nature and duration of the fracture, economics, and the expectation of performance level of the horse.

Conservative management consisting of the administration of NSAIDs and prolonged stall rest for 3–4 months followed by pasture turnout for 6–9 months may be selected in some cases. Horses with nonarticular or minimally displaced intra-articular fractures respond best to this approach and may be able to return to their intended use, depending upon the degree of OA that may develop.[16,37,75] The use of platelet-rich autologous plasma has been described as an adjunctive therapy to conservative management.[21]

Surgical management of horses with SGT fractures consists of either surgical removal of the SGT fragment or internal fixation with or without transection of the biceps brachii tendon. The goal of surgery is to prevent the development of secondary OA caused by joint incongruity.

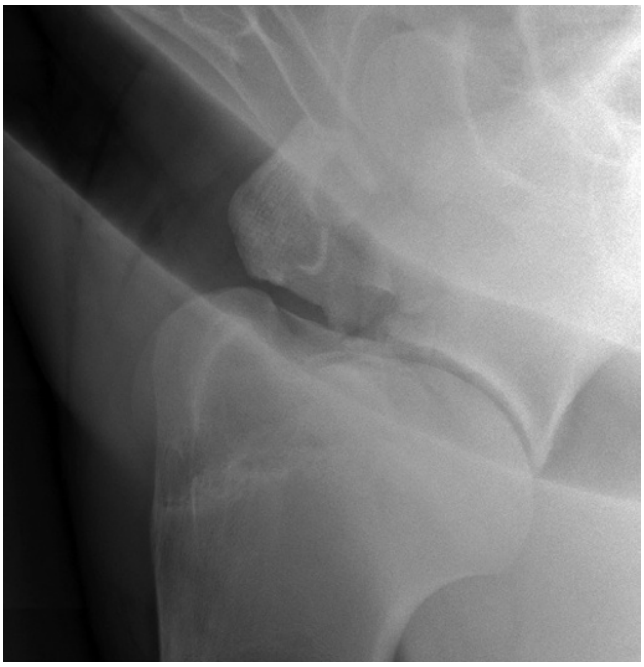


Figure 5.61. Fracture of the supraglenoid tubercle of the scapula with moderate displacement.

Removal of the fragment involves dissection of the tendinous attachments of the biceps brachii and coracobrachialis muscles of the SGT. Excising the fractured SGT decreases the pain created by fracture movement and prevents further joint damage that develops from impingement of the fragment on the articular surface of the humeral head.[9] Surgical excision of the SGT appears to be best suited for most chronic fractures and for comminuted articular fractures.[75,98] In one report, 4 of 7 horses returned to performance, including 1 racing Thoroughbred that had a successful career at a reduced performance level. Caudal luxation of the scapula can occur with excision of the fractured SGT.[15]

Several methods of internal fixation consisting of various combinations of interfragmentary compression with lag screws and tension band wires have been used. Cancellous bone screws placed in lag fashion across the fracture gap were reported to be unsuccessful in 2 horses.[57] Internal fixation using Kirschner wires in combination with cerclage wire placed in a figure-8 pattern to stabilize the fracture fragment was used successfully in one case.[57] Stab incisions made through the tendon of the biceps brachii muscle to place bone screws in a lag fashion was also used successfully in one report.[27] Bone plating of SGT fractures has also recently been advocated. Internal fixation has limited success, particularly in heavily muscled horses; the porous bone in the scapular neck does not hold screws adequately, fracture reduction is difficult, and fixation fails due to the tension exerted by the biceps brachii tendon.[2,15,16] Partial or complete transection of the biceps tendon has been advocated to eliminate the tension on the fracture fragment and prevent implant and bone failure.[2,15] Trauma and damage to the suprascapular nerve can occur due to the surgical procedures mentioned.

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Prognosis

A good prognosis for return to performance can be expected for conservative management of horses that have nonarticular fractures or minimally displaced articular fractures of the SGT.[16] The prognosis for return to athletic soundness is poor for conservative management of horses that sustain simple or comminuted displaced articular fractures, but pasture soundness is possible in some.[15,37]

The prognosis following surgical excision of the fractured SGT is better than that achieved with conservative management for return to performance in horses with displaced simple or comminuted articular fractures and for chronic articular fractures.[16,37,75,98] In one report, 4 of 7 horses treated with surgical excision of the fractured tubercle returned to performance, including one racing Thoroughbred that had a successful career at a reduced performance level.[16]

Presently the prognosis for internal fixation appears best if a combination of internal fixation and complete transection of the biceps brachii tendon is used for simple acute fractures.[2,16,27] In one report, 3 of 3 horses treated by this method were sound for their intended use.[16]

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THE TARSUS

W. Rich Redding

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INTRODUCTION

The forelimb receives 60%–65% of the weight of the horse and rider during locomotion, while the hindlimbs are used primarily to generate forward momentum. Performance horses (other than racehorses) require a rearward shift in weight distribution, which allows more impulsion from the hind end. In particular, the disciplines of dressage, eventing, and show jumping are good examples of horses that need to work "off the forehand" and drive through the hind end during collection and propulsion. This change in weight distribution increases the incidence of hindlimb injuries seen in sport horses. The tarsus accounts for many of the hindlimb lameness conditions in performance horses, and the appropriate use of diagnostic imaging is critical to accurately diagnose the specific site of the problem.

Horses with hindlimb lameness may demonstrate little evidence of the source of lameness. In addition, it may prove difficult to examine the hindlimb due to the potential risk of injury, be it real or perceived. Many equine professionals (owners, trainers, and veterinarians) rightly or wrongly suspect the tarsus or stifle to be the most common source of lameness. However, the gait characteristics of hindlimb lameness are similar irrespective of whether the source of pain occurs in the proximal or distal limb. A careful and consistent clinical examination and the use of diagnostic analgesia can identify the most likely region that may be the cause of the lameness. Radiographic and ultrasonographic examinations are complementary and quite effective tools in the evaluation of both bone and soft tissue structures of the limb of the horse.[20,23,32,35,36,58,63,106,116,140,144,146] When these techniques are unable to define a specific cause of lameness, then more advanced diagnostic techniques such as nuclear scintigraphy, computed tomography (CT), or magnetic resonance imaging (MRI) may be required.[7,13,14,21,44,45,49,59,102,103,109,115,119,125,137] An accurate diagnosis and prognosis becomes critical to effectively address and rehabilitate injuries in these cases of lameness.

Strain-induced injury of both the soft tissues (tendons and ligaments) and bone (modeling) are the most common causes of orthopedic injury in athletic animals, either equine or human. Injuries of the structures of the tarsus are quite common and are diagnosed with a combination of clinical examination, diagnostic analgesia, and diagnostic imaging.[7,17,24,40,43,54–57] While clinical examination can often suggest the region of the tarsus as a clinical problem, intra-articular and/or regional anesthesia should be utilized to confirm and to more accurately isolate the source of lameness. If the lameness has been isolated to as specific an area of the tarsus as possible, then diagnostic imaging should be focused on this area. Once an accurate diagnosis is attained, the clinician can develop a directed treatment strategy and rehabilitation schedule for that specific problem(s). Imaging of the tarsus should begin with a thorough radiographic and sonographic examination. The complicated anatomy of this joint can significantly compromise a complete examination with these two imaging tools,[59,60,77] and the use of nuclear scintigraphy, CT, or MRI may be required to find the source of pain. Currently MRI is considered the gold standard for imaging the distal limbs of the horse but is not without risk to the horse because it requires general anesthesia. Because of the complexity of the tarsus, the examiner must develop a keen understanding of the normal anatomy and the types of injuries that typically can occur in the tarsal region to most effectively utilize diagnostic analgesia and imaging techniques.

DIAGNOSIS OF TARSAL LAMENESS

Exam and Palpation

An accurate diagnosis is critical to effectively manage injuries of the tarsus. The diagnosis of tarsal disorders of the hindlimb can be challenging. Often clinical signs are not obvious or specific to the tarsus, and lameness is thought to originate from more proximal on the limb. Careful examination of the distal limb can provide some indication of a specific problem that coexists with tarsal problems. Structures in the distal limb and tarsus should be palpated with the horse standing on the limb and with the limb raised. Application of hoof testers should be performed in acute cases to rule out the presence of pain associated with the foot before evaluating the rest of the limb. Some injuries may involve multiple structures, so it is critical to develop a complete routine examination protocol of the entire limb. Manipulation of the limb by flexion, extension, and rotation may provide some indication of joint(s) involvement. Flexion tests to accentuate lameness may be helpful to differentiate lower limb from the proximal limb involvement. However, the reciprocal apparatus of the hindlimb makes it virtually impossible to flex the lower limb and proximal limb separately. The degree of flexion of the distal limb, however, can be exaggerated beyond that of the proximal limb by holding the leg as close to the ground as possible (similar to how the farrier holds the limb) and selectively flexing the lower limb. Performing the distal limb flexion first before the proximal limb allows a comparison between the respective outcomes. Many horses with proximal limb involvement may respond markedly to both distal and proximal limb flexion. In general, flexion tests of the hindlimb have low specificity, which should emphasize the use of diagnostic analgesia to confirm the source of lameness.

Diagnostic Analgesia

Diagnostic analgesia should be utilized to isolate the source of lameness either by sequential regional

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analgesia (predominantly by eliminating the distal limb as the source of lameness) or by intra-articular analgesia of the distal hindlimb.[28,42,72] If the clinical examination indicates the tarsal region to be the source of lameness, then intra-articular analgesia can be performed without fear of blocking the lower limb. If the clinician has no indication as to the source of lameness, then regional analgesia should be performed starting with a low plantar (six-point) nerve block. Once the distal limb is eliminated as the source of lameness (negative response to the low plantar), then intra-articular analgesia of the tarsometatarsal (TMT) and distal intertarsal (DIT) joints should be performed. Communication between the DIT and TMT joints is variable. Previous studies found communication between of the DIT and TMT joints ranged from 8% to 30% and even found variability between both hocks of the same horse.[11,80] Surprisingly communication between the distal tarsal (DT) joints and the tarsocrural (TC) joint was found to occur in about 3% of horses.[18] Therefore, the author recommends that both DT joints be injected for both diagnostic and treatment purposes.

Intra-articular analgesia is thought to be more specific than perineural analgesia, but either technique may be misleading particularly with respect to DT joint disease. Inaccuracies in the diagnostic analgesia techniques can and do occur. For example, a negative response to DIT/TMT intra-articular analgesia does not preclude DT joint pain. Narrowing of the DIT joint space or periarticular new bone can impede or prohibit intraarticular injection. In addition, the response to intra-articular analgesia may be very poor or at best delayed in the presence of extensive subchondral bone damage. Isolating the source of pain to the proximal plantar region requires that analgesia of the deep branch of the lateral plantar nerve (DBLPN) or perineural analgesia of the plantar metatarsal nerves at the subtarsal site should be performed. This would indicate that proximal suspensory desmitis (PSD) may be the source of lameness. Lameness due to PSD should be substantially improved (65%–70%) following these blocks. However, a recent study showed proximal diffusion of contrast media after DBLPN creating the potential for false-positive results.[28] In addition analgesia of the DBLPN can influence DT joint pain, and intraarticular analgesia of the TMT joint can abolish lameness in some horses with PSD (8%).[38] Lameness of the proximal plantar region may occasionally improve following a low plantar (six-point) nerve block most likely due to proximal diffusion of the local anesthetic. One study evaluating analgesia of the DBLPN revealed that 37.5% of horses had contrast in the tarsal sheath (TS) and 25% had sufficient mepivacaine in the TMT joint to produce analgesia.[28] Therefore, a comparison between the respective outcomes of intra-articular and perineural analgesia may be useful in those horses without radiographic or ultrasonographic abnormalities. If necessary, a tibial nerve block may prove useful to distinguish between DT joint pain and PSD pain, as analgesia of the tibial nerve will result in analgesia of PSD without significantly influencing DT joint pain.

A wide range of conditions can cause TC joint pain in horses. Some of these conditions that affect the TC joint may not respond to intra-articular analgesia but in fact require perineural analgesia of the tibial and fibular nerves to demonstrate an improvement in lameness. In one study evaluating horses with TC joint pain, intraarticular analgesia improved lameness >50% in 12/17 limbs (70.6%), but <50% in 3/17 limbs (17.6%).[51] Some periarticular structures may need to be carefully evaluated with diagnostic imaging and require the use of more specific analgesic techniques to confirm their significance. For example intrasynovial injection of anesthetic into the TS is necessary to isolate a lateral deep digital flexor tendon (LDDFT) injury as the source of lameness. Some clinicians use a subtarsal nerve block to eliminate the proximal plantar region. However, when subtarsal injection around the plantar metatarsal nerves (with/without the plantar nerves) was evaluated utilizing contrast medium, it was found that the TS was injected in 40% of the limbs.[42] No proximal diffusion was identified when the perineural injection was place in the correct location outside the TS. Contrast medium was identified in only one TMT joint, suggesting that the likelihood of subtarsal analgesia influencing the tarsus is relatively small.

Anesthetizing the tibial and the deep and superficial peroneal nerves above the point of the hock should desensitize the entire distal portion of the limb. However, the horse may not be able to utilize the limb appropriately and may start to drag the toe of the desensitized limb. The tibial nerve is a large nerve, and some clinicians have suggested that it may take up to 45–60 minutes before enough diffusion has occurred before it provides complete analgesia. Blocking the stifle before the tibial nerve is completely desensitized may lead to a false positive (the horse seems to improve to the stifle block, but the improvement is actually more complete analgesia of the tibial nerve). In addition, local diffusion from the tibial injection site may result in resolution of lameness associated with gastrocnemius tendonitis.

There is a close anatomical association of the TMT joint and the proximal suspensory ligament (SL) and its innervations. The lateral plantar nerve gives off a deep branch approximately 3 cm proximal to the head of the Mt4 bone.[72] The DBLPN branches into medial and lateral plantar metatarsal nerves, which provides sensory and motor innervation to the proximal SL. A previous study of injecting of contrast medium into the TMT joint demonstrated significant amounts of contrast located plantar and distal to the TMT joint following injection.[28] It was hypothesized that this may result in perineural anesthesia of the plantar metatarsal nerves and the proximal aspect of the SL. Perhaps more importantly, these plantar outpouchings of the TMT joints may permit inadvertent intra-articular anesthesia of the TMT joint following diagnostic anesthesia of the proximal metatarsus similar to what occurs in the forelimb.

Because it is considerably easier and safer to inject the TMT joint, some clinicians prefer to block the TMT joint first. If there is no or only partial improvement in baseline lameness, the DIT joint should then be blocked. Lameness is often improved within 10 minutes, but due to the extensive pathology in some horses, it may take longer to see complete clinical improvement. A positive response to intra-articular analgesia of DT joint pain may be limited (<50% improvement from baseline lameness). When utilizing perineural analgesia of the peroneal and tibial nerves, it may take a minimum of

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20 minutes to influence DT joint pain. Occasionally the response is even more delayed, making it preferable not to proceed with further blocking for at least 1 hour. In addition, some horses with DT joint pain may respond better to intra-articular medication than either intra-articular or perineural analgesia.

Imaging of the Tarsus

The equine tarsus is a complex joint and can be difficult to accurately image due to the unique anatomic arrangement and the superimposition of many structures.[59,60,77] Many of the changes that develop in and around this joint with disease are chronic. Radiographs have proven to be useful to evaluate bony changes associated with the tarsus.[23,36,63,106,145] While soft tissue swelling can easily be seen on radiographs, accurately identifying the specific structures involved can be difficult. Diagnostic ultrasound (US) is a very useful imaging technique that can provide valuable information about the soft tissues of the joint, the articular cartilage, and the subchondral bone surfaces.[32,35,59,116,140,148] Diagnostic US and radiology are considered the minimum imaging techniques to be utilized when evaluating a specific anatomical region of the musculoskeletal system. For example, ligament and tendon injuries frequently occur at the bone–soft tissue interfaces or insertions onto bone (enthesis) with the earliest change often being early bone production. Both radiography and US are necessary to effectively evaluate the injury especially since US has proven to be more sensitive than radiology at identifying early periarticular remodeling and enthesis new bone formation. More information on tarsal ultrasonography can be found in Chapter 3.

Nuclear scintigraphy may prove useful when radiographic and sonographic examination fails to reveal significant clinical information about the source of the lameness.[44,49,59,102,103,119,137] More recently MRI is being utilized for tarsal imaging due to short-bore high field strength magnetic systems when other imaging modalities are inconclusive. MRI of the tarsus has been useful in the diagnosis of abnormalities that may not be apparent radiographically or ultrasonographically.[13,14,21,45,83,125] Abnormalities seen with MRI include subchondral bone injury of the talocalcaneal joint, bone bruising of the distal tibia, intertarsal ligament enthesopathy with associated bone edema, focal osteoarthritis (OA) in the plantar aspect of the DIT and talocalcaneal joints with localized loss of joint space and subchondral bone sclerosis, and soft tissue injuries of the collateral ligaments (CLs) of the TC and the proximal intertarsal (PIT) joints, as well as tendonitis of the deep digital flexor (DDF) tendon at the level of the calcaneus.

Radiography of the Tarsus

Radiography is the most accessible and commonly used imaging modality for the diagnosis of bone lesions of the tarsal bones such as osteochondrosis (OC), OA, or fractures. Radiographic interpretation of images of the tarsus must consider the complex anatomy and superimposition of these bony structures of the tarsus. Radiographic examination evaluates bone pathology by the detection of subtle differences in bone density, trabecular detail, and cortical thickness. However, a decrease in bone density of between 30% and 50% has to occur before a change is visible radiographically.

Many of the bony conditions that affect the tarsus manifest abnormal radiographic findings that are located at the dorsum of the tarsus. Pathologic abnormalities of the tarsus tend to be in specific anatomical areas, and soft tissue swelling is helpful to localize abnormalities.[2] Radiographic examination of the tarsal region is often performed to evaluate the distal tarsus joints for OA (TMT/DIT) or to diagnose the presence of OC in the TC joint. Occasionally special projections are needed to elaborate an area not clearly defined with the standard radiographic examination such as the flexed lateral or skyline of the tuber calcis (TC) (Figure 5.62). Other common abnormalities of the tarsus visible on

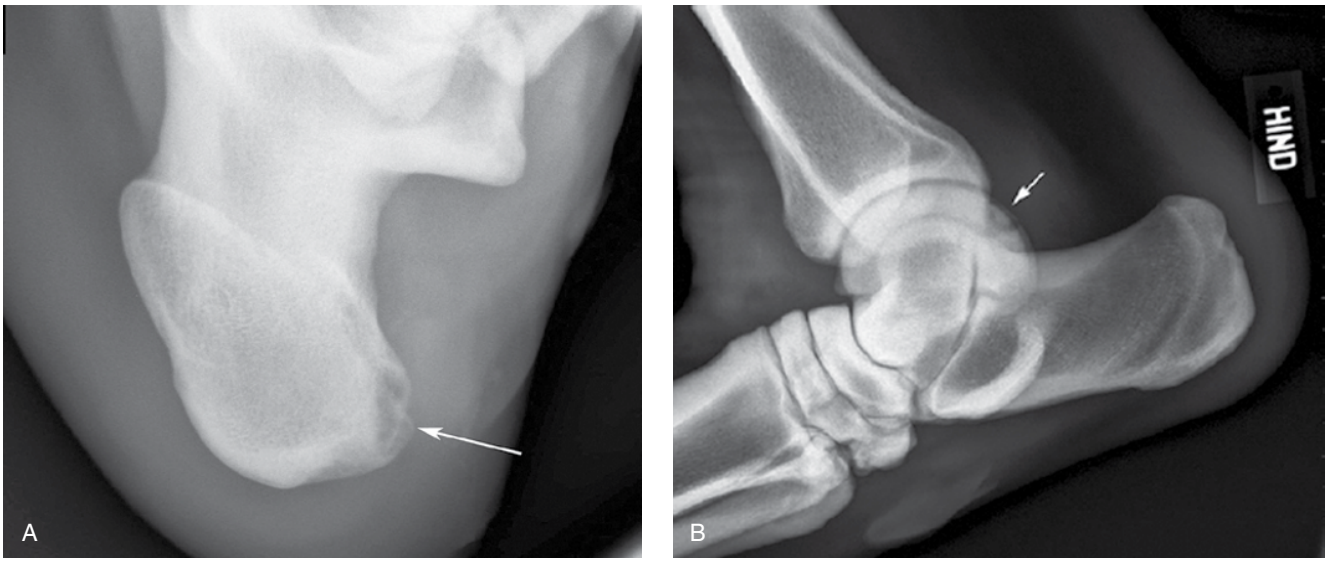


Figure 5.62. Skyline (A) radiograph that was necessary to document osteitis of the calcaneus (arrow) and flexed lateral radiograph (B) that revealed a large fracture fragment on the plantar aspect of the medial trochlear ridge of the talus (arrow) that was not visible with other views.

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radiographic exam include soft tissue swelling without bony change, fractures, and developmental disorders. The most common clinical condition requiring radiographic examination of tarsus is bone spavin. This arthritic condition of the DT joints manifests a variety of radiographic changes including osteophytes at the joint margins of the DT joints, loss of joint space, and the presence of sclerotic tarsal bones, particularly the central tarsal bone. However, joint space narrowing associated with the distal joints caused by bone spavin can be difficult to detect and are best demonstrated with radiographic projections that center the beam on the lower tarsal joints.

Another common clinical condition requiring radiographic examination of the tarsus is OC of the TC joint. The most common sites for OC in decreasing frequency of occurrence are the distal intermediate ridge of the tibia (DIRT), the lateral trochlear ridge (LTR), the medial malleolus (MM), the medial trochlear ridge (MTR), and the lateral malleolus (LM). OC of the TC joint most commonly manifests as osteochondral fragmentation, but subchondral lucencies and irregularity can also occur.

Ultrasonography of the Tarsus

Many of the structures of interest in the tarsus are superficial and lie just under the skin and subcutaneous tissue, and therefore US provides an excellent means imaging these soft tissue structures (Figure 5.63). A high-frequency (10–18 MHz) linear probe with variable focal zones is ideal and can be enhanced when a standoff works best. A scanning depth of 4–6 cm or less is appropriate for most structures. The sonographic examination of the tarsus should be methodically examined from the dorsal (extensor surface), medial, lateral, and plantar (flexor surface) aspects of the joint.[146] These areas should initially be examined with the animal weight-bearing, and because diagnostic US has the added benefit of providing real-time information, these structures should also be examined throughout their range of motion. When evaluating the dorsal and plantar aspect of the tarsus, it is important to utilize a normal range of motion with flexion and extension in an attempt to evaluate the weight-bearing and non-weight-bearing surfaces of the articular cartilage of the joint. This technique can also be utilized when evaluating the joint for OC and the position and mobility of osteochondral fragments predominantly in the dorsal and plantar aspect of the joint. Structures in close proximity that have similar sonographic density can be difficult to separate. Detailed descriptions of the US examination of the tarsus exist.[32,35,59,116,140,148]

Scintigraphic Examination of the Tarsus

Hindlimb lameness that cannot be conclusively diagnosed with radiographic and ultrasonographic examinations may be candidates for scintigraphy.[44,49,59,102,103,119,137] The bone scan technique can allow detection of subtle bone changes undetected by other diagnostic methods. DT joint pain is often associated with increased radiopharmaceutical uptake (IRU) in the DT joints compared with that seen in normal horses. Any acute onset of lameness thought due to a tarsal fracture but without radiographic evidence of a fracture is also a candidate for a bone scan. Increased radionuclide uptake does not always equate with clinical significance, emphasizing that interpretation of nuclear scintigraphic images without reference to the clinical examination and other imaging results can be potentially misleading (Figure 5.64).

Computed Tomography of the Tarsus

CT in the horse is indicated when other bone imaging technologies are inconclusive or when there is a fracture that needs more characterization. CT can provide information about a bony lesion not available with other imaging techniques.[109,115] Occasionally CT can be useful when other imaging techniques demonstrate tarsal lesions and there is a need for better interpretation (like

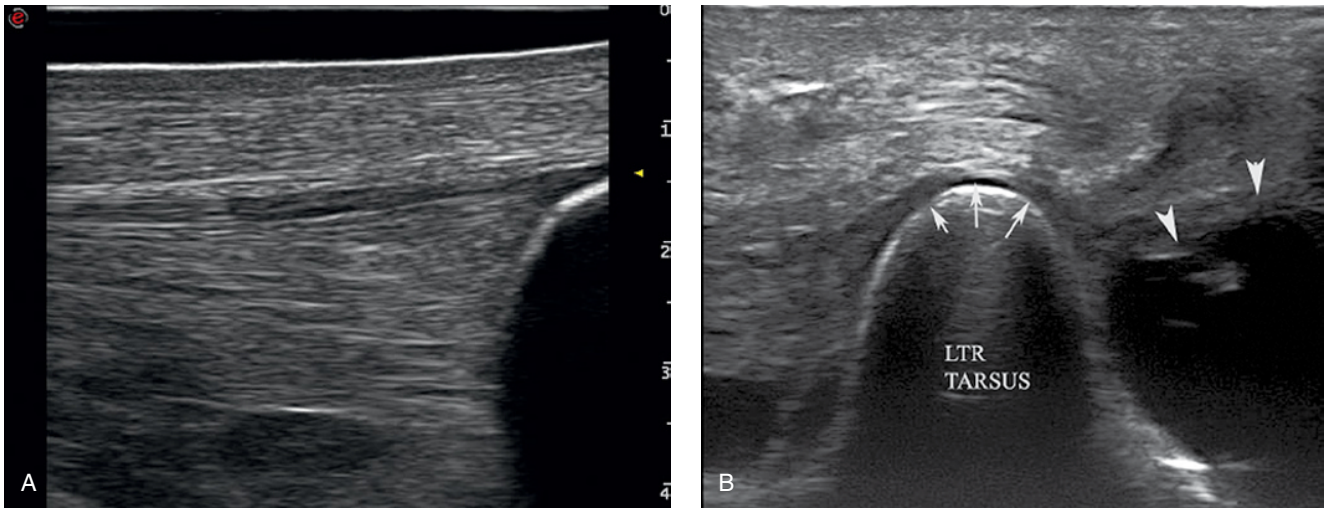


Figure 5.63. Ultrasonographic images of the normal gastrocnemius attachment to the tuber calcis (A) and the articular surface of the lateral trochlear ridge of the talus (B).

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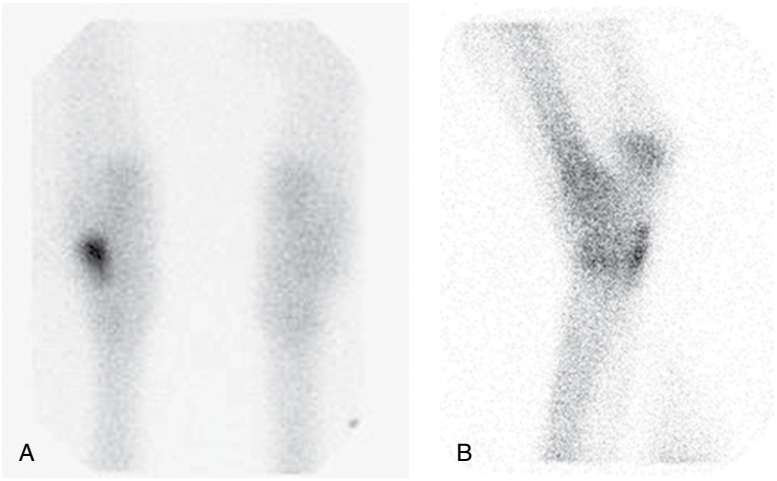


Figure 5.64. Nuclear scintigraphy can provide information about specific conditions that occur in the tarsal region that are associated with the bone modeling. Image on the left (A) is of horse with talocalcaneal OA, and the image on the right (B) corresponds to an area of calcification of the plantar ligament.



Figure 5.65. Cross-sectional imaging techniques such as CT eliminates the superimposition of the different bones that make up the tarsus. This CT demonstrates a comminuted fracture of the central tarsal bone and can be used to more accurately place screws to compress the fractures.

scintigraphy) (Figure 5.65). Subchondral cyst-like lesions, osteolytic lesions of any of the tarsal bones, degenerative changes of the DT joints, sclerosis of any of the tarsal bones (but particularly T3), and reaction around the interosseous ligaments can all be identified very clearly with CT. CT is especially helpful to determine the extent and complexity of a fracture better than radiography. Focal lesions located between the tarsal bone surfaces that are difficult or even impossible to detect with radiography and ultrasonography can be better characterized with CT.

MRI of the Tarsus

MRI can be useful for evaluation of DT pain because there is a lack of correlation between the presence and severity of radiographic changes compared with lameness in many horses (Figure 5.66). Protocols for MRI of the tarsus have been suggested, and equine tarsal MRI anatomy has been described using both low- and high-field systems.[13,14,21,45,83,125] In the normal tarsus, articular cartilage of the DT joints is very thin, which does not allow for distinction of proximal and distal cartilage layers in these joint spaces. Subchondral bone plates of the DT bones and the MTIII have homogeneous low signal intensity with a regular osteochondral junction and a smooth but undulating deep border. In competition horses that undergo high intensity training, subchondral bone thickness is greater medially in the DIT joint and laterally in the TMT joint.[21,125] This repeatable thickness pattern of subchondral bone is lost in horses with DT lameness. For instance, cadaveric tarsi from horses with histories of DT

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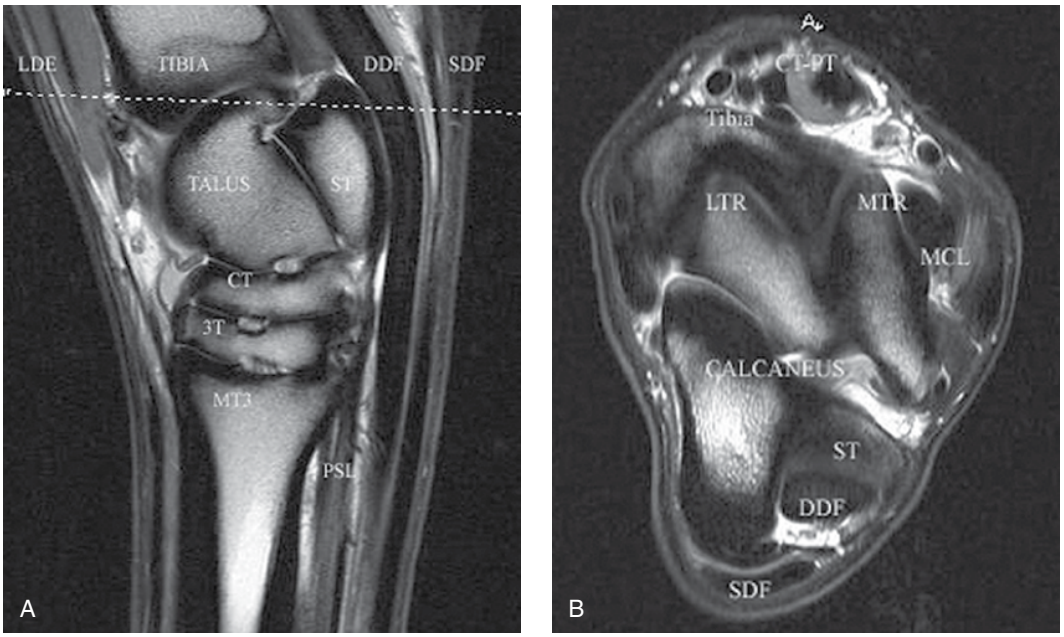


Figure 5.66. Sagittal PD (A) and transverse PD (B) MR images of the tarsus demonstrating normal soft tissue and bone anatomy. MRI is an extremely useful tool for detection of many types of pathology within the tarsus.

pain revealed variations in subchondral thickness compared with horses without histories of DT pain.

There are few reports on the incidence of MRI diagnoses in the tarsus of live horses. In one cadaver study, MRI was more sensitive and specific than radiography for detection of all types of pathology of the DT joints, including intertarsal ligament pathology, cartilage erosion, osseous cyst-like lesions, and subchondral bone irregularity. In live horses with lameness associated with the tarsal region, the presence of abnormal STIR signal hyperintensity compatible with the presence of bone bruising was reported in the central and third tarsal bones, talus, and tibia. MRI of the tarsus has also been helpful to diagnose abnormalities that are not identified with other imaging modalities. These abnormalities have included subchondral bone injury of the talocalcaneal joint, bone bruising of the distal tibia, intertarsal ligament enthesopathy with associated bone edema, focal OA in the plantar aspect of the DIT and talocalcaneal joints with localized loss of joint space and subchondral bone sclerosis, desmitis of the CLs of the TC and the PIT joints, and tendinitis of the deep digital flexor tendon (DDFT) at the level of the calcaneus.

ARTICULAR DISEASES OF THE TARSUS

Osteoarthritis (OA) of the Distal Tarsal (DT) Joints/DT Joint Pain

DT OA has been recognized as a cause of hindlimb lameness for centuries. It is frequently called bone or jack spavin or occult or blind spavin in the absence of radiographic abnormalities. Chronic cases often develop an enlargement over the medial or dorsomedial aspects of the DT joints. In addition, the term juvenile spavin has been used to describe the development of DT OA in horses less than 3 years old typically before the animal has done much work.[65] This is likely due to developmental lesions of the DT bones (i.e. OC) such as cuboidal bone hypoplasia that is seen in premature and dysmature foals that causes collapse of the central and third tarsal bones.[2,3]

Performance horses in a variety of disciplines and of all ages can develop lameness associated with the DT joints. The DIT and TMT joints are considered the joints of the hindlimbs most likely to develop OA from work-related stress (wear and tear). There are certain types of activities that may contribute to uneven or excessive loading of the lower hock joints. These include sports that require a great deal of hock flexion (dressage), stress (jumping), sudden stops or turns (western events, such as reining), or a great deal of concussion (Standardbred [STB] racing). In addition, OA of the DT joints can occur relatively frequently in mature horses that are ridden at a gallop and canter. While bone spavin is seen frequently in mature sport horses used for a variety of disciplines, it is also found as a source of lameness in young Thoroughbred (TB) and STB racehorses. Bone spavin often occurs in normally conformed horses. There are however some conformational abnormalities such as sickle hock, cow hock, or excessive straightness of the hindlimbs that may predispose to DT joint pain. These types of conformations are felt to produce greater stress in the medial aspect of the tarsal joint.

Etiology

OA of the DT joint (bone spavin) typically involves the TMT and DIT joints but occasionally may extend into the PIT and TC joint. DT OA is common in a wide variety of disciplines probably because these events cause increased torsional and shear forces on the DT joints. Many feel that excessive compression and rotation of the DT joints during training and competing create abnormal tension on the intertarsal ligaments and

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potentially excessive tension on the attachment of the major dorsal ligaments. It has long been thought that DT joint pain begins on the dorsomedial aspect of affected joints, which then progress dorsally. However, Dyson has reported that DT joint OA initially manifests radiographic and scintigraphic abnormalities on the dorsolateral aspect of the joints.[40,43]

OA of the DT joints commonly affects athletic horses during the most productive years of their lives and can limit career length.[7,17,24,43,56,57,99,100,138] The pathogenesis of spavin can be divided into three phases: (1) fibrillation of cartilage, (2) osteolysis, and (3) ankylosis. The first stage can be observed in young horses and can predominantly be found at two predilection sites on the articular cartilage of the DIT joint. The lesions can then progress further in the direction of the subchondral bone plate where osteolytic lesions may develop. In this phase, horses commonly start to show clinical signs of lameness. In some cases, the lesions may proceed further until a complete ankylosis occurs. The degree of osteolysis may significantly influence whether diagnostic anesthesia will be effective at eliminating the lameness or provide only partial improvement. Dyson[43] classifies horses with DT pain into three categories: (1) horses that respond to IA analgesia of either the DIT or TMT joints or both, but with no detectable radiological abnormality; (2) horses with predominantly periarticular osteophyte formation; and (3) horses with joint space narrowing, subchondral bone lysis, and sclerosis. However, these three categories are not necessarily considered continuums of OA of the distal tarsus. OA also has been associated with increased intramedullary pressure particularly within the cuboidal bones of the tarsal region.

In some breeds such as the Icelandic horse, there appears to be a genetic component to development of OA in the DT joints. While heritability can be difficult to prove, there has been a very obvious decrease in prevalence of bone spavin in the Royal Dutch Warmblood Studbook (KWPN) due to a strong and consistent selection process.[7] Selection was based on two main criteria: conformation and a reduction in radiographic signs of bone spavin. It has also been shown in the Dutch Warmblood that there is a strong relationship between a faulty conformation in the hock region (sickle hocks but also extremely extended hocks and abnormal outward rotation during the stance phase) and the occurrence of bone spavin. Animated gaits (such as seen with American Saddlebred and Tennessee Walking Horse) can also contribute to the development of bone spavin in these horses. For example, the rack is a fast four-beat gait during which the horse places their hindlimbs extremely far forward.

Diagnosis

The diagnosis of distal limb/tarsal disorders of the hindlimb can be challenging.[61] Frequently clinical signs are not obvious and not specific to the tarsus. Structures in the distal limb and tarsus should be palpated while standing on the limb and with the limb raised. Manipulation of the limb by flexion, extension, and rotation may provide some indication of joint(s) involvement. A moving examination should be performed at a walk and trot in a straight line and circle as well as a canter in a circle. If possible, watching the horse being ridden can be very useful and may be most effective at demonstrating the lameness that the owner/trainer has described. When the opportunity exists, the horse can be examined with a rider up and comparisons made about the gait before and after intra-articular diagnostic analgesia is performed.

Physical examination can be confusing and reveal pain and sensitivity through the back and pelvic regions. These clinical findings may give the impression that lameness originates from the topline or more proximal on the limb. Some horses with DT joint pain manifest a lower arc of the foot flight and toe dragging with wear patterns developing on its dorsal edge of the toe. In addition, some may manifest a change in foot conformation due to an alteration in how the horse loads the foot. In acute cases of moderate lameness, the application of hoof testers should be performed to rule out the presence of foot pain before evaluating the rest of the limb. Some lamenesses may involve multiple structures, so it is critical to develop a routine examination protocol that evaluates the entire horse while concentrating on the affected limb.

Joint distension is not a typical sign of OA of the DT joints, because the tarsal retinaculum over these small joints prevents its detection. However, changes in the silhouettes of the hock may develop medially. This can be due to soft tissue change such as with cunean bursitis and/or effusion of the cunean bursa. However, this change in shape is more often due to a firm bony swelling associated with more advanced stages of DT OA. Palpation of the horse may also demonstrate soreness of the epaxial muscles in the lumbar region and sometimes the caudal gluteal muscles. A compensatory forelimb lameness may also develop due to a reluctance of the horse to bear appropriate weight on its hindlimbs.

Physical examination may not reveal any obvious abnormalities. Palpating the tarsal region may reveal irregularity over the dorsomedial aspect of the tarsus, but often this is not sensitive to digital pressure. Some clinicians may perform a manipulation called Churchill's test, which is accomplished by directing digital pressure over the head of the medial splint as a reflection of DT pain. The test is considered positive when the horse abducts the limb away from the pressure. The lameness examination should begin with a flexion test of the least affected hindlimb first, because the more severely affected limb may remain lame after flexion, which can complicate the rest of the lameness exam. The disease is often bilateral, and flexing the unaffected limb may be quite positive or it may cause the hindlimb gait to even out because the pain is distributed more evenly behind. Proximal and distal limb flexion test should be performed separately. The proximal limb flexion test or spavin test is useful at accentuating lameness with tarsal joint pain in most affected horses. However, a positive response is not specific for the tarsal lameness because the reciprocal apparatus creates flexion of tarsus, stifle, and coxofemoral joints (to varying degrees) simultaneously. A stifle flexion test may be utilized to help differentiate lameness originating from the tarsus vs. the stifle. Many horses with proximal limb involvement may respond positively to both distal and proximal limb flexion.

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DT joint OA often occurs bilaterally, but occasionally it may occur unilaterally. There is often a history of gradual onset of lameness. Occasional horses with bilateral DT joint pain may not manifest overt lameness but may be thought to have a behavioral issue or a lack of performance. When lameness occurs, it may become worse when the horse is worked hard for several days and may improve with rest. Lameness tends to be worse when the horse is first started into work. Many mildly affected horses tend to warm out of the lameness after a short time. The horse may feel stiff or jerky when circled to the affected side, and it may refuse or be reluctant to go in the opposite direction or take the opposite lead. However, some horses show more lameness with the lame or lamer limb on the outside. Western performance horses (i.e. reining, roping, and cutting) or gaming horses that are required to stop aggressively can become reluctant to decelerate or perform sliding stops required for their discipline. An animal may develop a problem with the performance of discipline-specific maneuvers such as refusing to jump or to jump poorly with an increase in rails knocked down. The horse may have trouble picking up or holding a lead or may show a reluctance to turn in one direction. The development of these types of performance or behavioral issues can be consistent with the development of DT joint pain.

The way in which horses with DT joint pain manifest lameness can vary considerably. Some clinicians have reported a tendency for horses with DT joint pain to manifest a characteristic limb carriage and foot placement. This gait can be described as an axial deviation of limb during the swing phase of the stride with a rapid abaxial deviation of the limb just before contact with the ground (so-called J-step). While this limb carriage is common in horses with DT pain, it is not pathognomonic. In addition the horse with DT joint pain can have a reduced degree of flexion of the hock during the swing phase of the stride. This translates into a reduction in the height of the foot flight arc and a shortening of the cranial phase of the stride often with an associated dragging of the toe. Lameness associated with DT pain may include asymmetrical movement of the tubera coxae, shortening of the cranial phase of the stride, and sometimes a reduced extension of the fetlock during stance phase. In addition the affected limb usually lands toe first, resulting in excessive wearing of the toe. Kinematic gait measurements recorded after endotoxin-induced lameness of the DT joints were considerably different than what occurs clinically in horses with bone spavin.[79] This study reported that fetlock and tarsal joint extension decreased during stance phase, fetlock joint flexion and hoof height during swing phase increased, and vertical excursion of the tuber coxae became more asymmetrical.[79]

Diagnostic Analgesia

Flexion tests of the hindlimb have low specificity (multiple joints are flexed at the same time), which emphasizes the need to utilize diagnostic analgesia to confirm the source of lameness. Accurate placement of the intra-articular anesthesia of the TMT and/or DIT joints should be performed in many horses to confirm that the origin of the lameness is the tarsus. However, the accuracy of diagnostic analgesia may be questioned based on the potential for leakage of local anesthetic around the proximal plantar metatarsus and extension around the insertions of the cranial tibialis (CT) and peroneus tertius (PT) tendons following injection of the TMT joint. The injection of contrast media into the DT joints of the tarsus demonstrated that once the media filled the joint space, there was extension of fluid surrounding the proximal plantar metatarsus and origin of the SL in 70% of the limbs.[42] One study found that forced injection into the TMT joint caused rupture and extra-articular leakage of contrast around the head of the lateral splint.[80] Leakage was found to occur close to the area of the lateral plantar nerve and DBLPN, potentially leading to false-positive responses. In addition, the variability of communication between the TMT and DIT joints[11,42,80,121] suggests that these joints should be anesthetized separately.

The TMT joint is the easiest joint to inject and is approached plantaroproximal to the base (head) of the fourth metatarsal bone. The site for injection of the DIT joint is from the dorsomedial aspect of the limb distal to the palpable distal border of the medial branch of the tendon of the tibialis cranialis muscle (cunean tendon) and between the central, third, and combined first and second tarsal bones. In the normal horse this may be difficult to accurately palpate, making is difficult to comfortably inject this joint. Many clinicians prefer to inject the TMT joint first with 2–3 mL of mepivacaine until resistance is felt. Any more pressure increases the risk of anesthetic solution escaping the joint capsule. When communication between these two joints exists, needle placement into the DIT may allow for observing of local anesthetic dripping out of the needle. The clinician should interpret this carefully as the needle may easily enter the TMT joint if it is placed too far distally in the space between the third and second tarsal bones. When communication of these joints is found to exist, treating only the TMT joints may prove to be effective at adequately managing DT (both DIT and TMT) joint pain. That may not be the case once the pathological process progresses and begins to occlude or obstruct this communication. Interpreting the results of intra-articular analgesia of the DT joints may prove to be difficult. Intra-articular analgesia may only improve the lameness score by 50% due to moderate to advanced osteoarthritic and coexistent subchondral bone pain. Improvement is generally seen within 20 minutes, but occasionally the response is delayed. Some clinicians recommend delaying further blocks until at least 1 hour has elapsed.

Moderate to severe OA with proliferative periarticular bone can make placing a needle into a narrowed joint space difficult if not impossible. In these cases radiographic and US-guided control may be required to assist needle placement into the joint space. When intra-articular analgesia is too difficult or ineffective at eliminating lameness of the DT joints, perineural analgesia of the superficial and deep peroneal (fibular) and the tibial nerves should be utilized. Blocking of the caudal tibial and deep peroneal nerves is a reasonably accurate diagnostic method. However, other structures in addition to the DT joints are blocked, and the effect of this block may not be complete. Further, confirmation of complete

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analgesia is difficult, and desensitization of such a large portion of the limb occasionally alters the horse's movement. Perineural analgesia can be problematic and may take 30 minutes to an hour to become effective. The tibial nerve is quite large, and it may take a prolonged period of time for the analgesia to diffuse into and affect the nerve. This block is not specific for the DT joints and necessitates a methodical approach and sequential desensitization of the distal limb, proximal suspensory area (proximal plantar region), and TC joint. Once these have been performed without effectively eliminating the lameness, then a peroneal and tibial nerve block can be performed.

Perineural analgesia techniques around the tarsus can also provide inaccurate results. For instance, analgesia of the DBLPN can influence DT pain, and according to Dyson,[38] intra-articular anesthesia of the TMT joint can abolish lameness in horses with PSD (8% incidence).[38] Therefore a comparison between the respective outcomes of intra-articular and perineural anesthesia is useful. A tibial nerve block can be useful to distinguish between DT joint pain and PSD pain, as anesthesia of the tibial nerve will result in analgesia of the PSD without significantly influencing DT joint pain.

Imaging

Radiography

Radiography can be quite helpful in the diagnosis of OA of the DT joints. Radiographic examination to evaluate the DT joints should have the primary radiographic beam centered on the DT joint spaces and focused tangential to the joint spaces to best interpret changes that occur in these joints. Radiographic signs of bone spavin include (1) periarticular osteophytes ("spurs"), (2) irregularity and lucency at the intertarsal joint margin, (3) irregular width of the joint space and cysts in the subchondral bone, (4) narrowing or loss of joint space, (5) subchondral bone lysis, (6) new bone production of the distodorsal tarsus, (7) bulging of the dorsomedial contours of the central and third tarsal bones, and (8) ankylosis (Figure 5.67). The degree of lameness is not necessarily correlated with the degree or type of osteoarthritic change detected radiographically. Early radiographic changes of OA of the DT joints include periarticular osteophyte formation, subchondral lucent areas, subchondral sclerosis, periosteal new bone, and narrowing of joint spaces. Certain radiographic changes such as periarticular osteophyte formation or joint narrowing may be highlighted in the DLPMO and dorsoplantar projections.

Early cases of bone spavin have radiographic signs that occur on the dorsomedial aspect of the tarsus that progress dorsally. Some clinicians indicate the earliest radiographic signs occur on the dorsolateral aspect of the tarsus. However, not all horses with DT pain have radiographic evidence of OA. Scintigraphic examination can be a helpful diagnostic tool in these cases and may reveal subtle changes within the tarsus not detectable by radiographs. In contrast, some horses with radiographic evidence of OA are not lame. The correlation between the degree of lameness and the extent of radiological abnormalities is poor. However, even subtle radiographic lesions can support the diagnosis in the presence of appropriate clinical findings. There appears to be little association between the duration and degree of lameness, the response to intra-articular anesthesia, and radiographic findings in horses with OA of the DT joint. Some lameness clinicians believe that horses with cystic changes and subchondral bone lucencies on radiographs develop more severe lameness. In contrast, some horses with few radiographic signs are problematic in that degeneration never progresses, and they remain lame. Some horses predominantly develop periarticular changes, whereas others have abnormalities confined to the central and third tarsal bones and proximal MT3.

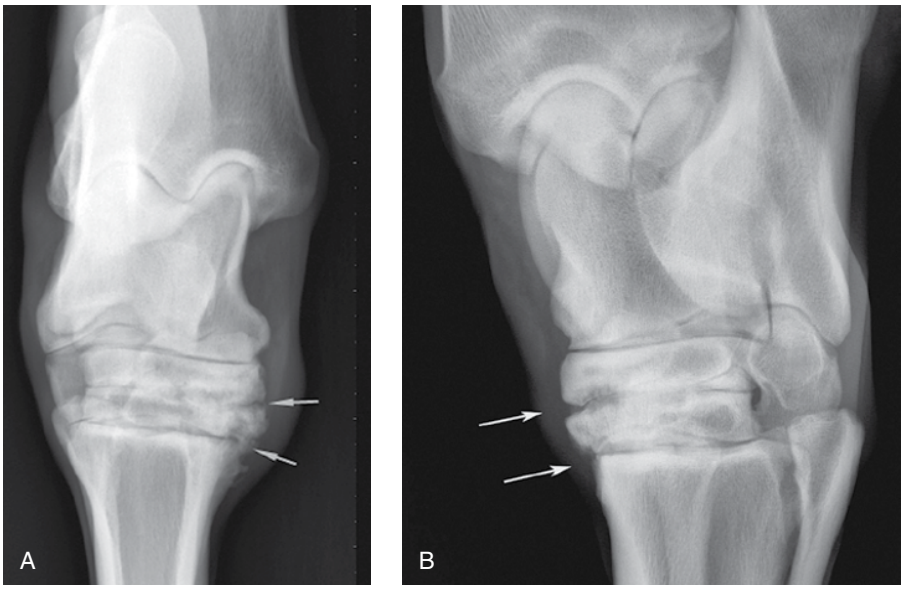


Figure 5.67. Radiographic changes of OA of the distal tarsal joints can vary considerably including periarticular osteophyte formation, subchondral lucent areas, subchondral sclerosis, periosteal new bone (A), and narrowing of joint spaces. The radiograph on the right is a horse with juvenile spavin with moderate degenerative changes of the DIT and TMT joints (B).

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Nuclear Scintigraphy

There is a repeatable pattern of RU in the DT region of normal horses, and an alteration in this pattern could provide an indication of tarsal pathology, related to altered patterns of bone remodeling. Nuclear scintigraphy is often unnecessary, but it can be helpful in difficult horses and those horses with performance problems rather than overt lameness. IRU may be focal or more diffuse, and this can vary between horses. Focal increased uptake may reflect intertarsal ligament enthesopathy rather than OA. Intense IRU may be present in the absence of radiological abnormalities, and these horses tend to respond poorly to intra-articular medication, despite a positive response to intra-articular analgesia. Horses with diffuse radiopharmaceutical uptake tended to respond to treatment better than those with focal uptake.[103] Diffuse uptake possibly indicates more generalized inflammation from use as compared to focal articular degeneration demonstrated by focal peaks of uptake. The distribution of inflammation may bear prognostic value as well. While nuclear scintigraphy can be useful adjunct to confirm DT joint modeling activity, it may also help to document other sites of involvement (Figure 5.68).

MRI

The primary indication for MRI is indicated when pain has been localized to the tarsal region using perineural analgesia or by intrasynovial analgesia, but there are no radiological or ultrasonographic abnormalities sufficient to explain the degree of lameness. However, an effusive TC joint may be a stronger indication for exploratory arthroscopy than for an MRI as the ability to surgically address the problem (if found) can be accomplished during the same anesthetic procedure. However, the most common use of MRI of the DT joints is to further evaluate horses that have pain originating from the proximal plantar region suggestive of PSD.

MRI examination of the tarsus requires that the horse be under general anesthesia. Acquiring diagnostic images of the tarsus in the horse in a low field strength magnet (usually found in standing systems) may prove to be difficult because of slower acquisition times and the inherent motion that occurs proximally on the limb. The contralateral limb would ideally be examined for comparison. See the imaging chapter on magnetic resonance imaging (MRI) for further details.

Using MRI, horses with DT pain have been found to have intertarsal ligament damage, focal cartilage damage, and localized bone pathology including cystic lesions and microfractures. It is therefore possible that DT pain incorporates a variety of different conditions, culminating in end-stage OA.

Differentiating DT Joint Pain from Proximal Plantar Region Pain

PSD of the hindlimb has become a frequently diagnosed cause of acute and chronic hindlimb lameness in mature horses that perform dressage, eventing, and general-purpose work.[38,39,82] The recent increase in the diagnosis of PSD may be due to improvement by veterinarians and equine professionals in recognizing this condition. However, lameness referable to this region is often initially assumed to originate from the DT joints. Subsequent intra-articular injection of corticosteroids into the DT joints may provide a local anti-inflammatory effect, which can minimize the pain associated with the active desmitis. This may allow the resumption of work before appropriate repair has occurred, leading to further damage to the SL. An accurate early diagnosis of PSD is critical to minimize further damage to the SL and justify aggressive and expensive treatment to salvage the horse's career. In one study of 40 horses with lameness

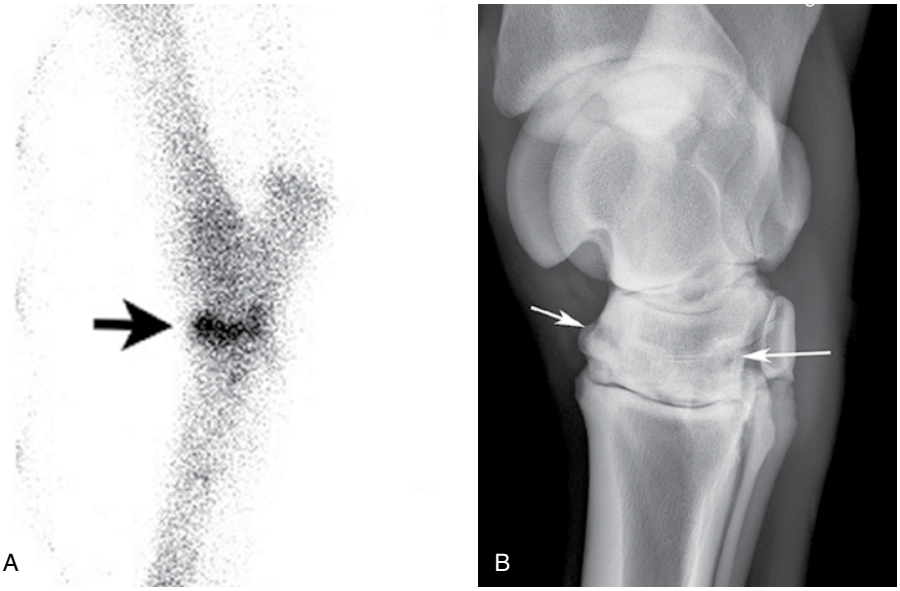


Figure 5.68. Lateral scintigram (A) and radiograph (B) of the tarsus of a horse with distal tarsal OA. There is diffuse uptake within the distal tarsal joints and joint space narrowing and lysis within the DIT joint (arrows) visible on the radiograph.

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that was eliminated by proximal plantar analgesia that underwent MRI of the region, some of the horses had OA of the DT joints, subchondral cystic lesions (SCLs) of the CT and 3T bones, intertarsal ligament pathology, and bone edema of the third and fourth tarsal bones without evidence of proximal suspensory disease.[82] Many of these horses had diagnostic analgesia of the TMT and DIT performed without significant response. The findings in this study continue to fuel questions about causes of tarsal pain and the accuracy of analgesia of the proximal metatarsal region.

Treatment of DT OA

OA of the DT joints commonly affects athletic horses during the most productive years of their lives and can limit their career potential. The goal of treatment is to provide significant, long-lasting pain relief that will permit the horse to remain comfortably in work. Horses with minimal radiographic changes of the DT joints may respond favorably to a short period of reduced activity, corrective shoeing, systemic medications (like nonsteroidal anti-inflammatory drugs [NSAIDs]), and a change in the work program. Intra-articular treatment options have included the use of corticosteroids, hyaluronan, or polysulfated glycosaminoglycans (PSGAGs).

A common strategy in the management of horses displaying mild bilateral hindlimb lameness (particularly those breeds and disciplines that are predisposed to DT lameness) is to medicate the DT joints with corticosteroids and evaluate for a response. A short-term response (less than 2–3 months) should be a concern that the primary source of pain was not effectively addressed and should be an indication to more carefully evaluate the tarsal region for other sources of lameness. The deposition of steroids into the TMT joint can have a local effect on the soft tissue structures in proximal plantar region and may suppress inflammatory changes in the proximal SL. Corticosteroids can also suppress healing by inhibiting the production of collagen, which may lead to a perpetuation/exacerbation of any injury of the proximal SL in this area. A short duration of effect for intrarticular (IA) corticosteroids could also be indicative of more advanced changes within the DT joints. Further clinical and imaging examination (potentially including MRI) of the tarsal region should be recommended.

Most clinicians recommend some form of intra-articular medication (see below) to help subdue the inflammatory changes within the DT joints. The response to therapy can often be predicted based on the extent of radiographic signs and to a certain degree on the duration of clinical signs. One study demonstrated a positive outcome following intra-articular medication in only 38% of cases with OA of the DT joints.[81] However, in that study only the TMT joint was treated in the majority of the horses, which may explain their low success rate. Another study reported that 52% of horses were able to return to their previous level of exercise, with an improvement in 70% of horses that had both the TMT and DIT joints injected.[24]

Historically it had been suggested that injection of corticosteroids (with or without hyaluronic acid [HA]) into the DT joints while maintaining the horse in work would eventually lead to ankylosis of these joints. It was felt that the presence of the corticosteroids within the joint would have a significant detrimental effect on chondrocyte metabolism and the continued work on compromised cartilage would lead to the advancement of the cartilage damage in these joints. However, clinically this does not seem to be effective, and there has been little evidence that ankyloses can be promoted by consistent intra-articular injection of corticosteroids. The current approach to managing bone spavin especially in a younger horse is to preserve the articular cartilage whenever possible.

Many factors may play a part in determining the specific treatment and management of horses with bone spavin.[64] These may include the degree of lameness, extent of radiological abnormalities, use of the horse, competition regulations, response to previous treatment, and financial constraints. Clinically horses with extensive radiological abnormalities or with scintigraphic abnormalities in the absence of radiological change seem to have a more limited response to intra-articular injections. Resting horses with bone spavin is usually not beneficial. Anti-inflammatory medications are indicated to reduce inflammation and pain in affected horses. Phenylbutazone (2.2 mg/kg, BID) can be continued for an extended period of time in most horses or given only when the horse is worked. Treatment with NSAIDs (particularly phenylbutazone) can be quite effective in some horses, and attempts should be made to find the lowest dose possible that will alleviate lameness. Long-term use of oral phenylbutazone at 1gram twice daily for a 500-kg horse is generally well tolerated. Any treatment should be combined with corrective trimming and shoeing. The inclusion of systemic treatment such as PSGAGs, pentosan, oral nutraceuticals, or any combination may also be chosen in certain cases.

Intra-articular Medications

Intra-articular medications such as corticosteroids, hyaluronan, and more recently IRAP can be utilized in joint inflammation.[43,53,81,90,130] Corticosteroids are potent anti-inflammatory agents and inhibit the inflammatory process at all levels and have been the mainstay of treatment for many years. Many horses with DT joint pain have been successfully managed throughout their athletic career with consistent (biannual to annual) intra-articular injections of corticosteroids. There are quite varying opinions about which drugs are most effective in the DT joints and for how long the clinical effects from these different drugs might last. It is not universally accepted which drug is most efficacious, what dose of any drug should be given, how frequently these injections need to occur, or if intra-articular antibiotics should be given in conjunction with the drug. The corticosteroids that are most frequently used include methylprednisolone acetate (MPA), betamethasone, and triamcinolone (TA) either alone or in combination with some form of hyaluronan products. Rest has been recommended after injection, but there is little evidence to support any prolongation of effect by doing so. In humans rest postinjection has been advocated after intra-articular medications in the belief that restriction of activity might reduce clearance of the medication and enable better penetration of intra-articular tissues.

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Depo-Medrol at a concentration of 40 mg/mL is most frequently used at 80–100 mg/joint. Use of Depo-Medrol at the 80 mg/mL concentration should be discouraged as this product has a high incidence of flare reactions. Triamcinolone can be used at 4–9 mg/joint depending on how many joints are to be injected. Most clinicians do not exceed a total body dose of 18–20 mg because of reported increased risk for laminitis, although this dose limitation seems to be a source of debate. The frequency of injection should not be more often than every 3–4 months and in most instances twice a year is sufficient. It is thought that corticosteroid injections can be repeated two or three times per year without promoting progressive radiological change. There are no controlled clinical trials assessing the relative efficacy of different corticosteroids or the use of the combination of corticosteroids and hyaluronan, although some clinicians believe that combination therapy provides a longer clinical effect. Hyaluronan alone is of questionable value.

If the PIT joint is involved, the control of synovitis is indicated to prevent, or at least retard, development of OA in the TC joint. Frequent injections of the TC (and therefore the PIT joint) should be discouraged as this is a high range motion joint and can be at risk of developing progressive OA. Some clinicians suggest that hyaluronan prolongs the anti-inflammatory effects of the corticosteroid and can counteract the negative effects of MPA; however there is no evidence to support this. There is even conflicting evidence about whether HA combination therapy with TA or betamethasone esters does in fact prolong the chondroprotective effects of the corticosteroids. Lastly, disagreement also exists with respect to whether HA should be used at all because of the limited fluid volume that can be injected into the DT joints. Chronic administration of intra-articular corticosteroid should prompt regular radiographic evaluation of cuboidal tarsal bone density to detect the degree of advancement of OA in these joints.

A recent study evaluated the concentration of MPA or its ester methylprednisolone (MP) in the DIT joint after injection of MPA into the TMT joint. Injection of 80 mg of MPA into the TMT joint allowed diffusion into the DIT joint.[130] It was thought that the MPA and/or MP may have diffused either through the tarsal canal and space between the fused first and second tarsal bones and the third tarsal bone or through the thin synovial membrane. Regardless of the mode of diffusion, molecules of MPA or MP were found to diffuse from the TMT joint into the DIT joint in a concentration that was believed to be high enough to suppress inflammation. The authors proposed that horses affected with OA of both the DIT and TMT joints that are treated by intra-articular injection of MPA can be treated by instilling MPA into the TMT joint alone. However, it is questionable whether this concentration is effective to manage DT pain. In addition, diffusion of MPA and MP needs to be evaluated in horses with clinical disease of these joints.

Autologous conditioned serum (ACS) is thought to act by blocking the receptor to the inflammatory cytokine interleukin-1 (IL-1).[53,96] Arthrex Biosystems independently patented the technique of IRAP enrichment of serum and subsequent injection of this enriched serum into joints as IRAP® and IRAPII®. The technique has been used in human orthopedics (Orthokine®) since 1998 for treatment of OA, rheumatoid arthritis, and spinal disorders. There have been no case-controlled or peer-reviewed publications published in the literature regarding the efficacy of IRAP in DT joint OA in the horse. When using IRAP/IRAPII for the DIT or TMT joint, the manufacturer recommends using 1–2 mL/joint every 14 days for three treatments. This treatment needs to be evaluated clinically and with controlled research.[53,96]

Alternatively, intra-articular injections of PSGAG (three injections given every 2 weeks) have also been used in competition and racehorses with DT joint pain.

Extracorporeal Shockwave Therapy (ESWT)

The use of extracorporeal shockwave therapy (ESWT) in horses with DT OA was first described in a series 74 predominately western performance horses (130 joints).[88] The horses had been treated medically and had either become refractory to IA treatment or the response had reached an unacceptably short duration. Horses were treated under general anesthesia and received 2000 pulses. Fifty-nine of 74 horses (80%) showed improvement in lameness of at least 1 grade 90 days after treatment, but only 13 (18%) were sound. No radiographic differences were detectable between pre- and 90-day posttreatment evaluations. The fact that ESWT stimulated no further ankylosis in the large series of horses with DT OA might cause one to more strongly consider pain relief as a significant component of the mode of action. An analgesic effect of ESWT was thought to be the cause of improvement seen in these horses.

Bisphosphonates

Bisphosphonates are thought to be the most potent inhibitors of bone resorption clinically available to human and veterinary medicine. Two bisphosphonates that are now commercially available for use in the horse are tiludronate (Tildren$_R$) and clodronate (Osphos$_R$). An intravenous form of tiludronate (Tildren$_R$) has been used in horses at 0.1 mg/kg bwt/day for 10 days and is currently licensed for the treatment of navicular disease and bone spavin. The main action of tiludronate is the inhibition of osteoclasts, which in turn inhibits bone resorption. Tiludronate slows down bone remodeling and is felt to restore a normal balance between bone resorption and bone formation. Tiludronate has also been shown to have anti-inflammatory properties by decreasing the amount of nitric oxide and cytokines released from activated macrophages, which promote the early inflammation response. A recent clinical study[62] showed that Tildren$_R$ was effective in treating bone spavin in horses particularly when utilized in association with a controlled exercise program. Horses with periarticular osteophytes and subchondral bone thickening were felt to respond better. Little work has been performed to evaluate clodronate (Osphos$_R$) in the treatment of bone spavin in horses.

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Whatever protocol that is selected to treat horses with DT OA, it should be combined with corrective

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trimming and physiological shoeing (Figure 5.69). Horses with tarsal pain or bone spavin may attempt to unload the dorsal (front) aspect of the small tarsal joints by redistributing their weight. The first indicator of a DT problem may be the pattern of shoe wear. When a horse that has been wearing the hind shoes level from heel to toe begins to wear the toe excessively, it may be an indication that the horse has altered its gait in an effort to minimize extension. A second indication of a hock problem may be dragging of the toe on the affected limb(s). However this has also been described in horses that have pathology involving the stifle. Excessive toe wear may be the result of the horse trying to minimize hock flexion and carrying the hoof in a low arc of flight. Low or underrun heels affecting the health of the foot, or as a cause of lameness, are often overlooked in the hindfeet and may contribute to lameness of the distal hock joints. Underrun or collapsed heels in the hindfeet may lead to subtle bilateral lameness, which is often attributable to hock, stifle, or back pain. Low heels or short shoes allow the ground surface of the hoof at the heels to move forward, changing the conformation of the distal limb, which alters the angulation of the hock. Damage to the heels of the hindfeet is easier to improve than in the forefeet, possibly due to the anatomy and the difference of the load encountered on the hindlimbs.

Hindlimb foot management is aimed toward improving foot conformation and to facilitate break-over by shortening the toe by squaring and/or rolling the toe of the shoe if necessary. Shoes to the hindfeet are fit the same as the front where a line is drawn across the widest part of the foot and the shoe is fitted so the line is placed in the middle of the shoe.[3] In the hindfeet, the branches of the shoe will extend beyond the end of the heels. The appropriate length of the hind shoe can be determined by dropping a vertical line from the bulb of the heel to the end of the shoe. Fitting the shoe in this manner will change the conformation of the distal limb and the angulation of the hock during impact and the stance phase of the stride. To help reduce weight, wide-webbed wedged (flat) aluminum shoes are used in some show horses, but one has to be cautious when applying a wedge to low heels as this may increase pressure on these structures. A lateral trailer or a lateral extension of the shoe tends to stop excessive twisting of the limb. However, these devices may be contraindicated in horses that have to stop and turn quickly, because the extension may dig into the footing and stop the distal limb abruptly, causing abnormal torque on the distal limb joints. Common shoeing recommendations for horses with DT OA include lateral (outside) extensions or trailers, heel elevations, and rolled or squared off toes.[107]



Figure 5.69. When shoeing the hindfeet in horses with DT OA, it is important to extend the branches of the shoe beyond the end of the heels. *Source*: Courtesy of Dr. Steve O'Grady.

Advanced Treatment Procedures

Surgical Arthrodesis of DIT and TMT Joints

When bone spavin becomes refractory to systemic treatment and intra-articular medications, more aggressive therapy may be warranted. The natural resolution of advanced OA (bone spavin) is ankylosis. Ankylosis can stabilize the joint sufficiently enough to minimize the pain creating lameness (Figure 5.70). However, ankylosis is unpredictable and can take some time to progress to soundness. This rarely happens without surgical intervention, and therefore surgical arthrodesis techniques have been developed to help accelerate joint fusion.[1,4,6,33,48,91,129,150,151] The optimal treatment for arthrodesis of the DT joints should be minimally invasive, create widespread necrosis of the chondrocytes and destruction of the articular cartilage matrix, cause little to no discomfort for the horse during the treatment period, lack serious complications, and be affordable.

A variety of techniques have been developed for surgical arthrodesis of the DT joints. Currently, intra-articular drilling of the joint spaces is the most widely used means to promote surgical arthrodesis. Focal mechanical destruction of the articular cartilage and subchondral bone via "three-path" technique with removal of less than 60% of the cartilage is probably the most common arthrodesis technique (Figure 5.71). Variations in technique include size of drill bit, use of bone graft, and degree of radiographic or fluoroscopic control utilized during the procedure. The drill tracts eventually heal with a bridging callus, which crosses the joint space acting to stabilizing the joint in a manner similar to spot welding of metal. One study found that of 36 horses having the three-drill tract procedure performed, 66% were able to return to their previous levels of performance.[33] An additional 14% of horses improved after surgery, but did not meet the criteria to be considered successful. In that study, no clinical, radiographic, or surgical factors were beneficial in predicting clinical outcome; however, there was a trend toward improved outcome when precise radiographic monitoring of the drilling procedure was performed at surgery.[33] The major disadvantages of this technique include expense and prolonged convalescence, which ranges from 4 to 12 months. There is also a concern that results in upper-level competition horses are less favorable. Careful

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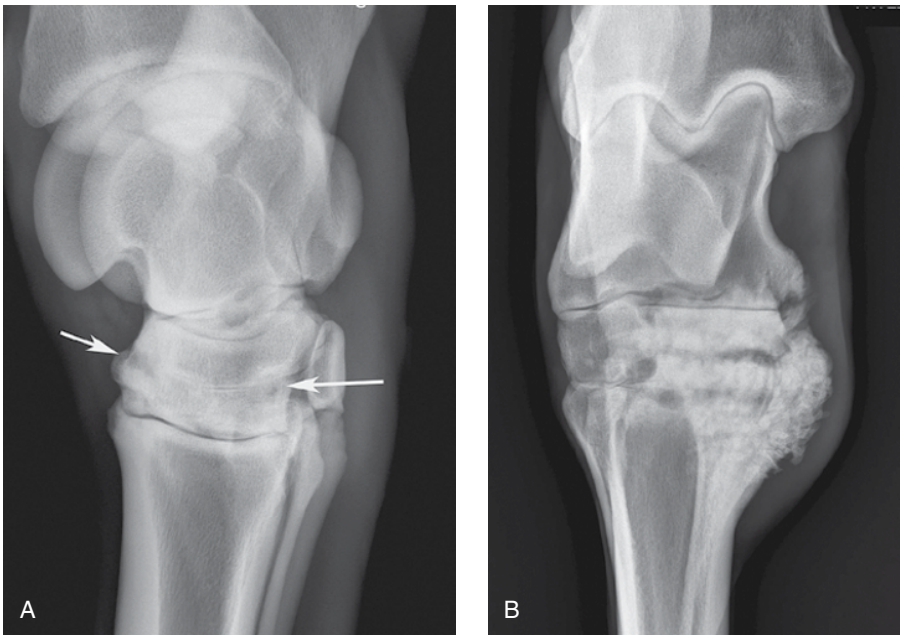


Figure 5.70. The outcome of horses that undergo natural ankylosis of the hocks is variable. The tarsus on the left (A) has nearly fused naturally and the horse is sound. The one on the right (B) is attempting to fuse secondary to an infectious process and remains consistently lame.

radiographically or fluoroscopically controlled surgical drilling of the articular cartilage is essential to maintain the drill within the joint space to allow the removal of the maximum amount of articular cartilage.

The use of internal fixation in horses with DT disease has not been shown to improve the success rate of arthrodesis beyond drilling alone. If the PIT joint is involved, the prognosis is less favorable, but it too should be drilled, because this offers the best chance of a complete recovery. Other techniques that have been utilized in addition to drilling include intramedullary decompression or fenestration of the DT bones. This has been proposed to provide rapid pain relief in some horses, but this has not been proven. Complications of IA drilling include fracture of the CT or T3 bone(s), development of OA in the PIT joint, and continued lameness despite apparent bone bridging of the DT joints. The latter may be due to micromotion between the tarsal bones and splint bones or bone on bone contact secondary to cartilage destruction from the treatment technique used.

Laser Arthrodesis

Laser-facilitated arthrodesis is another technique to promote fusion of the DT joints. This technique has been most commonly been performed using the neodymium–yttrium aluminum garnet (Nd:YAG) or 980-nm diode laser (Figure 5.72). Laser arthrodesis is thought to be effective because it destroys the articular cartilage by superheating and vaporizing synovial fluid. It has been suggested that the increased temperatures that develop within the joint result in chondrocyte death as well as a collagen shift in the intertarsal ligaments and joint capsule. Reports on horses with laser-facilitated arthrodesis suggest improvement in the successful outcome with minimal postoperative pain and decreased convalescent time compared with other methods.[67,68,130] Surgeons performing this technique suggest that horses with radiographic changes such as severe periarticular osteophyte formation or excessive mineralization of the joint capsule require a modification of the laser procedure to facilitate placement of the laser fiber into the joint. These changes may signify a less favorable prognosis after surgery.

In one study that compared three methods of laser arthrodesis, the energy applied to the DT joints did not result in generalized articular cartilage destruction when evaluated with MRI and histology.[151] Full-thickness thermal destruction of the articular cartilage extended only a few millimeters from the insertion site of the laser across the joint. It was also speculated that the laser may cause less postoperative pain due to thermal damage to nerve endings in the subchondral bone, synovium, and joint capsule, resulting in decreased pain perception in horses after treatment.[151]

Chemical-Induced Arthrodesis: Ethyl Alcohol

Ethyl alcohol is a colorless liquid that has been reported to provide clinical success in facilitated ankyloses of the DT joints. Ethyl alcohol is thought to affect chondrocytes (neurolysis and tissue destruction at the drug–tissue interface) in a manner similar to monoiodoacetate (MIA) but without the severe postinjection pain. MIA has been evaluated and utilized in the past to chemically induce arthrodesis but has fallen out of favor due to the severity of postinjection pain and the development of severe long-term complications (PIT and TC OA).[16,18,37,126] Like MIA, ethyl alcohol can be easily injected into the joints, and because of its neurolytic and nonselective protein destructive properties, it has the potential to block sensory innervation to the joint while causing necrosis of chondrocytes and hastening

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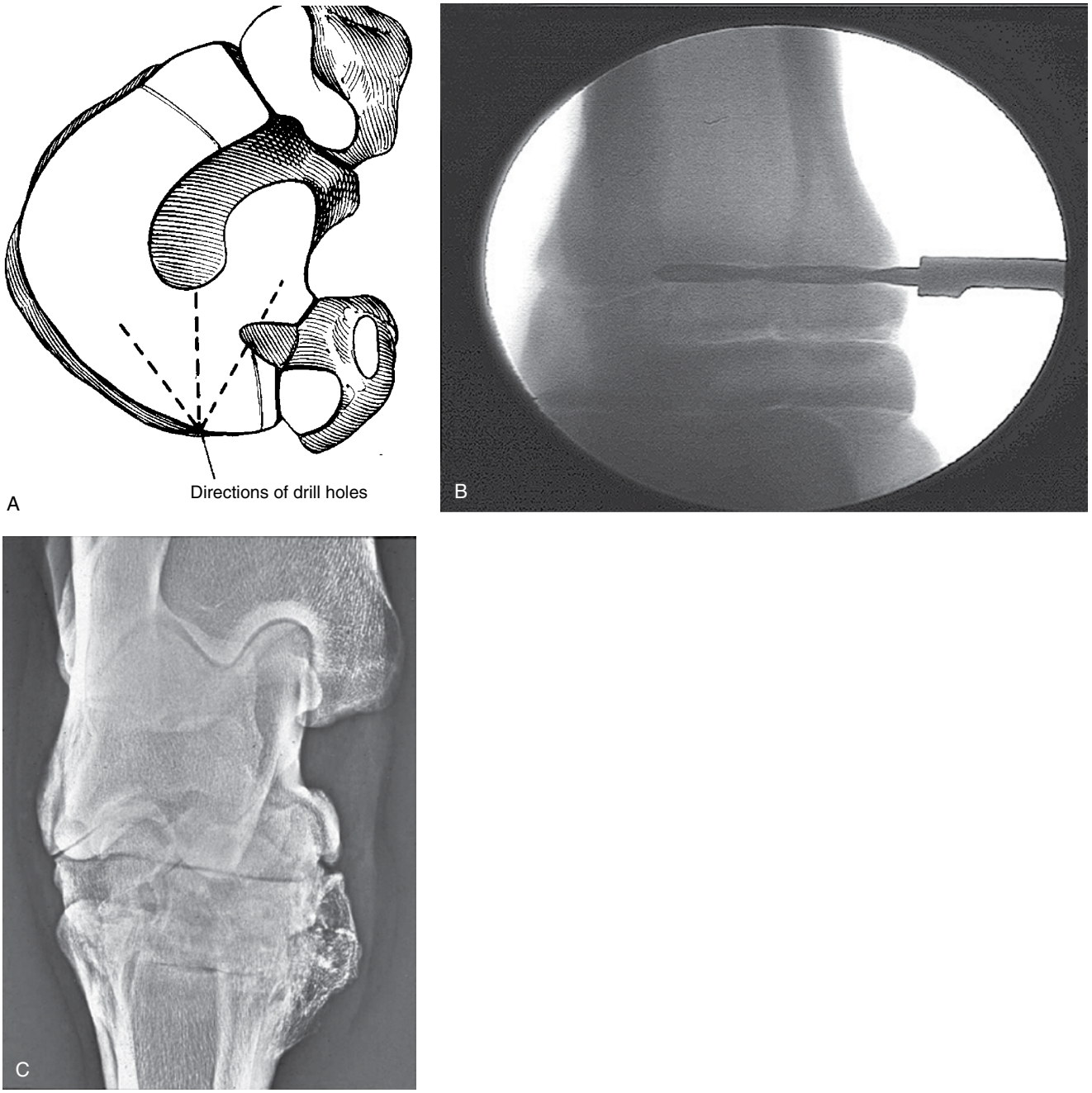


Figure 5.71. DIT joint surface of the central tarsal bone illustrating the 3-drill path technique for distal tarsal arthrodesis (A). *Source*: Courtesy of J.D. Sullins. The drill is inserted through a single hole, passed in three different directions, and can be monitored with radiography or fluoroscopy. (Image is with horse in dorsal recumbency.) (B). A significant amount of joint surface is undisturbed, which preserves stability but promotes arthrodesis of the distal two tarsal joints (C). *Source*: Courtesy of Dr. Gary Baxter.

arthrodesis. Minimal local irritation has been observed in areas of the injections (subcutaneous and intra-articular), with no long-term adverse outcomes such as skin necrosis or hair discoloration.

Administration of ethyl alcohol into the TMT joint of healthy horses appeared to facilitate arthrodesis of the joint in a pain-free manner. TMT joint fusion was achieved with either 70% or 95% ethyl alcohol in 15 of 16 horses without lameness or radiographic evidence of tarsal joint OA.[132] None of the horses were lame after injection, and joint fusion was complete by 8 months. No significant difference in lameness scores was observed between the use of 70% and 95% ethyl alcohol treatments. There was marked, generalized narrowing and extensive, multifocal bony bridging across the joint space between the third tarsal and third metatarsal bones, and the articular cartilage in areas without bony fusion was thin and discolored. The treatment appeared to cause minimal pain and suggested that ethyl alcohol may destroy the sensory innervation of the synovium, joint capsule, and subchondral bone, which permitted fusion of the joint without associated lameness.

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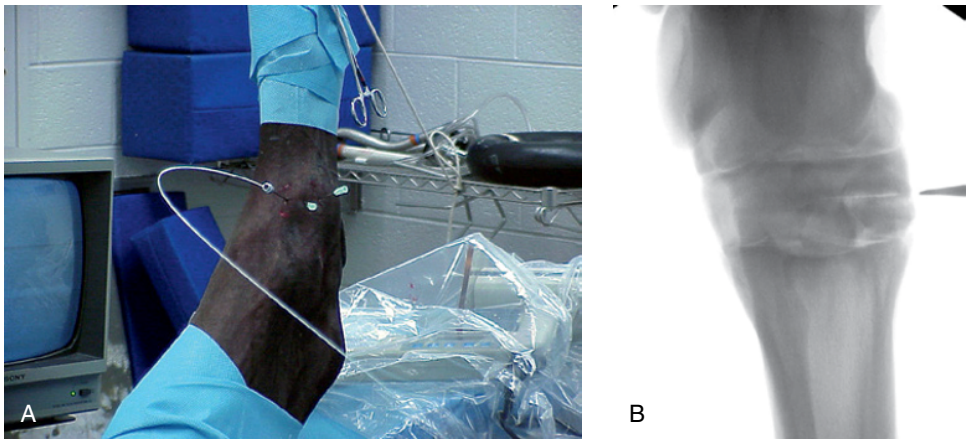


Figure 5.72. Laser arthrodesis of the tarsi is usually performed under fluoroscopic guidance with the horses in dorsal recumbency. The laser fiber is inserted through needles placed into each DIT and TMT joint (A), and the position is confirmed by fluoroscopy (C-arm) or radiographs (B).

A follow-up study using ethyl alcohol in horses with DT OA revealed a rapid reduction in lameness, such that horses could return to their intended use after injection.[25] Contrast arthrograms were performed in these horses and when no communication with the PIT or TC joint could be identified, 3 mL of 70% ethyl alcohol (prepared from 95% laboratory-grade ethyl alcohol) was injected via the needles used for the arthrographic evaluation. Lameness resolved in 18 of the 21 treated horses, but the horses did not develop the degree of joint fusion expected based on the original study in young, healthy horses. However, the horses with clinical OA had a rapid and sustained reduction in lameness such that they could be trained (i.e. brought into work) or competed within days following treatment.

CUNEAN TENECTOMY

The proposed rationale for performing cunean tenectomy is to reduce the torsional forces on the cuboidal bones of the tarsus, which presumably result from tension in the medial extension (cunean tendon) of the cranial tibial muscle.[46,55] The procedure is simple to perform in the standing horse. Clinical reports on the success rates for cunean tenectomy are lacking, but anecdotal information indicates efficacy in many horses. It seems that the clearest indication for cunean tenectomy would be in horses with chronic lameness that has failed to respond to medical treatments and is alleviated by local anesthesia of the cunean bursa. However, infiltration of the cunean bursa with local analgesia may not be specific because of the adjacent joint capsules. Efficacy of cunean tenectomy may depend on early return to exercise before scar tissue replacement becomes restrictive. Currently, cunean tenectomy is performed infrequently because it is unlikely to completely restore soundness.

Neurectomy of the Tibial and Deep Peroneal Nerves

Neurectomy of the deep fibular nerve or a partial neurectomy of the tibial nerves has been reported as a treatment for horses with bone spavin.[73] The technique has reported that approximately 60% of treated horses returned to full athletic function. The authors' conclusion was that the convalescence was shorter than following surgical arthrodesis, but that the surgery time was prolonged due to the sizable incisions that must be closed. However, with all the options present for arthrodesis and facilitated ankyloses, neurectomy of the tibial and deep peroneal nerves should be considered a salvage procedure and not a first-line approach to horses with nonresponsive OA of the DT joints.

Tarsocrural Joint Effusion/Bog Spavin

Synovitis and fluid accumulation within the TC joint is a common finding in young horses and is referred to by laypeople as "bog" spavin. Most often it is a benign nonseptic fluid of unknown cause. When distended, the TC joint protrudes into four distinct pouches. The largest and most prominent is located dorsomedially, with a smaller one dorsolaterally and two large plantar (plantaromedial and plantarolateral) pouches on each side of the joint just dorsal to the tuber calcaneus. Pressure exerted on any one of these swellings can move the fluid between the joint pockets. These fluctuant, movable swellings must be differentiated from periarticular edema (local or diffuse) with less distinct fluid pockets that is more characteristic of an extra-articular joint problem.

Injury to the TC joint can occur due to stops, quick turns, or other traumatic events that can create injury of the joint capsule. Occasionally, performance horses can develop acute TC effusion manifesting as a severe lameness (approaching that of sepsis). Synovitis and capsulitis are probably significant components of the pain creating lameness, but the cause can be elusive. In these cases, radiographic abnormalities are usually absent. Horses are treated with rest and local and systemic anti-inflammatory therapy. Response to therapy can be profound and rapid. However, structural damage such as CL sprain or nondisplaced fracture should be ruled out before using anti-inflammatories and returning the horse to work. As is often noted in horses with osteochondritis dissecans (OCD), the swelling is often not painful to palpation.

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Horses presenting with TC effusion should have a complete lameness evaluation and should have radiographic and ultrasonographic examinations centered on this joint. Horses may have a "bog" that persists for years without lameness even in successfully working horses. There may or may not be significant radiographic signs of disease. As a primary condition, the term "bog" should be limited to those clinical situations in which the inciting cause of the effusion cannot be identified. Lameness can be quite variable and often depends upon the severity of the synovitis. The underlying cause for the development of effusion includes straight hindlimb conformation (through the tarsus), presumably due to continuous trauma to the articular cartilage (Figure 5.73). Subtle OC lesions can worsen with time and can precipitate synovitis/capsulitis and the potential to develop OA. Horses that have mild but clinically significant disease, especially those that require intermittent intra-articular treatment, may eventually, and usually, become more lame as the OA advances.

Many different types of pathology can produce TC effusion (bog spavin). These include OC, OA (including the TC, PIT, and DT joints), traumatic injuries (including CL injuries and capsular tearing), poor conformation, and infection. These will be discussed in more detail in subsequent sections of this chapter. Focal partial- or full-thickness cartilage erosions on the major weight-bearing parts of the trochlea of the talus can exist but may not be apparent on radiographic examination. Surgical arthrodesis and facilitated ankyloses of the DT joints can change the dynamics of the lower joint function, which can lead to the development of OA within the PIT joint, which can manifest as TC effusion. Intra-articular tearing of the TC joint capsule has recently been recognized as a cause of effusion that can only be diagnosed arthroscpically.[5]



Figure 5.73. Horses with a straight limb conformation particularly with a straight tarsal region and a dropped fetlock appear to have a predisposition for DT joint disease presumably due to continuous trauma to the articular cartilage.

It is important to differentiate between TC effusion and periarticular edema. The two may exist concurrently, but they may indicate different problems. Periarticular edema often occurs with CL injury. Any indication that the injury is associated with the intra-articular portion of the short collateral (reported to be more common laterally) is a contraindication for intra-articular injections of corticosteroids (normally used for bog spavin). Scintigraphic examination may be considered with more acute and moderate to severe lameness when there is associated TC joint effusion. Diagnostic arthroscopy is indicated to evaluate the intra-articular environment and initiate treatment when intra-articular injection of anti-inflammatories fails to resolve the effusion or the effusion returns after a short period of time.[5,51]

Many clients want their horses to have a more normal appearance of the tarsus (cosmetically acceptable). Attempts to treat the swelling with HA and corticosteroids may have short-lived success, but the effusion usually recurs. A support bandage can be applied to assist in preventing of the return of the effusion. Repeated injections should be avoided. Some have reported that the use of atropine in nonresponsive cases may have an effect when the corticosteroids do not. There is no known pharmaceutical mechanism of action, but 4–6 mg is being used with noticeable success.

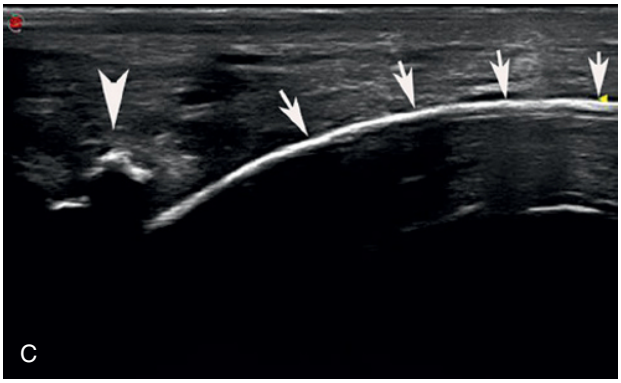
Diagnostic arthroscopy is a very important imaging tool in the evaluation of the effusion of the TC joint. It is considered the gold standard to assess the articular cartilage is many joints, and a study by Barker et al.[5] in 30 horses with soft tissue injuries found that TC arthroscopy provided diagnostic information that was not apparent with radiographic and ultrasonographic examination. TC joint arthroscopy also allows the direct inspection of the intra-articular portion of the CLs and permits completion of necessary surgical procedures (i.e. OC debridement). Arthroscopy is a particularly important imaging tool for many horses with idiopathic TC joint effusion.

OA of the TC Joint

Although uncommon, OA of the TC joint is usually readily apparent. There is usually marked swelling of the entire tarsus, and flexion of the tarsus is extremely painful. Radiographic examination of horses with OA of the TC joint can demonstrate chronic changes with significant periosteal new bone formation and enthesophytosis of the joint capsular attachments (Figure 5.74). A complete ultrasonographic exam is indicated in these cases to evaluate the periarticular tendons and ligaments, the joint capsule, and the joint fluid as well as providing valuable information about the cartilage and subchondral bone surface. In the TC joint, OA tends to cause cartilage degeneration and thinning on the trochlear ridges of the

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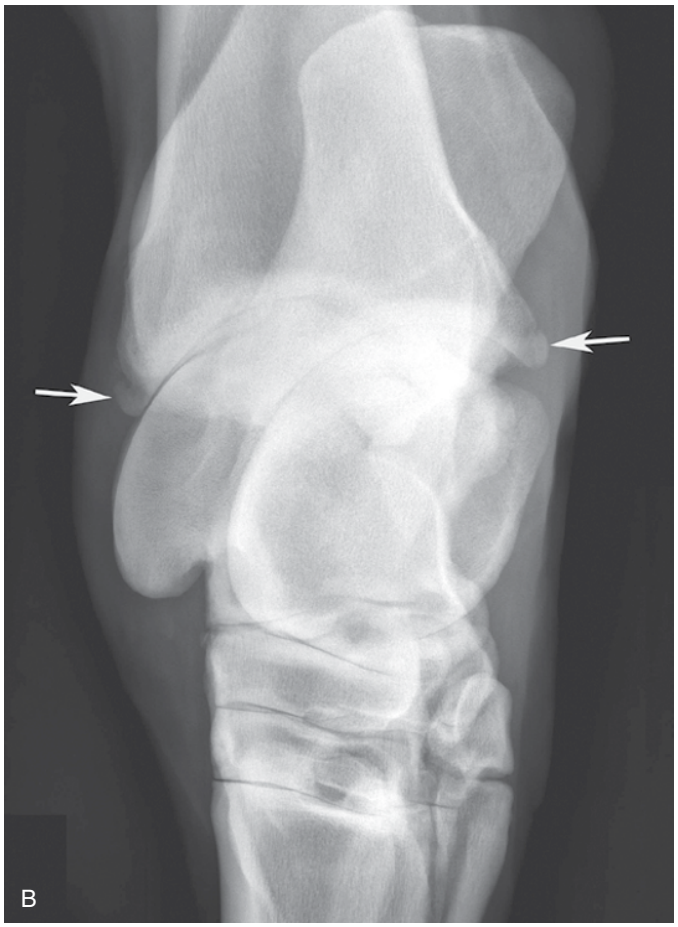


Figure 5.74. Horses with OA of the TC joint can develop chronic thickening of the joint capsule (A; small arrows) and significant enthesophytes of the joint capsular attachments (A and B; large arrows). The longitudinal US image of the lateral trochlear ridge (C) demonstrated significant loss of articular cartilage (arrowheads) and periarticular osteophyte formation (arrow). Proximal is to the left of the image.

talus (Figure 5.74C). Joint capsule thickening, with or without metaplasia, is also a common feature of OA.

Diagnostic analgesia is usually not necessary but may be indicated in some horses that have had persistent swelling but have only recently started to manifest lameness. Intra-articular analgesia of the TC joint may not effectively eliminate the lameness due to the presence of an intact articular cartilage and the presence of substantial subchondral bone pain associated with OA of the TC joint. Perineural analgesia of the tibial and peroneal nerves may be required to improve the lameness.

Most TC disorders have joint distension (bog spavin) as a component of the clinical presentation. As discussed before, not all horses with bog spavin are lame. In addition, the majority of horses with benign effusion of the TC joint will not have a marked response to proximal limb flexion (spavin test). However, the presence of a bog spavin in a horse manifesting lameness needs more careful evaluation of the TC joint. However OA of the TC joint can occur as a primary disease but usually develops due to an underlying problem such as an OC lesion (long-standing DIRT lesion left *in situ* is most common), or it can be a sequela to some traumatic injury of the joint. Long-term administration of corticosteroids into a chronically effusive TC joint can lead to OA particularly when treatment is instituted without an accurate diagnosis. As mentioned earlier, OA of the TC can be seen in conjunction and may be due to extension of OA of the DT joints (it is possible that there is a causal relationship). Some horses that are diagnosed with TC pain may have concurrent talocalcaneal OA as these two joints freely communicate.

Acute onset of a marked to severe weight-bearing lameness associated with swelling of the TC joint frequently indicates a traumatic event. This is often associated with joint distension and/or periarticular swelling of the tarsus. Intra-articular fractures or synovial infections are typically easy to localize and demand immediate attention. Penetrating wounds that breach the synovial membrane of any of the four pouches can lead to significant heat, swelling, and pain of the TC joint. Management of these types of injuries is discussed later in this section.

The presence of a partially mobile OCD fragment such as a DIRT lesion may predispose to OA of the TC joint. Asymptomatic OCD of the intermediate ridge of the tibia is often left alone with the horses placed in show regularly. It is not uncommon to have chronic long-standing DIRT lesion in a horse that suddenly

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develops synovitis, effusion, and moderate lameness later in their career. These horses should have these fragments removed arthroscopically. It is not uncommon to find cartilage damage consistent with OA at time of surgery. These horses can be difficult to keep sound because of persistent low-grade lameness.

OA of the Talocalcaneal Joint

The talocalcaneal joint is a curved articulation between the plantar aspect of the talus and the dorsal aspect of the calcaneus. Its soft tissue support arises from 4 ligaments—the medial, lateral, proximal, and interosseous talocalcaneal ligaments.[60] The mechanics of this joint have not been described, but it is considered a low-motion joint.

OA of the talocalcaneal joint is not common, and descriptions of this condition are based on case reports.[134,139] However, when it occurs, it is usually a moderate to severe unilateral hindlimb lameness. There are usually no localizing clinical signs. Lameness is typically exacerbated by proximal limb flexion of the hindlimb. The clinical signs resemble those of horses with DT OA but tend to be more severe. The cause of this condition is unclear, but because of the usually sudden onset of lameness, it is assumed to be an acute soft tissue injury probably related to disruption of the talocalcaneal ligaments.

Diagnosis requires elimination of the lameness by diagnostic analgesia and confirmation with diagnostic imaging. Talocalcaneal joint analgesia has not been found to effectively eliminate the lameness and at best gives only partial improvement. Perineural tibial and peroneal analgesia has been the most reliable method of reducing the lameness. Clinical examination, history, results of regional analgesia of the tibial and peroneal nerves, the presence of radiographic changes of this joint, and nuclear scintigraphy can help to confirm talocalcaneal OA as the cause of lameness (Figure 5.75). The lateromedial radiographic view is the best projection to evaluate this joint for changes consistent with OA including subchondral bone lysis, sclerosis, and irregular joint space width. Nuclear scintigraphy can demonstrate marked IRU in the plantaromedial aspect of the tarsus.

In the largest reported case series of 18 horses with talocalcaneal OA, 14 horses were treated conservatively with stall or pasture rest, with or without intra-articular corticosteroids, HA, or PSGAG, and all remained lame. Intra-articular corticosteroids appeared to have little effect on any horse.[133] Follow-up exams often demonstrated a rapid progression of radiographic changes. Because of the poor response to conservative management, more aggressive therapies are now being attempted. Subchondral forage and local neurectomy have been utilized but have been unsuccessful. Six horses have had a surgical arthrodesis using 2 or 3 5.5-mm screws placed in a neutral fashion. All of these horses improved significantly, but none became sound. Interestingly, one of these horses developed contralateral talocalcaneal OA 2 years later. While surgical arthrodesis is now the treatment of choice, the prognosis remains poor for a return to performance soundness.

Developmental Orthopedic Disease of the Tarsus

Developmental orthopedic disease (DOD) is a term used to describe many orthopedic problems seen in the young horse. It is discussed in further detail in Chapter 10. The term describes a series of growth disturbances of young horses including OC (OCD), SCLs, angular deformities, flexural deformities, physitis, cuboidal bone abnormalities, and juvenile OA. All the forms of DOD can occur in the tarsus.

OC of the TC joint occurs at very consistent sites.[64,66] TC OC tends to occur at the edges/limits of the talus

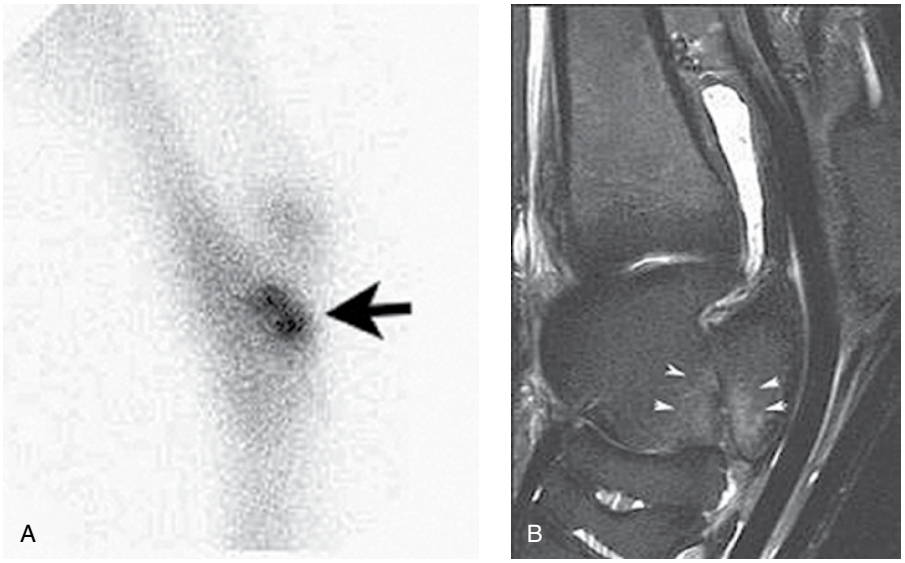


Figure 5.75. Horses with talocalcaneal osteoarthritis (OA) usually present with unilateral moderate to severe hindlimb lameness without localizing clinical signs. Nuclear scintigraphy (A) and MRI (B; arrows) can be very helpful to identify the site of bone remodeling/pathology within the joint.

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and tibial articular surfaces where the makeup of the cartilage between articulating and non-articulating surfaces is different. Weight-bearing produces sheer stresses within the immature cartilage at these sites, which damages the vascular supply to the articular cartilage. Increases in weight and activity of the foal increase the biomechanical stresses placed on the articular cartilage that can increase the incidence of lesion development. Silent or quiescent lesions may become apparent only when the horse is started into training and the joints become challenged by athletic activity. The age at which this occurs can vary with the discipline of the horse. For example, Warmblood horses commonly present with clinical signs of TC OCD (TC joint effusion) around 3 years of age or older, whereas racing TB and STB manifest signs much earlier (2 years of age) because they begin race training earlier in life. OC lesions can also undergo spontaneous resolution. This occurs by intramembranous ossification of granulation tissue that forms deep in the lesions progressively filling of the OC defects with bone. While OC of the DIRT and LTR of the TC joint can be radiographically evident within a few months of life, they may not be clinically detectable until later. Lesions found before the age of 5 months may not persist, but those lesions detected after 5 months typically will persist thereafter.[36]

OC can occur in all joints, but most often they occur in the hock, stifle, and fetlock joints. Multiple joints can be affected, but usually only one joint is involved. Bilateral involvement (i.e. both stifle joints or both hock joints) occurs often enough so that the opposite joint should always be radiographed. Bilateral occurrence can also occur with only one joint becoming clinically apparent (manifesting effusion). This can occur in the TC and femoropatellar (FP) joints in more than 50% of clinical cases. However, it is uncommon for both the hocks and stifles or the hocks and fetlocks to be involved in the same animal at the same time. Lameness is seen more often in FP OC than in TC OC and is most likely to occur with an acute onset of effusion. Lameness also often occurs when there is a loose or semi-loose osteochondral fragment observed radiographically. It is also uncommon to treat OCD in one joint(s) and have it develop at a later time in other joints.

Osteochondritis Dissecans of the Tarsocrural Joint

OC occurs in the TC joint commonly.[10,12,22,66,84,89,92,93,110,122,136] Horses of all ages can be affected. Many horses are sound at a walk and trotting in hand, but may display lameness at faster speeds or when put into work. Lameness when it does occur varies with location and severity of the OCD. These fragments are usually found when radiographs of the joint are taken for another reason, most often during prepurchase examination. TB sales horses are routinely radiographed as a screening for the yearling sales. The most common clinical sign of OC in the tarsus is **effusion** in the TC joint. Lesions can be identified as fragments *in situ* (intermediate ridge) or surface irregularities of the trochlear ridge(s) or malleoli (Figure 5.76). OC lesions of the TC joint often develop into OCD lesions because they often form separated fragments. This is probably due to the site vulnerability at the edges of the articulation mentioned earlier. The most common locations for OCD in order of occurrence are the DIRT (81%), the distal aspect of the LTR (16%), the cranial distal aspect of the MM (3%), the MTR, and the LM.[5] The distal end of the tibia is the most common site of OC formation and may be related to impact to or impingement of the DIRT onto the talus/central tarsal bone when the tarsus is in full flexion. Acute severe synovial effusion can occur when the fragment(s) is disturbed presumably by hyperflexion and impact onto the central tarsal bone. The synovial distension can present clinically as severe lameness. If clinical signs appear after training has begun, lameness is more likely. However, once the horse has adjusted to the capsular stretching, the lameness quickly subsides. Once loose, micromotion of the fragment(s) can create persistent inflammation within the parent bone with

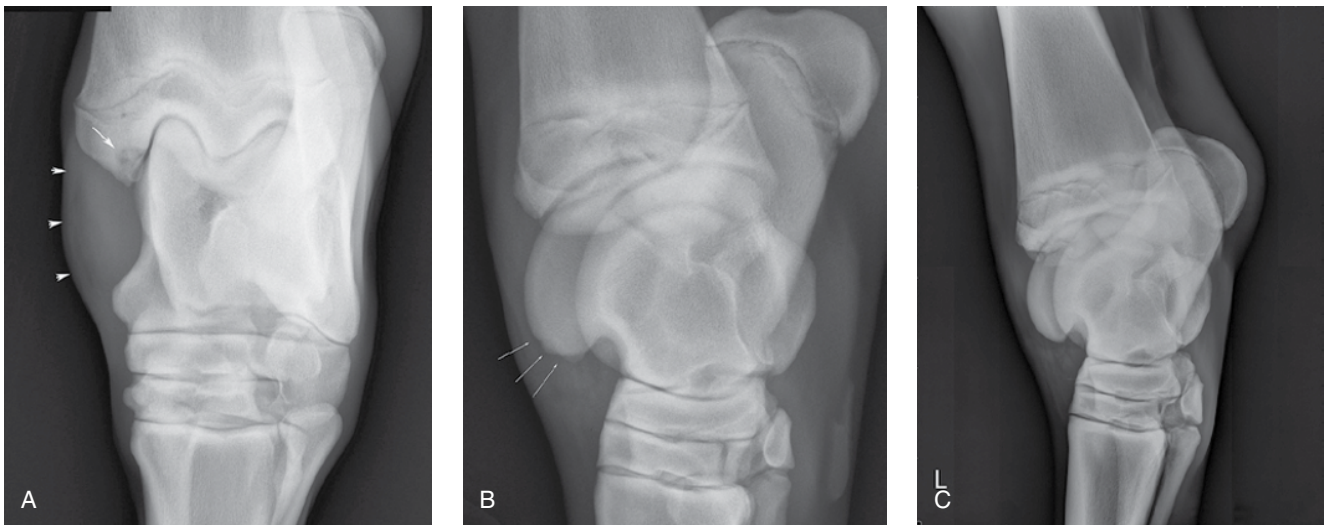


Figure 5.76. The most common clinical sign of OC in the tarsus is effusion (A; small arrowheads) in the TC joint. Osteochondrosis lesions can be identified as in situ fragments of the distal intermediate ridge of the tibia (C), surface irregularities of the lateral trochlear ridge (B; arrows) but occasionally the medial trochlear ridge, and lucency of the medial malleolus (A; large arrow).

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release of biochemical/inflammatory and physical debris into the joint. Continual shedding of biochemical and physical substances into the joint can lead to chronic synovial effusion or thickening of the fibrous joint capsule of the joint. In sites other than the DIRT, the OCD lesions often remain loosely attached in their bone of origin, but the debris that is released into the joint from beneath the flap results in synovitis or joint inflammation. Occasionally an OCD fragment is made entirely of cartilage and cannot be identified on radiographs. The radiolucent defect left in the parent bone may be all that is apparent in many cases. If the OC and cartilage flap is more chronic, then the cartilage flap may begin to ossify and be radiographically apparent. In contrast, some older horses are diagnosed with OCD incidentally without apparent clinical signs.

The radiographic abnormalities associated with OC/OCD include fragmentation; irregular, flattened contour of the subchondral bone surface, with or without subchondral radiolucencies and/or sclerosis; and occasionally secondary OA. DIRT lesions are best visualized on the DM-PlLO projection. Lesions of the trochlear ridges may be evaluated on the DM-PlLO (lateral ridge), DL-PlMO (medial ridge), or LM projections. OC of the MTR is rare and must be differentiated from the normal variably sized protuberance of this ridge, where normal spurs and fragments can occasionally be seen.[23,27] Free fragments seen in the dorsal aspect of the joint can originate from the trochlear ridges (usually at the distal aspect of the LTR) but can also originate from the DIRT area.[137] OC/OCD of the MM is best demonstrated with a DPl or DL-PlMO projection and must be differentiated from avulsion fractures of the CLs. Flexed projections may be useful to demonstrate loose fragments in the plantar recesses of the TC joint or OC lesions in the proximal trochlear ridge.[17,104]

Radiography often underestimates the extent of the cartilage and bone damage that is later identified at surgery, particularly for LTR lesions. The TC joint can manifest radiographically silent lesions (lesions identified at surgery where no abnormality was seen on radiographs) more commonly than in other joints. Occasionally, purely cartilaginous loose bodies that are not seen radiographically can be well identified using arthroscopy.[17]

The use of diagnostic US should be considered in the evaluation of tarsal OC to complement radiography. Diagnostic US can be a very useful tool to help evaluate the thickness of the articular cartilage and the surface of the subchondral bone plate (Figure 5.77). It can also help provide valuable information about the joint environment (synovial membrane, synovial fluid, articular cartilage, and subchondral surface) in any horse with joint effusion. Diagnostic US can be particularly useful in cases where OC is suspected, but the radiographs are inconclusive. This examination of the joint can be quite useful to help determine the primary site of OC and to assess the size and the extent of the lesion before surgery.

Resolution of the effusion generally requires surgery and the removal of the abnormal tissue. In the tarsus, surgery can be considered early after the recognition of the OC assuming the foal is older than 7 months of age. Unlike stifle lesions, OC lesions in the tarsus are not at risk of maturing or increasing in size. The earlier the OC is removed, the more likely the effusion and joint capsule swelling will resolve, although not all horses with OC of the tarsus require surgery. Horses with small lesions, with minimal effusion, and without lameness may not require surgery particularly if they are pleasure horses or light use horses. However, loose flaps and large areas of abnormal cartilage and subchondral bone can lead to persistent inflammation in the joint and over time may lead to the development of OA. If significant degenerative changes of the articular cartilage are identified at surgery, the prognosis for the return to soundness is less favorable.

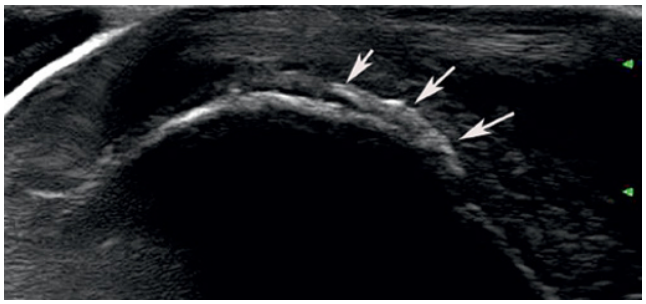


Figure 5.77. Osteochondrosis lesions can also be accurately identified on ultrasound examination as an irregularity to the subchondral surface, as thickening of the articular cartilage, and occasionally as a calcifying flap separating from the subchondral surface (arrows) as demonstrated in this transverse ultrasound image of the lateral trochlear ridge.

Treatment of choice for OCDs of the TC joint is arthroscopic removal of the fragment and debridement of the damaged cartilage and subchondral bone. The removal of the OCD lesion surface (unstable articular surface) stops the inflammatory process within the parent bone and eliminates the shedding of debris into the joint. If surgical removal of the fragment(s) and debridement of the parent bone occurs before permanent damage to the articular cartilage occurs, the prognosis is excellent for resolution of the synovial effusion and restoration of normality to the joint.[92,94] Fortunately OCD of the TC joint often occurs at sites that are easily accessible/amenable to surgical removal and debridement. Osteochondral fragmentation of the distal aspect of the MTR is not often a surgical problem as it is usually within the synovial attachment of the TC joint capsule. Because the OC lesion is embedded within the synovium, it does not shed debris within the joint and rarely becomes a clinical problem.

The clinical significance of DIRT lesions and the effect they have on performance have often been questioned. There appears to be little apparent influence on performance of the young Thoroughbred racehorse.[10] Comparisons between the racing performance of horses with TC OC treated conservatively and those without TC OC were unable to demonstrate any significant differences in the lifetime racing performance.[22] More subjective influence on performance may exist in more mature performance horses. Historically, DIRT lesions that were found in sport horses on prepurchase or on routine radiographic examination of the tarsus were left *in situ* provided they were not creating effusion or lameness. However, it is not uncommon to have these horses

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develop synovitis and lameness later in their career. To appropriately manage these horses, the fragments should be removed arthroscopically. Cartilage damage consistent with OA is often found at time of surgery, and some of these horses can be difficult to keep sound because of persistent low-grade lameness. It has previously been suggested that older show jumpers may dislodge intermediate ridge fragments resulting in acute lameness. Based on these experiences, it may be advisable to remove potentially mobile fragments in the TC joint because of the potential for horses to develop OA as mature performance horses.

In young horses the prognosis for athletic activity after surgical removal is good. TC effusion resolved after surgery in 89% of racehorses and 74% of nonracehorses. Effusion appears to be less likely to resolve if the lesions were on the LTR or MM. However, there appears to be no correlation between resolution of effusion and athletic performance. Lesion location and unilateral vs. bilateral lesions had no effect on ability to start. Although the trend was present in all groups, only 2-year-old Standardbreds were significantly less likely to start a race when multiple sites within the TC joint were affected.[12]

Proximal Intertarsal Joint Synovitis and OA

Loose fragments, usually shed from the intermediate ridge of the distal tibia, can become lodged in the dorsal pouch or dorsal joint capsule of the PIT joint. Although rarely of clinical significance, these fragments can be removed arthroscopically through the TC joint if considered necessary.[136] Because of their limited clinical significance and the relative difficulty of finding and removing the fragments, it has been recommended that they be removed only if this can be easily accomplished during surgery for the primary lesion of the TC joint (Figure 5.78). Synovitis/osteoarthritis of the PIT joint is usually due to progression of DT OA but can also occur from external trauma. Radiographic signs of PIT OA are very similar to those of the distal joints and are usually more prevalent on the medial aspect of the tarsus (Figure 5.79).



Figure 5.78. Loose fragments (arrows) usually shed from an OCD lesion on the distal intermediate ridge of the tibia can become lodged in the dorsal pouch or dorsal joint capsule of the PIT joint. Although rarely of clinical significance, these fragments can be removed arthroscopically through the TC joint.

Subchondral Cystic Lesions (SCLs)

SCLs are typically seen in young horses but can occur in any aged animal.[55,69,71,98] Not all horses with SCL are lame and many can be clinically quiescent. Cystic structures associated with the tarsus have occurred in the MTR or LTR of the talus, the intertrochlear groove, the MM or LM, DIRT, distal aspect of the tibia, calcaneus, CT, T3, and proximal MT3 (rare). SCLs are characterized by radiolucent areas of bone (from several millimeters up to and even larger than 3 cm) and are often accompanied by a thin, but well-demarcated, sclerotic rim (Figure 5.80). The cysts can be uni- or multi-loculated and may or may not communicate with the joint. Lesions that are close to or involve the articular surface are more likely to cause lameness. Lameness associated with SCL is believed to be the result of synovitis and increased intracystic or intraosseous pressures leading to subchondral bone pain. However, it is unclear why some SCLs are asymptomatic.

There are three predominant hypotheses about the possible etiology for SCLs in the tarsus: (1) related to a developmental failure of endochondral ossification, (2) acquired and caused by some form of cartilage injury and subchondral bone trauma and (3) due to an infectious cause. SCLs occur in younger animals and are thought to be related to OC lesions that occur at sites of major weight-bearing.[98] Compressive loads on an OC lesion can lead to a further weakening of the subchondral bone and formation of SCLs. Mature animals can develop SCLs potentially from some form of mechanical trauma. A supraphysiological load on a normal articular surface may initiate sufficient bone injury that can lead to the creation of an SCL. The *hydraulic theory* assumes the formation of SCLs is caused by joint fluid being forced into a cartilage defect. Infection is known to cause SCLs in many locations in young animals.

The standard radiographic examination of the tarsus may not adequately demonstrate SCLs because of their small size and position within a complex joint. Nonstandard radiographic views may be necessary to document these lesions. When visible, an SCL appears as a discreet radiolucency varying in size and shape but often surrounded by a sclerotic border. Advanced diagnostic imaging modalities such as CT and/or MRI that represent the limb in cross section are ideal to document their location but not always practical as they require general anesthesia. Scintigraphic examination can be helpful

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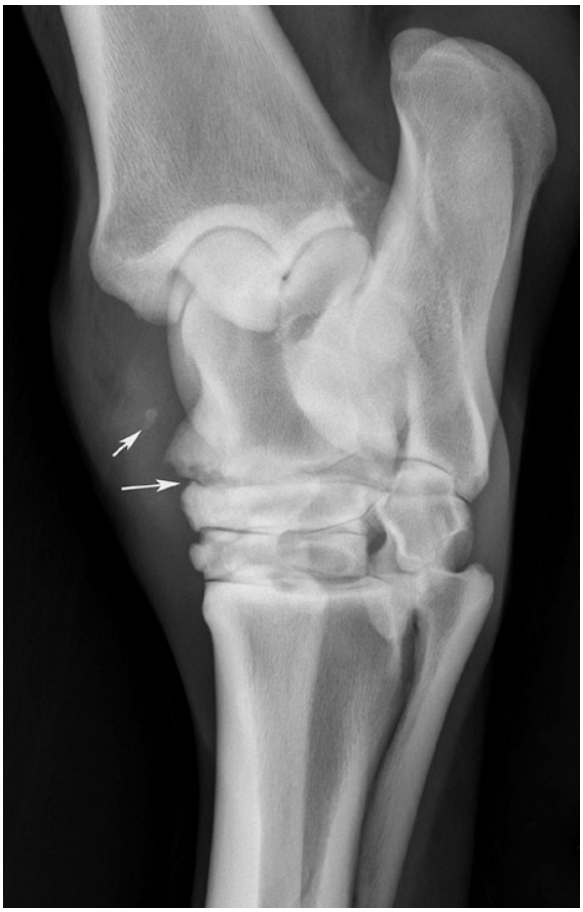


Figure 5.79. Osteoarthritis (OA) of the proximal intertarsal joint (PIT) tends to occur in conjunction with and in progression with OA of the distal tarsal joints. Radiographic changes typically are more evident medially as seen on this radiograph (arrows).

to identify SCLs as discrete foci of IRU in the tarsus consistent with an active bone remodeling response.[58] However, scintigraphy lacks the specificity to characterize the size and to accurately locate the cystic structure and determine communication with the joint. In fact some cystic lesions are portrayed as a photopenic area, making them difficult to see these small cystic structures. Visualization of SCLs in the talus and distal aspect of the tibia by diagnostic arthroscopy has been described.[69]

Depending on the location and extent of the lesion, surgery may be required to prevent cyst enlargement and promote filling of the lesion with osseous material. Depending on the cyst location, this can be performed arthroscopically or via an extra-articular approach. Additional techniques to improve the healing response in bone and cartilage may be indicated to preserve articular function.

Malformation of the Distal Tarsal Bones

Malformation of the DT bones can occur in dysmature/immature foals but is most often recognized in premature foals.[2,3] The small cuboidal bones of the carpus (knee) and tarsus (hock) begin as a cartilage model with a central area of ossification *in utero*. As the wave of ossification progresses, the carpal/tarsal bones change from a circular central area of ossification into a bony cube. In normal foals, tarsal bone ossification continues after birth and is recognized radiographically by a wider appearance of the joint space due to the presence of more cartilage compared with an adult horse.[23] If a foal is born prematurely or is dysmature at birth, standing and normal activity on an incompletely ossified bone can crush the soft cartilage model.[3] (Figure 5.81A) Wedging of the dorsal/dorsolateral aspect of the central/third tarsal bones can occur (Figure 5.81B). These foals will often appear to have an angular limb deformity (lateral or medial crushing) in the carpus or a sickle-hocked conformation (dorsal crushing) in the tarsus.

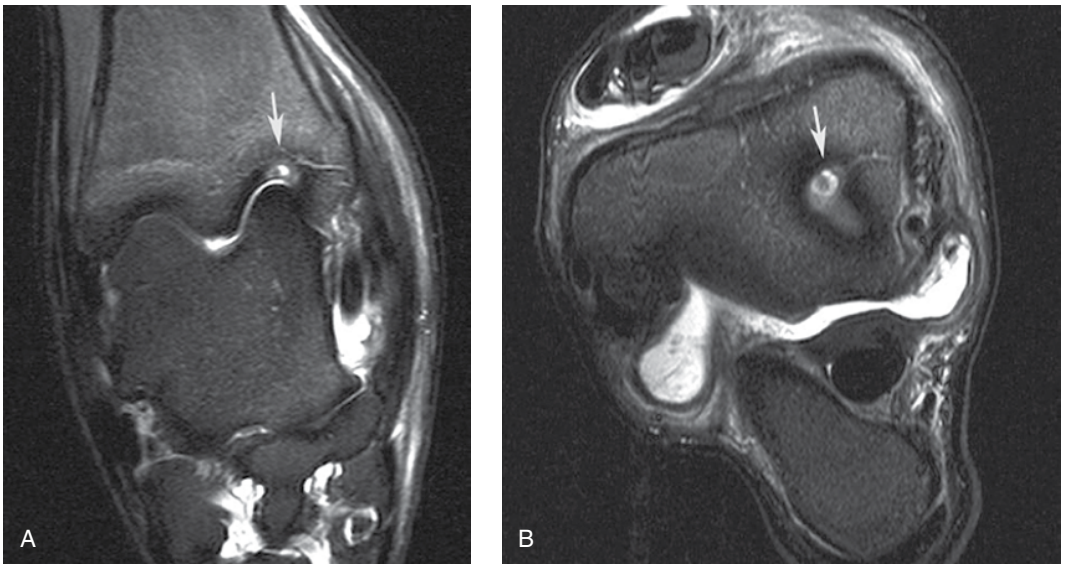


Figure 5.80. An SCL in the distal tibia identified on these two MR images (A and B; arrows) communicated with the TC joint in this horse and caused significant effusion.

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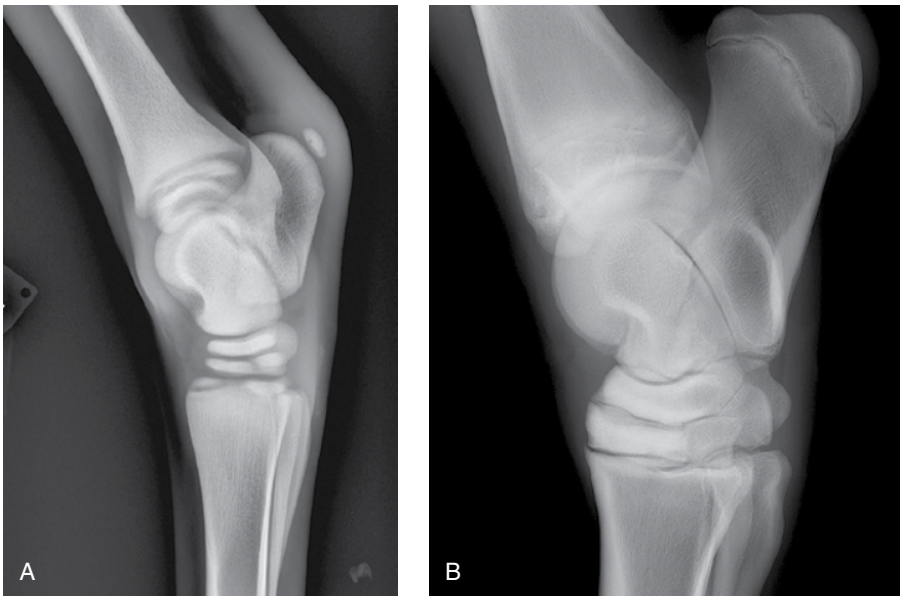


Figure 5.81. Malformation of the distal tarsal bones can occur in dysmature/immature foals but more often occurs in premature foals (A). Developmental abnormalities such as wedging of the third tarsal bone may be a predisposing factor to formation of degenerative changes within the distal tarsal joints (B).

The disorder is often bilateral.[2,23] Once the cartilage model/immature bones are crushed, the malformation becomes permanent. The cuboidal bones continue to ossify around the malformed cartilage model. Continued loading on these immature bones may result in valgus deformities and/or pathological fracture or collapse of the tarsal bones. The reader is referred to Chapter 10 for more information about this condition.

Angular Deformities of the Tarsus

Angular limb deformity refers to an axial deviation of a limb in the frontal plane. The nomenclature to describe an angular deformity is derived by combining the name of the involved joint (i.e. with cuboidal bone abnormalities) or the joint immediately distal to the affected growth plate and the type of deviation. A lateral deviation of the distal limb is termed valgus, while a medial deviation is termed varus. Tarsal angular deformities often originate from within the physis of the distal tibia but can also occur from the cuboidal bones of the tarsus (see above). Tarsal valgus deformities are more common than tarsal varus deformities. Perinatal factors are usually involved when a foal is born with an angular limb deformity. In neonatal foals angular deformity can be a consequence of laxity of the supporting soft tissues of the tarsus or related to hypoplasia and/or incomplete ossification of the tarsal bones. A large number of foals are noticed to have some type of angular deformity at birth. The cause of angular limb deformities is complex and probably multifactorial and is discussed in more detail in Chapter 10.

FRACTURES AND LUXATIONS OF THE TARSUS

Fractures and luxations of the tarsus are rare probably due to the dense supporting structures that cover the bones in this area.[29,52,75,97,104,105] Fractures when they do occur are usually associated with a significant external force or impact, such as a kick from another horse. Twisting and torqueing of the tarsus while the lower limb is fixed is felt to contribute to disruption of collateral and periarticular ligaments and subsequently lead to fractures. These animals are almost always non-weight-bearing lame, and the tarsus is diffusely swollen. Fractures of the talus can range from simple fragmentation of the trochlear ridges to comminuted fractures of the entire body of the talus. The degree of comminution usually determines the severity of the lameness and the extent of associated swelling. The degree of lameness associated with fractures of the tarsus can be mild to moderate in the case of chronic central tarsal bone fractures to severe and non-weight-bearing such as in comminuted fractures of the talus or calcaneus. External trauma can fracture specific areas of exposed bone such as the tibial malleoli, trochlear ridges, tuber calcaneus, and fourth tarsal bone. Injuries that involve the TC joint are frequently more apparent due to swelling of the dorsomedial and dorsolateral synovial pouches. Fractures of bones within the TMT and DIT joints are less noticeable due to the dense ligamentous coverage and tight joint capsules that limit swelling and effusion. Luxations of the tarsus are uncommon and often involve significant trauma that disrupts many of the support structures around the tarsus.

Traumatic Luxations of the Tarsus

Traumatic luxations that involve the tarsus are usually created from kicks from other horses, trauma from moving objects, or by having the limb entrapped in fixed objects such as a fence or cattle guards. The soft tissue injuries and fractures that accompany TC luxation are often so significant and incapacitating that salvage of the horse is frequently not possible. Lameness is usually

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severe with significant soft tissue swelling and palpable instability and crepitus. The diagnosis is normally not difficult and is substantiated through radiographs. Luxation of the TC joint is often accompanied by a severe angular limb deformity centered at the affected articulation of the tarsus. Traumatic luxations of the other joints of the tarsus usually occur at either the TMT joint or the PIT joint presumably because of the stabilizing effect of the fourth tarsal bone (Figure 5.82). Clinical diagnosis is not difficult, but it is best to verify the injury through diagnostic imaging (radiographic and ultrasonographic examination). Stress radiographs of the tarsus can be helpful to demonstrate abnormal hock motion (in a sagittal plane) not easily seen with conventional radiography. Specific stress maneuvers can include traction, axial rotation (clockwise and counterclockwise), hyperextension, hyperflexion, and fulcrum-assisted medial and lateral flexion. Each maneuver is designed to demonstrate instability in a particular part of the tarsus and, in the process, to identify damage to one or more specific ligaments. Some luxations and subluxations of the tarsus may reduce spontaneously, but they can still be demonstrated with stressed dorsoplantar radiographs. The presence of fracture(s) associated with a luxation generally decreases the chances for successful treatment. General anesthesia is usually required as it can be very painful to the horse. Luxations need to be carefully evaluated for vascular injury to the arterial supply to the distal limb before moving forward with management.

Treatment usually requires first aid to safely transport the horse to a surgical facility for reduction and stabilization in some fashion.[19] TC joint luxation with intact CLs can be difficult to reduce, and manipulation of the tarsus with the limb in a flexed position may help to facilitate reduction (Figure 5.83). A decision about repairing the injury should be made quickly and attempted during the same anesthetic period. While luxation through the TMT or PIT joints can be repaired, these injuries usually significantly compromise athletic soundness. Treatment is aimed at reestablishing comfortable weight-bearing. Reduction of the luxation with either cast stabilization or surgical repair should be performed, and an assisted recovery performed if available. Tarsal luxations in the absence of significant fractures can be treated with a full-limb cast for 2–3 months. Full-limb cast application should extend from the foot to the level of the tibial tuberosity. Cast immobilization of a hindlimb is not without problems and can often be associated with significant complications. The most common cast complications included pressure sores, cast breakage, and secondary fractures. To minimize the risk

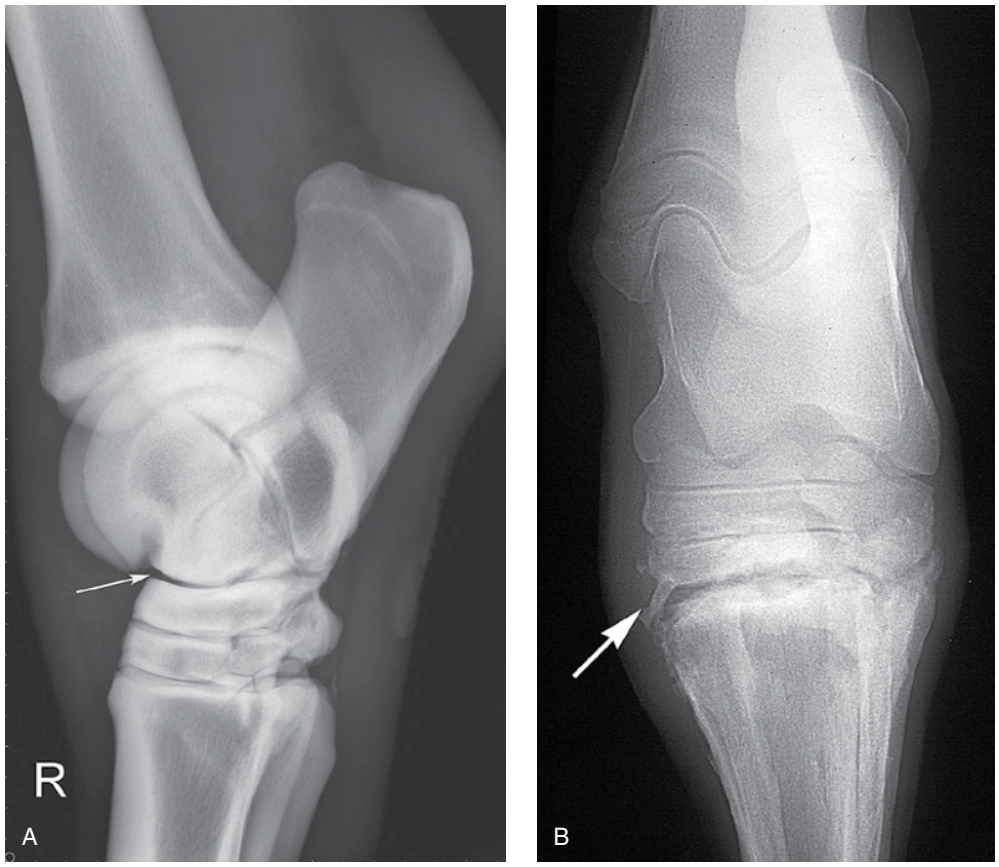


Figure 5.82. A stressed lateromedial radiograph (A) used to help demonstrate opening of the joint surface in this horse with a PIT joint luxation. This horse was maintained in a full-limb cast. Dorsoplantar radiograph (B) of a foal with luxation of the medial aspect of the TMT joint (arrow). A medial plate was used to stabilize the luxation and arthrodese the distal tarsal joints concurrently. *Source*: Courtesy of Dr. Gary Baxter.

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of complications of a full-limb cast application, the limb can be maintained in a cast for 4–6 weeks if the CLs are intact and 6–8 weeks if the CLs are ruptured. After the cast is removed, the hindlimb is placed in a heavy cotton bandage with rigid splints for an additional 4–6 weeks before turnout in a small area is allowed.

Surgical arthrodesis of tarsal luxations can be performed to help achieve pasture soundness for breeding purposes.[1,105] However, the prognosis is still guarded. Surgical techniques described for management of tarsal luxations in horses include articular cartilage removal combined with internal fixation using transarticular screws or bone plating. Cast immobilization is usually combined with surgical management. Open reduction and internal fixation with a plate is usually recommended for tarsal luxations when there are associated fractures. Surgical arthrodesis of the TMT, DIT, and PIT joints can be achieved by placing a plate on the plantar surface of the joint. The plate is placed on the plantarolateral aspect of the tarsus and extends from the top of the calcaneus to mid-MTIII.



Figure 5.83. TC luxation can cause significant soft tissue injury(s) and secondary fractures that are often so significant and incapacitating that salvage of the horse is frequently not possible.

Slab Fractures of the Small Tarsal Bones

The DT bones are subjected to axial compression, torsional, and tensile forces during exercise. Their main function is to absorb concussion and neutralize these forces. The DT joints resist these forces and are stabilized by a thick layer of ligamentous tissue that surrounds and unites the small tarsal bones. The dorsal tarsal ligament (DTL) is a major component of these ligamentous structures. The DTL originates from the distal tuberosity of the talus on the medial aspect of the tarsus and spreads out as a sheet over the face of the DT bones restricting/limiting movement. The dorsal aspect of the tarsal region also has the extensor bundle comprised of the tendons of insertion of cranialis tibialis and PT and, laterally, the long digital extensor.

Slab fractures of the CT or T3 bones occur most often in racing disciplines or other high-speed events.[101,141] Slab fractures are felt to be exercise-induced injuries in racehorses and result from accumulated bone damage on the dorsal aspect of the DT bones.[141] They occur at similar locations and configurations in the absence of specific trauma (Figure 5.84). Third tarsal bone slab fractures occur most commonly in young racehorses with one study reporting the median age of 2 years. Fractures of the T3 bone are more common in TB and STB racehorses and are normally found on the dorsal and dorsolateral aspects of the bones (dorsomedial to plantarolateral orientation). Tarsal slab fractures do occur in other breeds and disciplines but are less likely to be due to adaptive remodeling from exercise but more likely due to some

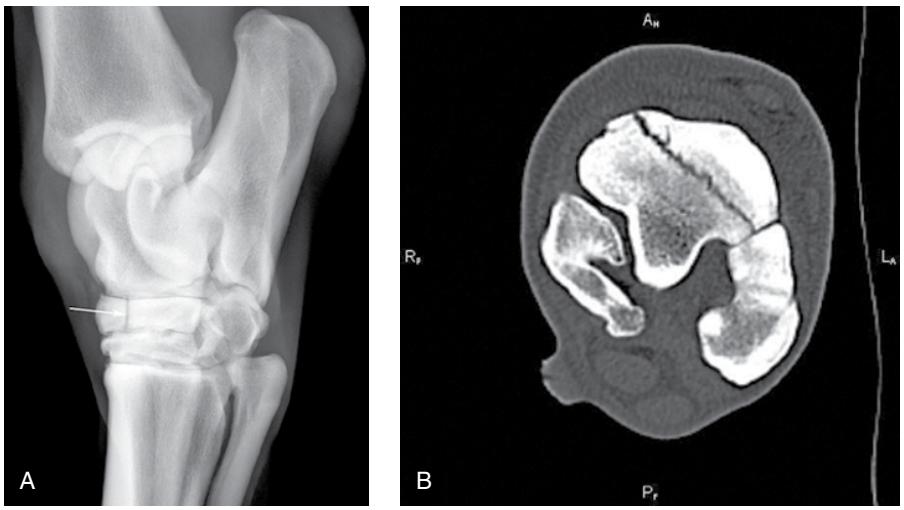


Figure 5.84. Tarsal radiograph (A; arrow) and CT (B) demonstrating a slab fracture of the central tarsal bone. These slab fractures occur most often in horses used for racing disciplines and other high-speed events.

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form of specific trauma. These fractures are more likely to involve both the CT and T3 bones and are more likely to be comminuted (Figure 5.85).

Horse with slab fractures can initially present with severe lameness that diminishes over several weeks. There can be little indication of a slab fracture. Simple slab fractures of the T3 bone often have minimal swelling or synovial effusion to indicate the location of the injury. Synovial effusion may occur with CT fractures that enter the PIT joint. Racehorses that have a moderately positive response to proximal limb flexion test when compared with the opposite limb should be evaluated for tarsal slab fractures. When the CT bone is fractured, intra-articular anesthesia of the TC joint may partially alleviate lameness, but it can take 30–45 minutes for maximal effect. Diagnosis of CT and T3 bone fractures is confirmed with standard radiographic views but can require multiple oblique projections. Radiographic confirmation of these fractures may prove difficult because the radiographic beam angle must be tangential to the fracture plane to identify them. This requires that radiographic exam includes a series of different oblique angles to more accurately demonstrate the fracture. The radiographic diagnosis can be further complicated because the curved nature of the fracture plane makes it difficult to image. In addition, because the fracture line may be difficult to appreciate soon after injury, serial radiographic examinations may be necessary to identify the presence of a slab fracture. When conventional imaging techniques (radiography and ultrasound) cannot detect a problem, nuclear scintigraphy can be utilized to demonstrate active bone remodeling associated with the fracture. CT and MRI can provide cross-sectional imaging that is ideally suited for an accurate diagnosis but also a prerequisite for surgical repair. Multiple fracture lines that were undetectable with standard radiographic exam can often be identified making the surgical treatment (screw placement) more accurate even though the prognosis becomes less favorable.

Developmental abnormalities such as wedging of the T3 may be a predisposing factor to formation of slab fractures of the DT bones. These tend to occur in dysmature foals with hypoplastic tarsal bones that have collapsed on the dorsal or dorsolateral portion of the third tarsal bone creating a wedge shape. Third tarsal bone slab fractures occurred more commonly in the left hindlimb of racehorses, while TC bone fractures were more often complex and comminuted.

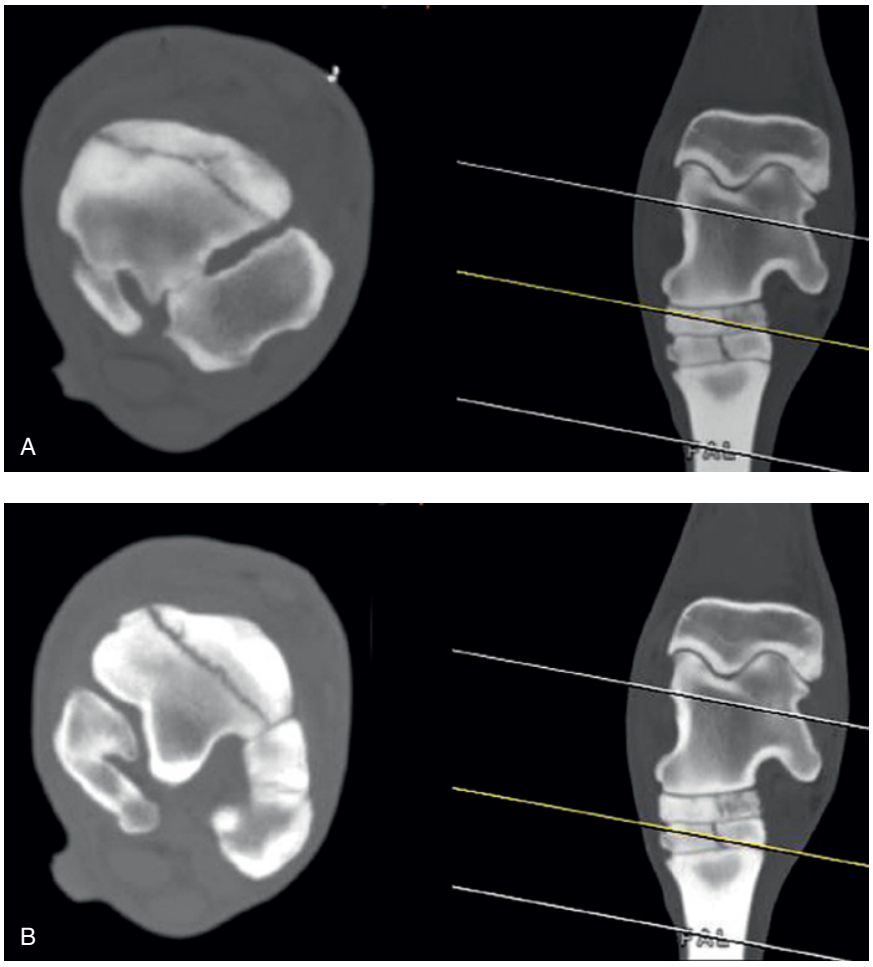


Figure 5.85. Slab fractures that occur in nonperformance breeds are often due to some form of external trauma and are more likely to involve multiple tarsal bones and be comminuted. A comminuted CT bone fracture can be seen in the top CT image (A), and a T3 bone fracture is visible in the bottom CT image (B).

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Treatment of choice is lag screw fixation of the slab fracture(s) of CT and T3 bones. Conservative treatment (prolonged stall rest) has also been utilized successfully with some horses returning to athletic use.[101] However, treatment by screw fixation offers the best prognosis, because conservative management usually results in increasing dorsal displacement of the fracture fragment creating articular incongruity leading to OA. The goal for articular fracture fixation is to promote primary bone healing and maintain and/or restore bone congruity and diminish the chances for the development of OA. The screw placement should be placed as close to perpendicular (90°) to the fracture line as possible and as close to the middle of the fragment (usually in a dorsolateral to plantaromedial orientation). Some comminuted CT fractures are impossible to repair and are best treated conservatively. Horses are rested for 4–6 months after surgery.

Prognosis is dependent on the detection of all of the fracture lines, particularly with CT fractures. If prolonged lameness persists in spite of apparently successful repair, removal of the screw may be necessary. Horses that undergo very strenuous activity will likely do better if treated by lag screw stabilization. Residual lameness can persist after either surgical or conservative management and eventually may require intra-articular medication, chemical or surgical arthrodesis, or screw removal after fracture healing.

Sagittal Fractures of the Talus

Stress fractures associated with high-speed exercise tend to occur at similar locations and configurations and usually without a known specific traumatic event. Racing or competitive speed events can occasionally create exercise-induced sagittal fractures of the talus.[31,69,95] These fractures are rare and most commonly diagnosed in the STB and TB racehorse but occasionally can be encountered in other breeds. The diagnosis is suspected when there is moderate to severe lameness in association with distension of the TC joint. These fractures are often incomplete and nondisplaced and may prove challenging to diagnosis with standard/conventional radiographic techniques. Extra views including a dorsal 10°–20° lateral to plantaromedial oblique and a flexed dorsoplantar (skyline) view can be helpful. Nuclear scintigraphy can be very helpful in confirming the presence of a stress reaction or fracture, usually by revealing an intense focal IRU in the proximal aspect of the talus. Eleven racehorses with a history of chronic, mild hindlimb lameness that became acutely lame during a race were diagnosed with a sagittal fracture of the talus.[31,69] Sagittal fractures of the talus found in other types of horses are thought to be associated with trauma and may be more complicated than what is found in the racehorse. Stall rest until the horse becomes sound, and then 2 months of paddock exercise will normally result in resolution of the problem and a favorable outcome.

Horses with complete sagittal fractures can be treated with lag screw compression of the talus using two 4.5-mm cortical bone screws.[31,69,95] Prognosis may be favorable in horses with simple acute fractures, but the prognosis for horses with comminuted fractures is poor. The use of CT is advantageous and can improve the prognosis by providing accurate fracture assessment and is the best imaging tool to accurately direct screw placement. The medial fragment is usually smaller, and fractures can be repaired with two or three 4.5- or 5.5-mm cortex screws placed in lag fashion from medial to lateral. Prognosis is thought to be favorable if accurate anatomic reduction can be achieved.[69] Comminuted fractures of the talus are often associated with severe soft tissue trauma that can lead to destabilization of the TC joint. These cases are inoperable in most cases.

Fractures of the Trochlear Ridges of the Talus

Significant effusion of the TC joint exists with most fractures of the talus. As with most fractures of the tarsal area, flexion tests are often moderate to severely positive. The clinical signs vary considerably and depend on the size and location of the fragment, the amount of displacement, and the presence of any comminution. The position that the limb was in when the horse was kicked (i.e. flexed or extended) dictates the location of the fracture (point of contact). For example, the caudal aspect of the proximal MTR can be traumatized when the limb is flexed (Figure 5.62B), and the distal aspect of the LTR is exposed when the limb is extended. Fractures to both trochlear ridges are more serious because of the loss of TC joint stability created by the anatomic interdigitation of the trochlear ridges with the cochlea of the distal tibia. Fractures that involve both trochlear ridges can develop during severe twisting movements of the affected limb with the hock partially flexed, which transfers the tibial contact region to the distal areas of the trochlear ridges of the talus. Fractures of the trochlear ridges are not typically difficult to differentiate from OCD fragments particularly of the LTR. Standard radiographic examination of the tarsus should help to confirm the diagnosis but often needs to be supplemented with a flexed lateromedial and flexed lateromedial obliques to highlight the different trochlear ridges.

Conservative therapy in most cases is unrewarding. Surgical treatment is often indicated to alleviate the lameness and persistent effusion. Intra-articular fragmentation of the trochlear ridge(s) warrants arthroscopic removal. Large fragments consisting of the distal one-third to one-half of the LTR may be candidates for anatomic reduction and lag screw compression of the fragment to the talus. This may require recessing the heads of 3.5-mm cortical bone screws or small cannulated screws. Diverging polydioxanone (PDS) resorbable pins may also be useful in these fractures. The plantar aspects of the TC joint should be examined to evaluate the proximal portion of the trochlear ridges for damage and to clean up floating debris. Fragmentation of the plantar trochlear ridges can also occur and usually require removal. The arthroscopic portal is placed on the same side of the joint as the fragment(s) and arthroscopic removal of the fragment(s) via the plantar pouch is performed on the ipsilateral side.

Fractures of the Tibial Malleoli

Traumatic injuries of the tarsus can lead to fragmentation of the CL attachment onto the malleolus (most often the LM of the short lateral collateral ligament

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[sLCL]). These fractures may exist intra- or extrasynovially. Fragmentation that occurs intrasynovially may cause a persistent synovitis, and this may be a primary indication for surgical removal. However, US examination of the affected CL is critical to assess its degree of damage to prevent further injury that may occur on recovery from general anesthesia.

The origin of both medial and lateral CLs are located both on the LM and MM of the tibia, respectively. Fragmentation of the attachment of the sCL is most common although avulsion fractures of the long lateral collateral ligament (lLCL) do occur. Fractures and/or bony change associated with the CLs is best demonstrated on the dorsoplantar radiographic projection. Multiple radiographic projections may be necessary to assess the extent of bony change(s) associated with the injury. Fragment removal should not compromise the stability of the TC joint provided that the long CLs (MCL, LCL) are not affected. The LM appears to be more frequently affected than its medial counterpart; 42 of 44 cases that have been reported in the literature have involved the LM.[75,108,148] This is probably due to the fact that the LM is considered to be the distal end of the fibula and develops as a separate center of ossification that fuses to the distal tibial epiphysis by 1 year of age.

Lateral malleolar fractures are usually associated with a fall or kick (Figure 5.86). A traumatic force from lateral to medial on the limb fractures the LM. Common localizing signs such as TC effusion and periarticular/periligamentous swelling occur soon after injury. Tensile forces applied by CL to the attachments on the malleoli fragments probably contribute to displacement. Fragments can rotate and displace distally. Ultrasonography is a very useful tool to determine the exact location of the fragment(s) and to assess the extent of injury to other structures. For example, a fragment from the LCL is often located within the dorsolateral pouch of the TC joint but may displace into the plantar pouch necessitating a different surgical approach.

Surgical removal of the fragment is frequently recommended. Healing is compromised due to the displacement of the fragment inhibiting primary bone healing. In addition, the intrasynovial position of this fracture fragment perpetuates the shedding of debris into the joint, increasing the chance for widespread cartilage injury and the development of OA. Lateral malleolar fractures are typically smaller, displaced, and comminuted but carry a reasonably good prognosis compared with MM fractures. Two recent reports document that LM fragments can be successfully removed with arthroscopic techniques. In those reports, horses with fractures of the LM had a favorable prognosis for return to full athletic performance following arthroscopy and that arthroscopic fragment removal is the preferred treatment method. Medial malleolar fractures are often larger, are more difficult to remove, and may have lMCL involvement. Surgical removal of LM fragments carries a reasonably good prognosis for attaining soundness.

Large fractures of the tibial malleoli are almost always articular (Figure 5.87). Large fragments with significant CL compromise may result in significant instability if removed. Fractures of the malleoli typically have significant displacement, making primary bone healing unlikely. Large fragments should be repaired rather than removed. Successful repair requires accurate anatomic reconstruction with internal fixation. Lag screw fixation is considered sufficient to stabilize the fracture and counteract the distracting forces applied by the CLs. Fragments of the LM larger than 3 cm are best reattached to the parent bone using 3.5-mm cortical screws placed in lag fashion. Large avulsion fractures of the MM can be repaired with one or two 4.5- or 5.5-mm cortex or cancellous screws placed in lag fashion. A full-limb cast is applied to provide mediolateral stability during recovery from anesthesia and maintained during the initial postoperative period. Nondisplaced fragments

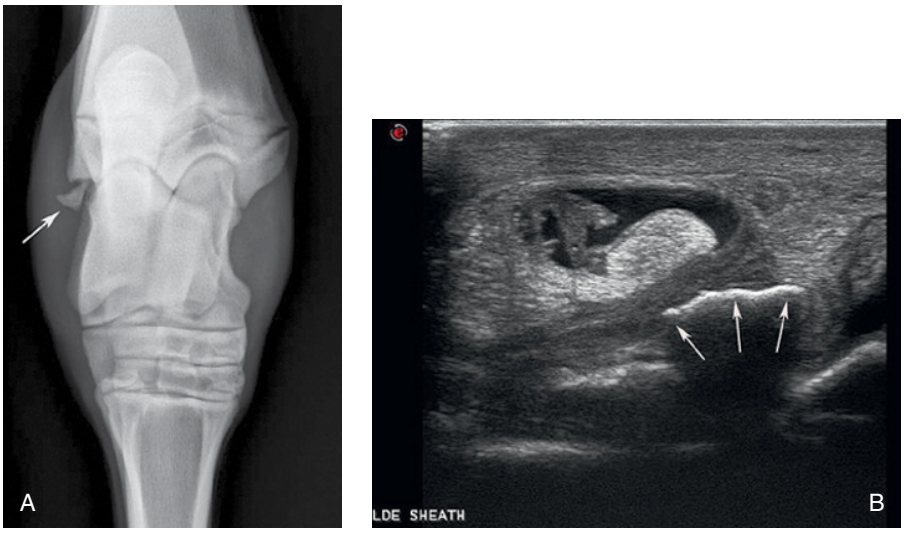


Figure 5.86. Lateral malleolar fractures are usually associated with a fall or kick that creates a traumatic force from lateral to medial on the limb. The DP radiographic projection on the left (A) demonstrates displacement of the fragment due to the pull of the collateral ligament. The ultrasound image (B) demonstrates the fragment (arrows) and effusion within the lateral digital extensor tendon sheath because of their close anatomical position.

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of the malleoli are at risk of displacing over time and, therefore, may also be ideally managed with lag screw fixation.

Fractures of the Calcaneus and Sustentaculum Tali

Calcaneal fractures are almost always the result of some external traumatic event leading to injury to the TC. Kicks that impact a fixed object such as walls, doors, fences, or machinery can injure the calcaneus and often lead to open fractures. Skin lacerations may result from the fracture fragments penetrating the skin from the interior or from the fixed object externally. The clinical signs reflect the severity of the bony derangement and possible secondary complications. Swelling and crepitus can be severe with major fractures. Significant lameness and a lack of Achilles tendon function are the primary indications for surgical repair (Figure 5.88). Diagnosis is made by radiographic examination, which should include a flexed lateral and a flexed skyline projection for complete examination of the calcaneus. Stressed radiographs may help to demonstrate minimally displaced fractures of the calcaneus. Accurate determination of the fracture configuration of the calcaneus is necessary to help provide a prognosis and determine if surgical repair is possible.



Figure 5.87. Oblique radiograph of a horse that had been kicked on the medial aspect of the tarsus. This articular fracture of the medial malleolus was repaired with lag screws. *Source*: Courtesy of Dr. Gary Baxter.

The prominent ST is positioned on the medial aspect of the tarsus. The tarsal canal is formed by the ST of the calcaneus dorsally, the TC laterally, and the flexor retinaculum plantar and medially. The ST is prone to injury from direct trauma. In the event of a fracture of the ST, involvement of the TS and lateral digital flexor tendon (LDFT) should be evaluated.[30,47,70,123,124] Bone damage may be confined to the medial edge of the ST and not affect the TS and LDFT. Larger fractures however can disrupt the fibrocartilaginous gliding surface and can lead to damage of the LDFT and are much more serious. Wounds associated with the traumatic incident may introduce debris and bacteria into several synovial structures such as the TS, the TC joint, or possibly the calcaneal bursa. Any wound in this area should be investigated for possible synovial involvement as synovial sepsis and osteomyelitis of the medial border of the ST may occur. Osteomyelitis, sequestration of fracture fragments, and chronic drainage are potential complications of fractures in this area (Figure 5.89). Fractures on the medial surface of the calcaneus and the ST can be difficult to manage surgically. In cases in which the TS is involved but not infected, the bony fragments may be removed, but the trauma incurred often results in a guarded prognosis.

Tenoscopy allows the exploration of the TS to assess the presence of tendon injury and to explore the recesses of the sheath. Some bony lesions might be outside of the tendon sheath and are best approached by direct cut down and dissection of the fragment. Careful curettage of the fracture bed and flushing of the tendon sheath with copious amounts of fluids is indicated after fragment removal. Conservative management is contraindicated,

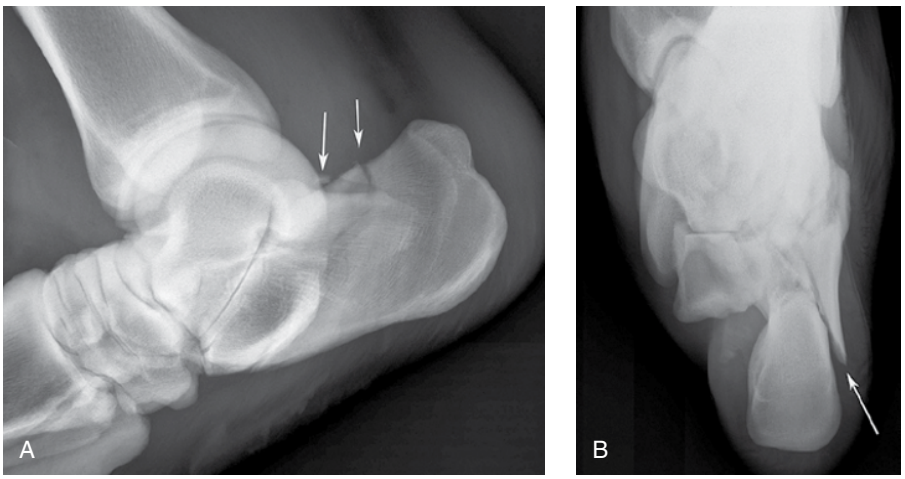


Figure 5.88. These flexed lateromedial (A) and flexed skyline (B) radiographic views demonstrate a comminuted calcaneal fracture in a horse that was hit by a car. Both views were helpful in determining the severity of the injury.

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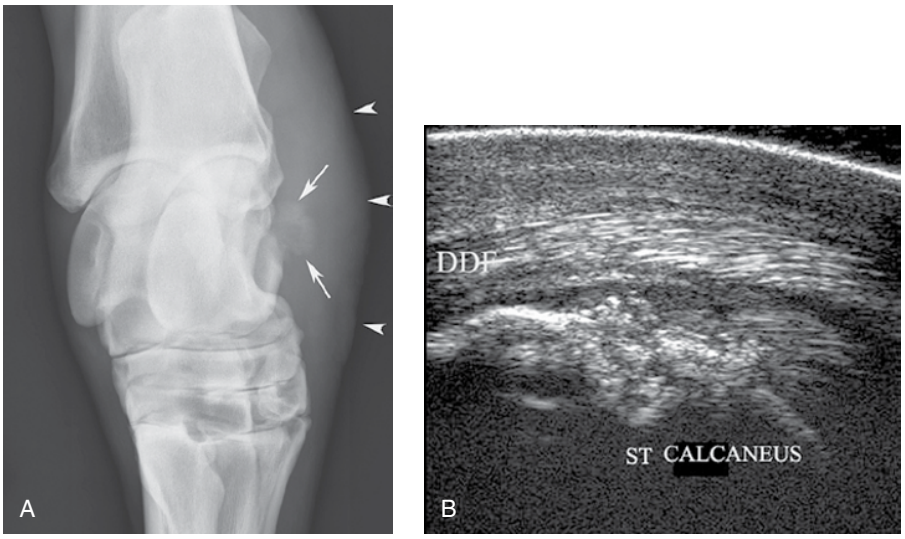


Figure 5.89. Chronic infection of the ST may lead to substantial proliferative bone response and an established infection of the tarsal sheath. (A) Oblique radiograph demonstrating extensive new bone production arrows) and soft tissue swelling (arrowheads). (B) Ultrasound image of tarsal sheath demonstrating moderate bone lysis of the ST associated with osteomyelitis.

especially in open injuries. Involvement of the gliding surface of the ST is a key parameter in the decision for surgery. If the fracture involves a significant amount of the flexor surface, reconstruction and reattachment with cortical screws may be considered, especially if the tendon sheath was not penetrated at the time of injury. If treatment is delayed, proliferative changes can develop, leading to chronic lameness with a poor prognosis for future usefulness of the horse. With immediate, effective treatment, a guarded prognosis for return to athletic is typical. Neglected cases have a grave prognosis.

Fragmentation of the Proximal Tubercle of the Talus (PTT)

The proximal tubercle of the talus (PTT) is situated in the plantar aspect of the tarsus on the proximal and medial region of the talus. The anatomy of this region of the tarsus is complex with a variety of ligaments and synovial attachments associated with the PTT. Fragmentation of the PTT in horses is rare, and its clinical significance is questionable. The etiology of fragmentation of PTT is unknown, but OC has been suggested to have a role. The dorsal 45° medial to plantarolateral oblique view or lateromedial view is reported to demonstrate the fragment best. A flexed lateromedial, flexed dorsoplantar, and plantaroproximalplantarodistal oblique (calcaneal skyline) views may also help identify the fragment.

In one report 9 horses with fragmentation of PTT were identified, seven of which were Warmbloods.[50] Diagnosis was made based on radiographic findings, but ultrasonography and CT were also utilized in some of the horses. Less than half (3/9) had TC joint effusion, and only one horse was lame and that horse had a history of prior trauma and resultant lateral tibial malleolus fracture. Flexion tests did not produce lameness. One horse underwent arthroscopy, but the fragments were not visible and it was presumed that the fragments were extra-articular.

Conclusions of that study were that fragmentation of PTT appeared to occur more frequently in warmbloods but more importantly that it was not usually associated with lameness. Affected horses remained clinically and radiographically stable over time. The information from this report is important for practitioners who perform radiographic screenings during prepurchase examinations. Finding fragmentation of PTT does not appear to be a risk factor for the development of lameness associated with the tarsus (Figure 5.90).

SOFT TISSUE INJURIES OF THE TARSUS

Tarsal Collateral Ligament Injury

The distal tibia is quadrangular in form and larger medially than laterally. These grooves and the corresponding trochlear ridges of the talus are directed obliquely forward and laterally and are bound on either side by the tibial malleoli. The tibial malleoli have extensive CL attachments from both the long and short components, and their anatomy has been previously described in detail.[143] The lLCL has distal attachments to the talus, calcaneus, T4, T3, and MT4 bones. There are two sLCLs that lie axial to the lLCL. The long medial collateral ligament (lMCL) has distal attachments to the fused first and second tarsal bones, talus, CT and T3 bones, and MT2 and MT3 bones. There are three short medial collateral ligaments (sMCL) that, like their lateral counterparts, lie axial to the MCL and are named according to their respective insertions.

The stability of a TC joint is a product of the congruency of the joint surfaces, the integrity of the joint capsule, and the strength of the CLs. In general, CLs typically have a uniform fascicular orientation and, therefore, a homogeneous sonographic appearance. The lLCL spirals during flexion and straightens during extension as it takes the load from the sLCLs. A mixed arrangement of fascicles is necessary to allow the CLs to function in both extension and flexion.[34,35] In the TC

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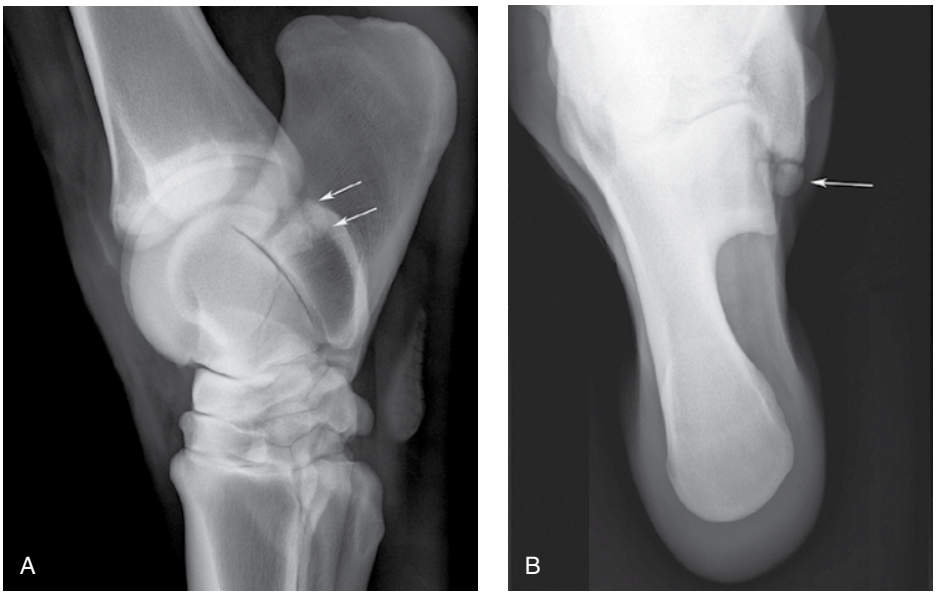


Figure 5.90. Lateral (A) and skyline (B) views demonstrating a fragmentation of PTT lesion (arrows). This fragmentation does not appear to be a risk factor for the development of lameness but must be differentiated from OC that develops on the plantar trochlear ridges.

joint, the superficial CLs are under tension while the limb is extended and relaxed while the limb is flexed, whereas the short CLs are tensed only when the tarsus is flexed. Therefore, the short CLs should be examined while the leg is flexed.

CL injury of the TC joint most often occurs due to some form of traumatic injury.[15,131] Lameness varies from mild to severe, depending on the degree of damage. Hindlimb proximal limb flexion significantly worsens the lameness. Physical exam findings usually include tarsal swelling with edema and/or fibrosis localized over the medial CL (MCL) or lateral CL (LCL) region. The presence of TC effusion often occurs but the amount can be quite variable. Desmitis associated with the enthesis of the CLs is often performance limiting. The diagnosis is usually made with a combination of radiographic and ultrasonographic examinations. Stress radiographic examination may occasionally be necessary to more effectively demonstrate instability associated with the MCL and/or LCL. The initial radiographic exam of acute injuries often only reveals soft tissue swelling, but by 6–8 weeks after the injury enthesophytes may become evident along the origin and attachment sites of the CLs. Fragmentation of the malleolar attachment of the CL attachment (most often the LM of the sLCL) can occasionally occur.

Enthesopathy of the lLCL and less commonly the sLCL has been reported in STB racehorse.[15] Pacers were found to be more at risk of this condition than trotters. Distension of the TC joint capsule and subtle, localized soft tissue swelling were present, and lameness improved with analgesia of the TC joint. Scintigraphy was also utilized to show focal, intense IRU on the lateral aspect of the calcaneus, and enthesopathy of the CL was detected radiographically. The authors of that study theorized that cyclic stress associated with the speed of racing was responsible for the injury. Local injections with MPA and sarapin resulted in improvement in the lameness.

Ultrasonographic evaluation can be quite helpful to identify CL injury and define the type and severity of the injury. The ultrasonographic appearance of the CLs should always be compared with the opposite CL, and this should always be used as a reference for normal. Injury can change the size, shape, linearity, and architecture (appearance) of these structures. Serial US examinations are critical to monitor the repair process and to evaluate healing. The malleolar insertion of the CLs should be carefully evaluated in longitudinal view as changes can often at this location. Chronic injury may be recognized as calcification (enthesis new bone) within the insertion of the CL. Insertional change can also occur in the short CL attachment onto the talus. Treatment for the disease is restriction of exercise for 2–3 months. A gradual reintroduction to exercise is predicated on the improving sonographic appearance. The prognosis is favorable for resolution after a period of rest.

Desmitis of the CLs, particularly the long CL, has been described.[34,35,131] Injury to the CLs are thought to occur with twisting and torqueing of the tarsus while the lower limb is fixed to the ground. Increasing tensile stress (and probably the rate at which it is loaded) within the CLs may lead to fractures of the malleoli. High loads are felt to develop within the sMCL/sLCL when the horse falls on the lower limb while it is flexed. Initial treatment involves stall rest, cold water therapy, topical treatment (diclofenac or dimethyl sulfoxide with or without corticosteroids), and systemic NSAIDs. Rehabilitation will require increasing periods of controlled exercise determined by an improving ultrasonographic appearance of the injured structure. Prognosis depends on the severity of injury and effective treatment and rehabilitation. Horses with moderate injuries (and develop extensive periosteal new bone) have been able to return to full athletic function, although some residual enlargement of the hock has persisted.

Injuries to the CLs that exist within the intrasynovial space of the TC joint may be difficult to accurately

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assess with standard imaging techniques. These types of injuries usually present with moderate effusion of the TC joint, and confirmation of the structure(s) involved may require arthroscopic exploration. Arthroscopy permits direct visualization and debridement of torn fibers of the sCL. In a recent report of 20 horses with intrasynovial CL, all injuries involved the sCL with four also involving the lCL.[5] Medial sCLs were torn in 14 cases and lateral sCLs in 11 (including five cases with medial and lateral tears). No arthroscopic evidence of joint instability was found when the tarsus was manipulated in any case. All horses recovered uneventfully from general anesthesia, and there were no significant postoperative complications.

Nonseptic Tenosynovitis of the Tarsal Sheath

In the normal horse the TS is not often apparent on clinical or ultrasonographic examination of the plantar aspect of the tarsus.[34,35,116,146] Thoroughpin is a morphologic description of swellings of the TS that occur from a variety of problems and with varying degrees of inflammation (Figure 5.91). Often the causes of effusion within the TS are unknown and called idiopathic as nothing is found on radiographic and ultrasonographic examination. Many of these cases can be reclassified if endoscopy of the sheath is performed because LDFT tendinitis or injuries to the ST are often found as the cause. True idiopathic tenosynovitis of the TS can occur and is often bilateral and most likely related to limb conformation.

Clinically, moderate effusion of the TS typically presents as two swellings cranial to the calcaneal tendon on both the medial and lateral aspects of the distal crus. In more pronounced effusions of the TS, a swelling may be seen distal to the tarsus on the medial aspect of the DT joints just proximal to the chestnut. Distension of the TS must be distinguished from TC joint distension. If the TC joint is distended, fluid can be moved from the plantar synovial pouches to the dorsal pouches by manual compression. Unilateral benign distension associated with idiopathic tenosynovitis often decreases with exercise and worsens with shipping or stall confinement. Effusion will often resolve spontaneously. Treatment is unnecessary if no lameness is present. Horses with effusion combined with lameness and/or a positive response to tarsal flexion most likely have an underlying cause. An accurate diagnosis and directed treatment provide the best chance for a return to soundness.

Intrathecal analgesia of the TS can be performed to confirm the TS as a source of lameness particularly if nothing is found with diagnostic imaging. However, intrasynovial diagnostic analgesia of the TS is not often performed and does have limitations. It is not uncommon for analgesia of the TS to manifest significant improvement but not alleviate lameness. However, more improvement is often seen following a tibial and peroneal nerve block. Intrathecal analgesia of the TS can be difficult when there is minimal effusion. A good landmark for injection is a site dorsal to the LDFT, 2 mm plantar to the middle of the palpable plantar edge of the flexor groove of the ST. However, without recovery of synovial fluid, it can be difficult to determine if the needle is actually in the TS. Therefore, synoviocentesis of the TS should be performed either directly with US guidance or after seeing fluid pockets with US examination in one of the several recesses of the sheath.

A 10- to 14-MHz sector-scanner transducer with a standoff pad at a display depth of 5–6 cm should be used to evaluate the character of the synovial fluid and detect fibrin deposition, adhesion development, and lesions within the LDFT. This technique can be used to assess the integrity of the fibrocartilaginous groove, flexor retinaculum, and edge of the ST. The course of the LDFT

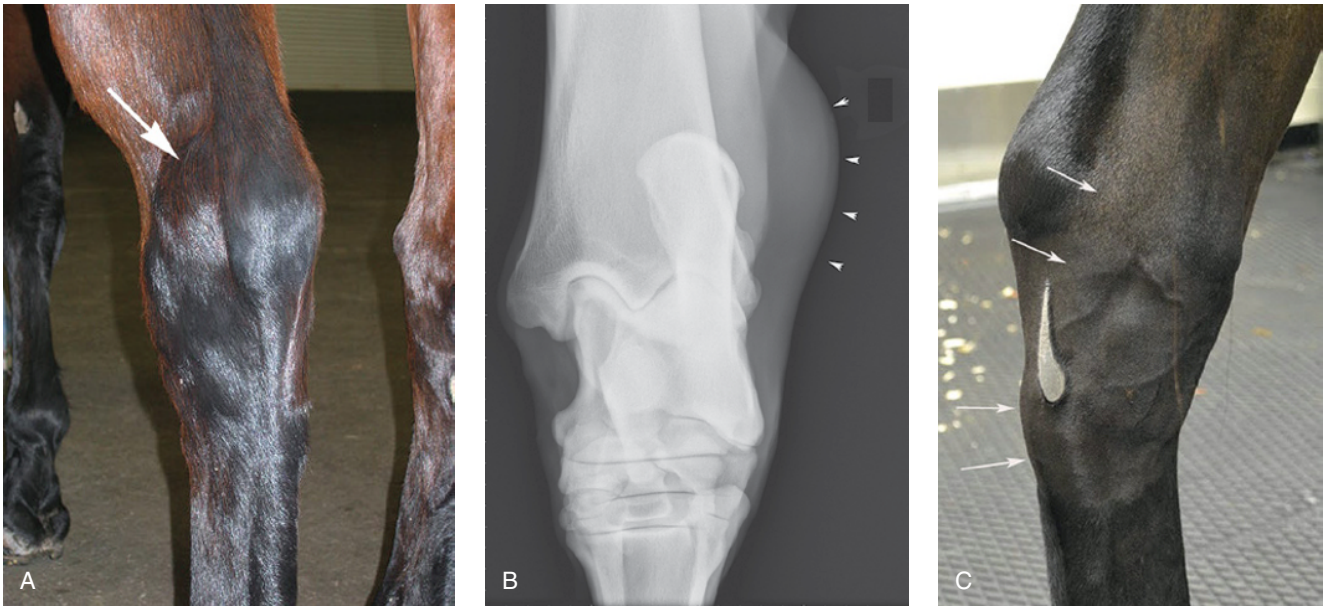


Figure 5.91. Clinical (A; arrows) and DP radiographic observations (B; arrows) of a horse with thoroughpin presenting with swelling primarily on the lateral aspect of the tarsus. Thoroughpin may also be observed on the medial aspect of the tarsus (C; arrows).

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around the ST requires constant manipulation of the scan head for proper evaluation of the architecture throughout its course on the medial aspect of the limb. The chestnut can interfere with scan head footprint limiting interpretation of the LDFT at this site. Longitudinal tears in the LDFT and superficial fraying of the tendon are the most common types of lesions detected via ultrasonography and should not be confused with proliferative synovial hyperplasia of the TS. The LDFT may be examined from the plantaromedial aspect of the tarsus and is a large oval-shaped structure with well-defined margins. The dorsolateral aspect of the tendon may appear slightly less echogenic than the remainder of the tendon, and care should be taken not to misinterpret this as a lesion. Sonographic examination infrequently visualizes associated tendon injury. Primary lesions of the LDFT in the tarsal region are rare but do occur in mature horses.[32]

In addition to the standard radiographic views of the tarsus, the skyline and flexed lateromedial projections should be included to emphasize the ST and plantar regions of the tarsus. Acute injuries such as fracture(s) of the ST may be identified. More chronic diseases create destructive and/or proliferative changes to the ST and may appear as mineralization of the LDFT, the long plantar ligament (LPL), and parts of the TS. Contrast radiography/tenography can be useful to evaluate the position and shape of the TS and allow some delineation of the LDFT, but this technique is rarely utilized. Contrast radiography/tenography or contrast fistulography may help to identify foreign bodies.

TS effusion can occur due to a variety of soft tissue and bony injuries. Acute TS effusion usually occurs unilaterally, with sudden onset of moderate to severe lameness. Acute tenosynovitis of the TS is most often due to a traumatic insult to the medial aspect of the tarsus. The medial edge of the ST is a prominent structure on the medial aspect of the tarsus and is prone to fracture from direct trauma. Overstretching of the sheath may cause aseptic tenosynovitis of the TS. Intrathecal hemorrhage from synovial trauma induces marked inflammatory response, and it can lead to formation of restrictive intrasynovial adhesions. Lameness that develops in these animals may be due to the formation of intrathecal adhesions. In the event of a fracture of the ST, the LDFT may be unaffected if bone damage is confined to the medial edge of the ST, but larger fractures can disrupt the fibrocartilaginous gliding surface.

Horses with lameness thought to be associated with chronic unilateral TS effusion should have thoroughly investigated with US and radiography. Common findings visible on US include synovial proliferation, intrathecal hemorrhage, adhesions, fibrous masses, and mineralization/calcification within the TS wall. US examination may demonstrate fibrillation of the LDFT, or more severe architectural changes include enlargement and/or hypoechoic areas consistent with tendon injury. Mineralization in the LDFT and the surface of the ST does occasionally occur. Radiographically, new bone proliferation can be seen in the region originating from the original trauma or possibly in response to chronic tendon sheath distension. Mineralization may also occur within the LDFT and is felt to be a poor prognostic indicator.

Treatment of acute tenosynovitis should include rest, NSAID therapy, and cold therapy. Intrathecal injection of hyaluronan and short-acting corticosteroid should help resolve acute synovitis, but corticosteroids should be used with caution if tendon damage is found or suspected. If inflammation and lameness continue (especially in the event that no structural damage can be found) but particularly if bone or tendon damage is documented, tenoscopy of the TS is indicated.

Accurate treatment of these chronic problems involves tenoscopic exploration to assess the specific pathology that exists.[26,47,123,124,145,147] Lesions of the TS and LDFT and fragmentation of the ST are visible with tenoscopy and can be addressed directly with this technique. Debridement of the damaged tendon is most effective when the tendon injury is peripheral and associated with tendon fiber fraying. Distension of the sheath is often resolved when the tendon is appropriately debrided. Synovial masses and fibrous adhesions can be identified and resected. Fragmentation of the ST is best managed with fragment removal and fracture site debridement via tenoscopic technique. If the fragments are outside the retinaculum on the medial aspect of the ST, they can be removed by making a separate extrasynovial incision directly over the fragment.

The prognosis of horses with TS tenosynovitis is variable and quite dependent on the cause. Horses with idiopathic synovitis have a very good prognosis except for the cosmetic blemish. Horses with small fractures of the ST can do well, provided damage to the LDFT is minimal. However, chronic changes associated with fracture of the ST have been associated with a poor prognosis for return to athletic soundness.[47]

Septic Tenosynovitis of the Tarsal Sheath

Lacerations and puncture wounds on the medial aspect of the tarsus can be highly problematic. Soft tissue swelling often accompanies these injuries, making an early accurate diagnosis difficult. Lameness is often quite severe particularly when the puncture wounds heal over quickly. The size, depth, and direction of the wound can affect many vital structures. The potential for the involvement of any of several synovial structures dictates that a careful evaluation be performed. Contaminates such as hair and/or debris may be driven deep into the wound providing a nidus for infection. Wounds that have bony involvement (fragmentation) of the tarsal groove of the ST can cause severe lameness due to the motion of the LDFT against the roughened bone even without infection (Figure 5.92). In addition the tarsal retinaculum can act as a constricting band when the LDFT or TS is inflamed and swollen. Other bony abnormalities such as osteomyelitis or sequestra of the ST or calcaneus can also be associated with wounds to the tarsus and can further complicate treatment.

Clinical pathological analysis of synovial fluid and diagnostic imaging are necessary to determine what structures are affected and the necessary treatment(s). Early diagnosis and aggressive treatment are critical for a successful outcome. Septic synovitis is best treated with a combination of systemic antimicrobial therapy, joint lavage, and/or regional intravenous perfusion with

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antimicrobials.[9] Chronic cases of TS sepsis are candidates for tenoscopic evaluation. Surgical intervention in such cases may involve incision into the sheath to allow drainage of fluid, placement of indwelling drains, TS tenoscopy, and transection of the tarsal flexor retinaculum. Bony abnormalities involving the ST or tuber calcanei should be treated as the primary source of infection and addressed surgically. The prognosis for return to athletic function is frequently guarded and often requires a protracted course of treatment. Further information on treating synovial sepsis can be found in Chapter 12.

LDFT Tendinitis

Primary injury to the LDFT is a rarely reported cause of hindlimb lameness in performance horses but has recently been reported in 4 horses that had MRI examination of the tarsal region (Figure 5.93).[32] Lameness was mild/moderate in severity and insidious in onset in all horses. Responses to flexion tests were variable. TS effusion was slight in three horses and mild/moderate in one horse. Two horses were positive to DBLPN block, but all horses showed improvement (70%–90%) in lameness after TS analgesia (US-guided injection). Radiographic, scintigraphic, and ultrasonographic findings were

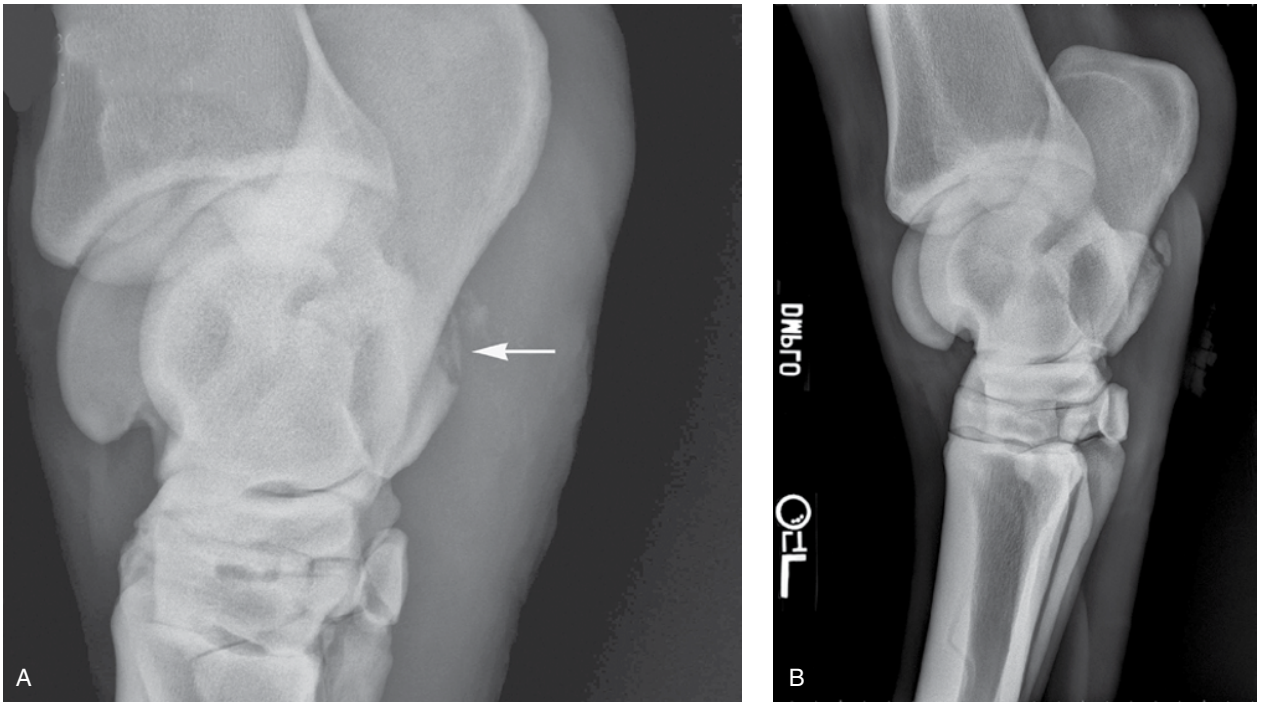


Figure 5.92. (A) Oblique radiographs of two different horses demonstrating an acute, open (A) and chronic, closed (B) fracture of the sustentaculum tali (arrows).

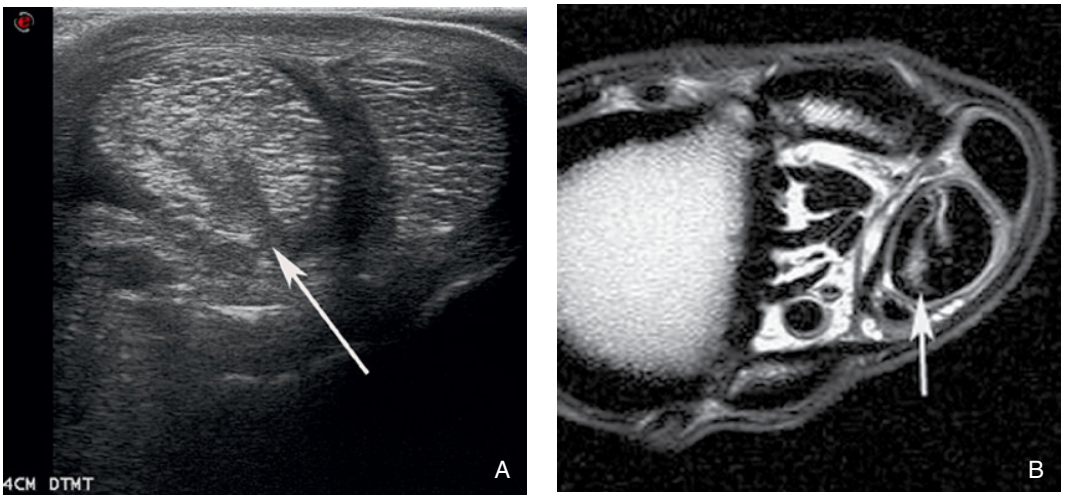


Figure 5.93. Ultrasound (A) and MR image (B) of an LDFT injury (arrows) that occurred within the tarsal sheath at the level of the distal tarsal region. These lesions can be difficult to detect with ultrasound alone.

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inconclusive. MRI characteristics included increased T2, PD, and STIR signal intensity within the LDFT in the area of the TS. Ultrasonography was able to distinguish LDFT injury when examined with the knowledge of the MR findings. This study supports the use of MRI for diagnosing LDFT within the TS in horses. Affected horses appear to have a good prognosis for return to athletic performance following appropriate medical treatment.

Calcaneal Bursitis/Capped Hock

The anatomy and nomenclature of the calcaneal bursae (CB) is complicated and can be confusing. The CB includes the subcutaneous calcaneal bursae (SCB), the intertendinous calcaneal bursae (ICB), and the gastrocnemius calcaneal bursae (GCB).[113] The SCB is a potential bursa between the subcutaneous tissue and the superficial digital flexor tendon (SDFT) at the point of the TC. The SCB and ICB have also been called the calcaneal bursa of the SDFT. The intertendinous calcaneal bursa (ICB) is further divided into the gastrocnemius calcaneal bursa (GCB) and the intertendinous calcaneal bursa (ITB). However, these two bursae communicate in most horses and should be considered as a single synovial structure. The CB extends approximately 9–10 cm proximally and 6–7 cm distally to the TC. The ICB is a true synovial cavity analogous to the bicipital bursa and navicular bursa, and problems within this anatomic structure are much more problematic than those within the subcutaneous bursa. However, the ICB and the GCB appeared to communicate with the SCB in 39% of limbs.

Swelling at the point of the hock (tuber calcaneus or TC) is usually caused by damage to the SCB, giving the appearance of a capped hock (Figure 5.94). This is usually caused by self-inflicted trauma, typically from the horse's kicking a fixed object (wall or trailer). These injuries may or may not be associated with a wound. The SCB at the TC is analogous to the acquired bursa at the point of the olecranon. Because there is a communication between the SCB and the ITB in about 40% of horses', wounds that involve point of the hock down to and including the SDFT should be considered at risk of developing an infection of the ICB. Sonographic examination typically reveals the subcutaneous position of the bursa and, in the early stages, a primarily fluid-filled pocket. The fluid may have fibrin or the appearance of a large clot within the cavity. Careful evaluation of the ITB is critical to rule out more extensive involvement and appropriate treatment of all affected structures. Radiographic examination should be performed to evaluate bony lesions.

A capped hock appearance has been reported in cases of gastrocnemius tendinitis and in some bony abnormalities of the TC[41,109] (Figure 5.95). Lameness and swelling in the region of the distal common calcaneal tendon are most commonly due to a tendonitis of the gastrocnemius tendon (GT). A mild to moderate enlargement of the ICB and a "capped" appearance can be present possibly attributable to distension of the bursa dorsal to the insertion of the GT that may communicate with the ICB. LPL desmitis or curb should also be differentiated from swelling of the ICB and GT (see gastrocnemius tendinitis later in this section for further information).

Horses with signs of chronic swelling of the calcaneal bursa should be evaluated for osteolytic lesions on the tuber calcanei associated with the insertion of the GT ("gastrocnemius enthesitis"). A flexed, proximoplantar to distoplantar tangential (skyline) radiographic view of the calcaneus is advantageous for identifying this lesion and is recommended for all horses with calcaneal bursitis. The prognosis for athletic soundness in horses with chronic calcaneal bursitis and associated osteolytic lesions of the tuber calcanei should be considered guarded. Well-circumscribed osteolytic or osseous cyst-like lesion(s) on the TC at the insertion of the GT may be evidence of an enthesopathy or enthesitis and calcaneal osteitis (Figure 5.96). A US exam may reveal mild thickening of the wall of the calcaneal bursa, often distended with anechoic fluid. The GT, SDFT, LDFT, and TS should all be evaluated with US.

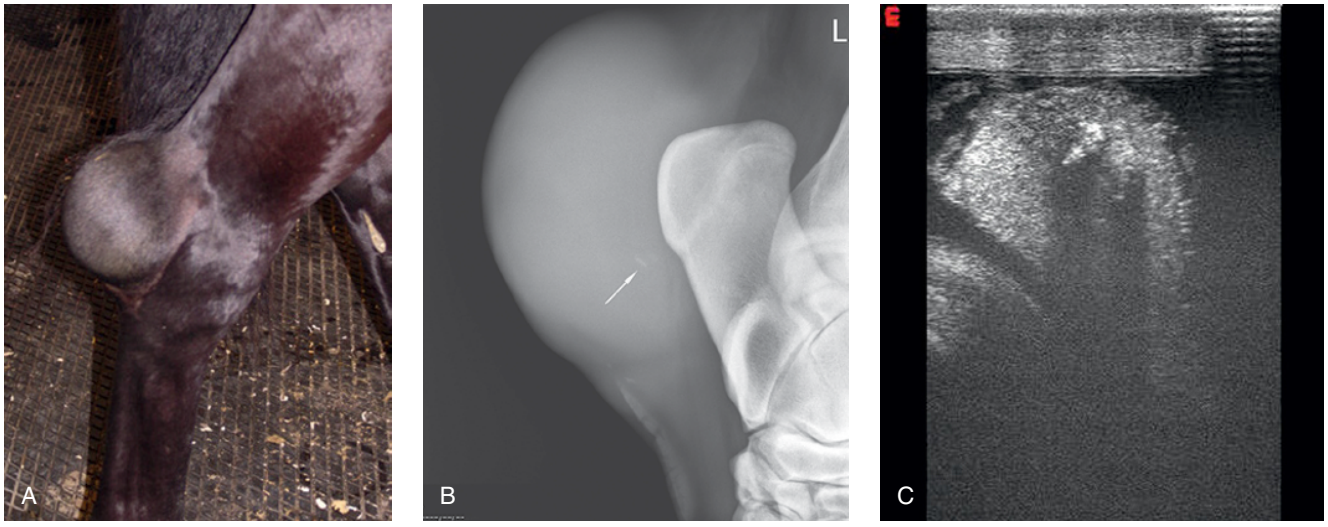


Figure 5.94. Clinical (A) and radiographic (B) appearance of a horse with swelling at the point of the hock (capped hock). Ultrasound (C) revealed a radiodense object within the swelling that can also be seen on the lateral radiograph (arrow).

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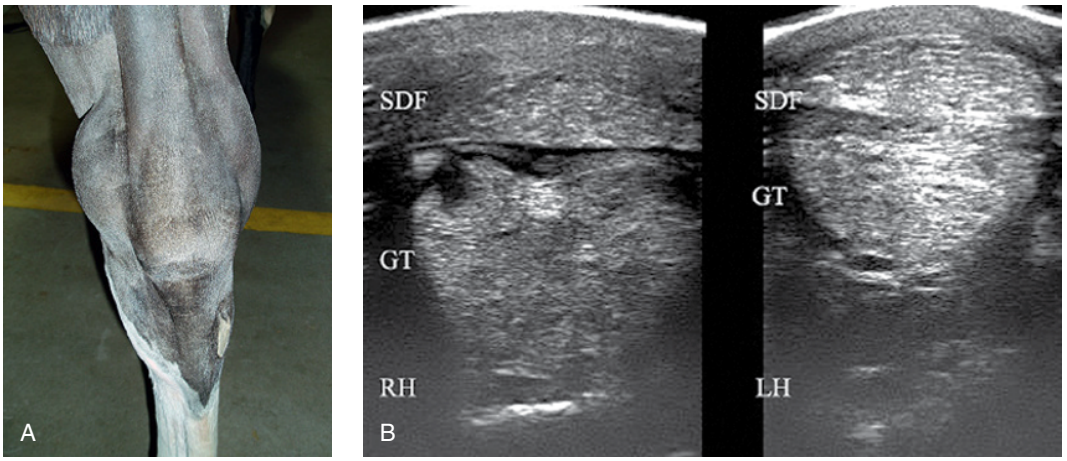


Figure 5.95. Severe swelling in the region of the calcaneal tendon has been reported in cases of gastrocnemius tendinitis and in some bony abnormalities of the TC (A). Ultrasound can be used to confirm the diagnosis (B). Comparison to the opposite side can help confirm changes associated with injury to the calcaneal tendon.

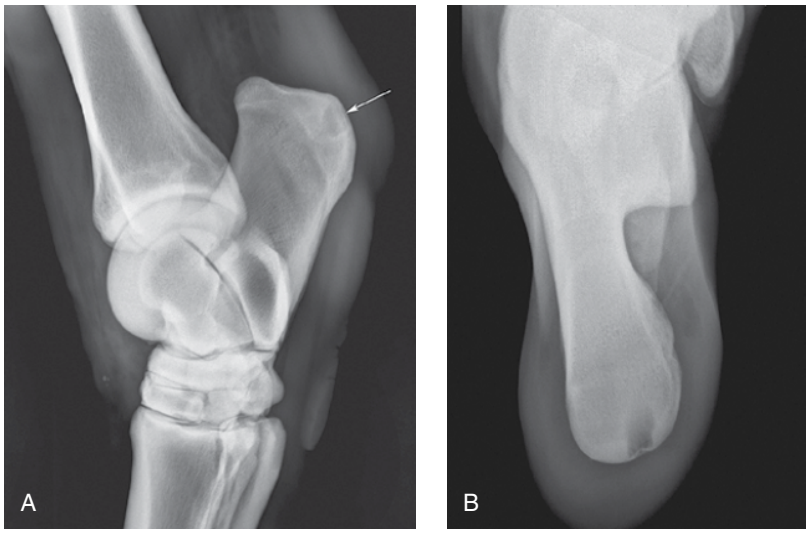


Figure 5.96. Well-circumscribed osteolytic or osseous cyst-like lesion (arrow) on the TC at the insertion of the GT may be indicative of an enthesopathy and calcaneal osteitis (A). A flexed, proximoplantar to distoplantar tangential (skyline) radiographic projection can often help identify bony lesion(s) of the TC (B).

Wounds that involve the plantar surface of the TC need to be carefully evaluated as communication with the SCB can increase the risk of extension of the septic process into the ICB. Wounds superficial to the subcutaneous bursa can often resolve with routine wound care and have a good prognosis. Wounds that directly involve the ICB can be more problematic and should be treated aggressively to prevent an established synovial infection. Radiographs are recommended to document sequestrum formation or osteomyelitis, which may be the primary problem, with the bursa secondarily infected. Contamination of the CB must always be considered to be possible with wounds in the plantar tarsal region.

Endoscopic exploration (bursoscopy) of the ICB can be a very useful diagnostic tool to help identify and treat the cause(s) of ICB bursitis.[74,112,113] For example, peripheral tendon lesions of the GT or SDFT can occasionally only be identified and treated effectively with endoscopy. Endoscopy can also be used in cases of infectious bursitis.

Osteitis of the Calcaneus

The TC has minimal soft tissue protection and is at risk of traumatic injury and subsequent infection.[8,70,87] Management of these injuries can be complicated if the TC joint, ICB, and TS are involved. Traumatic injuries generally occur from kicking a fixed object or being kicked by another horse. Calcaneal involvement can lead to a focal osteitis, which often causes moderate to severe lameness, cellulitis, and synovitis. The injury often compromises the vascular supply to the periosteum resulting in cortical bone damage that often leads to sequestrum formation. Bone infection of the tuber calcaneus rarely extends into the medullary cavity so that an accurate description of the condition is an osteitis rather than osteomyelitis.

Lateromedial, flexed lateromedial, and plantaroproximalplantarodistal (skyline) radiographic views are the most helpful (Figures 5.62A and 5.96B). Radiographic

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signs of calcaneal osteitis or sequestrum formation may take several days or weeks to appear, and thus sequential radiographic evaluation is recommended. The radiographic appearance of calcaneal osteitis depends on the duration of infection before treatment and if other structures are involved. Ultrasonography can be useful to assess synovial structure involvement and provide direct visualization of needle placement into a pocket of fluid for the aspiration of synovial fluid for analysis.

Acute injuries to the TC with minimal contamination may resolve with local wound management without the development of bony lesions. Osteitis of the TC is best addressed by curettage of the affected bone. Sequestrectomy is indicated if a sequestrum develops. Secondary sepsis of the ICB or TS should be treated aggressively similar to any synovial infection. More complex surgical intervention may be required in more traumatic injuries with heavy contamination and devitalized and infected soft tissue and/or bone. The prognosis for horses with calcaneal osteitis depends on the structures involved and the duration of infection before treatment. In one study, 9 of 18 horses with infection of the TC were used as broodmares, and 9 horses returned to athletic function.[87]

Dislocation of the Superficial Digital Flexor Tendon (SDFT)

The SDFT contains very little muscular tissue and consists almost entirely of a strong tendon forming part of the stay apparatus in the hindlimb. Its action is to flex the digit and extend the hock, but this is considered largely to be a mechanical effect resulting from the action of other muscles of the stifle joint. A wide, flat, fibrous bundle arising from the SDFT attaches the tendon to the calcaneus laterally and medially. This structure, called the retinaculum, stabilizes the tendon at that level of the TC. The dislocation occurs when one of the fascial attachments (usually the medial retinaculum) of the SDFT to the calcaneus ruptures with displacement laterally (Figure 5.97).[118,127,128,135,149]



Figure 5.97. Caudal view of the tarsi in a horse with severe swelling within the right calcaneal bursa and evidence of lateral luxation of the right SDFT from the tuber calcanei (arrows). *Source*: Courtesy of Gary Baxter.

Because of the severe swelling over the point of the hock, an inaccurate diagnosis of capped hock or calcaneal bursitis may be made. The tendon may remain completely luxated (stable luxation) or can reduce and then displace while walking (unstable luxation). Most common is a tearing of the medial retinaculum with a lateral displacement, but medial displacement does rarely occur. One case report described a situation where the tendon fibers of the SDFT split near its center over the point of the hock (one-half of the SDFT displacing medially and the remaining half laterally).[127]

Luxation of the SDFT tends to be an injury that occurs in horses that compete at speed or due to trauma. Initially, affected horses appear quite uncomfortable after the injury. In the acute to subacute injury, diffuse swelling around the point of the hock can make it difficult to accurately define what is injured. However, an unstable luxation of the SDFT tendon allows the tendon to move back into its original position only to luxate again when the horse walks off. Typically, the tendon tends to luxate when the tarsus is flexed. The position of the tendon can become more easily appreciated as the swelling subsides. On palpation, the luxation of the SDFT can often be appreciated. Horses with actively luxating tendons may become quite distressed. If the tendon remains permanently luxated, the horse's distress diminishes quickly. Horses with a permanently luxated tendon (usually laterally) can, with conservative management, return to useful work. By contrast, those with unstable subluxations, in which the tendon spontaneously reduces and then displaces, often remain permanently compromised frequently with continued anxiety. There have been rare cases of horses that have had the SDFT luxate bilaterally.

A definitive diagnosis is usually made by visualizing the displacement of the tendon. However, radiographs and US examination should be performed to more accurately define the extent of injury. Radiographs are taken to rule out the possibility of a fracture or other associated bony disorders of this area. US can assist in the diagnosis by defining the structures that are involved and the degree of damage. Most often the medial retinaculum is torn completely. However, partial tears of the retinaculum exist with subluxation of the SDFT occurring rather than complete luxation. Because the retinaculum exists within the ICB, fraying and tears of this structure can create moderate effusion of the calcaneal bursa. Partial tears could easily be misdiagnosed as a simple capped hock.

The literature would suggest that the treatment of choice for luxated tendons (unstable displacements) is surgical correction with postoperative immobilization in a cast for 4–6 weeks. There have been a number of surgical procedures recommended, all of which include open reduction with some form of suture of the torn retinaculum, some of which had a retinacular reinforcement with or without imbrication and some using a synthetic (polypropylene) mesh. There are only a few reports of reduction representing a total of nine documented cases, but the results have often been disappointing. Recovery in a full-limb cast and the possibility of cast complications are always a concern.

Endoscopy of the CB may provide another alternative approach to luxation of the SDFT.[148] Exploratory

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endoscopy of the bursa allows surgical debridement of the torn tissue and exploration of the calcaneal bursa for any other damage. Horses with unstable displacement of the tendon also have disruption of the tendon fibrocartilage cap. Removal of this tissue (torn retinaculum and the damaged fibrocartilage cap) results in stable subluxation and can return horses to athletic activity. Both lesions can be detected with preoperative ultrasonography.[149]

With conservative management the prognosis for breeding soundness or light pleasure riding is usually good for stable luxations. Realistically, too few cases have been treated surgically with long-term follow-up to make an objective comment about the prognosis for athletic ability, because every case is different.

Gastrocnemius Tendinitis

The SDFT begins medial to the GT and rotates/courses from medial to caudal to attach to the calcaneus before continuing distally. The LDFT is deep to both the SDFT and GT and courses distally over the plantaromedial aspect of the calcaneus and the ST. The combined SDFT and GT form the common calcaneal tendon, which is the major extensor of the tarsus. Severe injuries to the common calcaneal tendon can cause partial or complete loss of support to the tarsus. Lameness and swelling in the region of the distal common calcaneal tendon are most commonly due to tendonitis of the GT, which is considered a rare cause of hindlimb lameness in the horse.[41,114] Lameness may be sudden or gradual in onset, and the severity of lameness varies depending on the injury. Lameness is usually exacerbated by a proximal limb flexion test. Lameness is usually improved by perineural analgesia of the tibial nerve.

In association with enlargement of the GT, there can be a mild to moderate enlargement of the calcaneal bursa (ICB and CB) (Figure 5.98). This can give the limb a "capped" appearance possibly attributable to distension of the bursa dorsal to the insertion of the GT that may communicate with the ICB. Ultrasonography may be used to best detect enlargement of the tendon, poorly defined margins, focal or diffuse anechoic to hypoechoic lesions, and loss of normal fiber alignment (diffuse > focal). The lesions seem to occur predominantly in the body of the tendon and less frequently at the insertion on the TC. In the majority of horses, however, GT occurs without concurrent tendinous or ligamentous injury. Severe injuries may allow the point of the hock to "drop" because the reciprocal apparatus is no longer functional.[76,85,142] Radiographs should be obtained for the detection of a fracture of the calcaneus that can manifest a similar appearance. Diagnostic US can help confirm the location of the injury but is not necessary for an accurate diagnosis. Conservative treatment with stall rest and controlled exercise for 6–12 month is usually indicated.[85,142] Rehabilitation protocols can include water treadmill exercise. Horses with mild to moderate lesions have a reasonable prognosis for return to athletic work, but the prognosis for horses with more severe lesions is guarded.

Horses with chronic calcaneal bursitis often have insertional changes of the GT. Chronic calcaneal tendinitis may have osteolytic lesions at the insertion of the GT on the TC ("gastrocnemius enthesitis"). Well-circumscribed osteolytic or osseous cyst-like lesion(s) on the TC at the insertion of the GT may be associated with enthesopathy or a calcaneal osteitis as a component of focal nonseptic osteitis. A radiographic examination of the tarsus should include a flexed, proximoplantar to distoplantar tangential (skyline) view, and a flexed lateral radiographic view of the calcaneus is advantageous for identifying this lesion and is recommended for all horses with calcaneal bursitis. Radiographic signs include increased soft tissue swelling, osseous lysis, fragments/sequestra, and new bone production more commonly associated with chronic conditions.

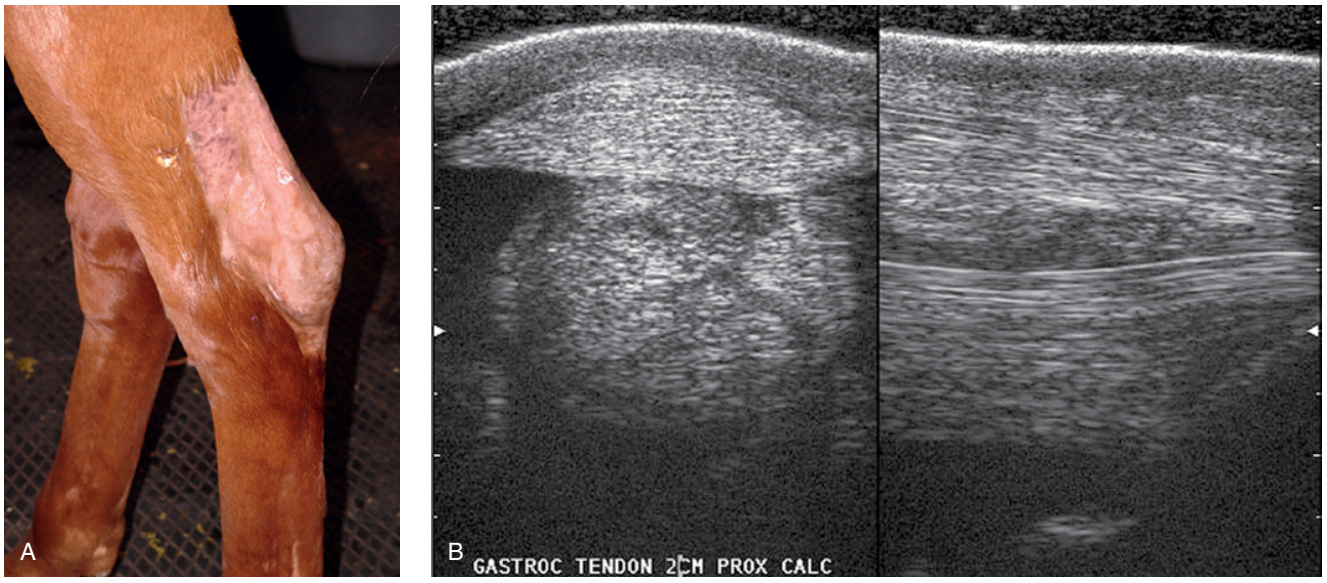


Figure 5.98. Thickening and swelling was identified in the calcaneal tendon of this horse (A). Ultrasound demonstrated enlargement of the gastrocnemius tendon with focal and diffuse hypoechoic areas with loss of normal fiber alignment on both the transverse and longitudinal planes (B).

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US frequently shows enlargement of the GT with focal or diffuse hypoechoic areas with loss of normal fiber alignment when viewed in the longitudinal plane. Mild thickening of the wall of the calcaneal bursa due to chronic distension with primarily anechoic fluid may be identified. Infection and trauma tend to involve the attachments of the GT, SDFT, and calcaneal bursa. The GT and SDFT and the LDFT and TS should all be evaluated with US. The prognosis for athletic soundness in horses with chronic calcaneal bursitis and associated osteolytic lesions of the TC is guarded.

Curb

The term curb describes a swelling along the plantar aspect of the calcaneus distal to the point of the hock/tarsus but does not typically addresses etiology. While desmitis of the long plantar ligament (LPL) can manifest as a swelling in this region, it is not the only cause of curb.[120] It is felt that curb may result from excessive violent tension or direct trauma. Trauma may occur from kicking walls or tailgates in trailers and violent attempts to extend the hock. In addition, there is some speculation that conformation may predispose to the development of curb. Lameness, if it occurs, is typically minimal and of short duration. Horses with persistent lameness, particularly those with swelling more distal to the LPL, should be evaluated more carefully. Superficial digital flexor (SDF) tendinitis of the proximal metatarsus may have a similar appearance, with swelling distal to the calcaneus. Occasionally, a similar swelling exists without lameness. This swelling has a fluid consistency and appears sonographically as a fluid pocket surrounding the SDFT. Drainage of the fluid may alleviate the problem, but persistence of the fluid may warrant corticosteroid injection.

While curb is primarily an injury of racehorses, and most commonly the STB racehorse, it does occur in nonracehorse sport horses (Figure 5.99). US examination of this area reveals thin subcutaneous fibrous tissue layer, the SDFT as a crescent shape, narrow in a medial to lateral direction, and somewhat thickened from that seen proximally. In the longitudinal scan, the normal SDFT has a dense parallel fiber pattern. Deep to the SDFT, the LPL is at full thickness (plantar to dorsal direction), is rectangular in shape, and is firmly attached to the calcaneus. At this level, the DDFT is out of view medially and must be evaluated by placing the transducer plantaromedially. In fact, only 30% of horses in one study had injury of the LPL.

Curb was reported to be caused by excessive stress or strain from race training (80 horses), direct trauma (11 horses), previous injection (3 horses), associated injuries (3 horses), and an apparent "bad step" or unknown cause (13 horses). The most common reported form of direct trauma was stall or wall kicking. Horses with LPL desmitis, SDF tendonitis, or DDF tendonitis or those with combined soft tissue injuries exhibited most pronounced lameness. Horses with curb are more likely to have swelling and injury of peritendinous and periligamentous tissue and/or SDF tendonitis than injury of the LPL. Horses with severe SDF tendonitis, LPL desmitis, LDF tendonitis, or combined soft tissue injury received long-term rest (3 months) and controlled return to exercise, but they were the most likely group to do poorly or to remain lame. Diagnostic US examination may reveal that the swelling is due to peritendinous or periligamentous fibrosis, SDF tendinitis, DDF tendinitis, or LPL desmitis. Curb is primarily an injury of racehorses but does occur in nonracehorse sport horses. Within racehorses, the STB is predisposed. Gait differences between the STB and TB may account for this predisposition, but conformation may play a role. Sickle-hocked conformation is more prevalent in the STB breed and seems to predispose these horses to the development of curb. It is felt that overload or excessive strain of plantar soft tissue structures would be exacerbated in horses with sickle-hocked conformation. Etiology of curb in the sport horse may be different than that in racehorses because direct trauma was a common etiology in nonracehorse sport horses. Horses with more severe lameness may have acute LPL inflammation and tearing due to violent exertion, trauma from kicking, and violent attempts to extend the hock.

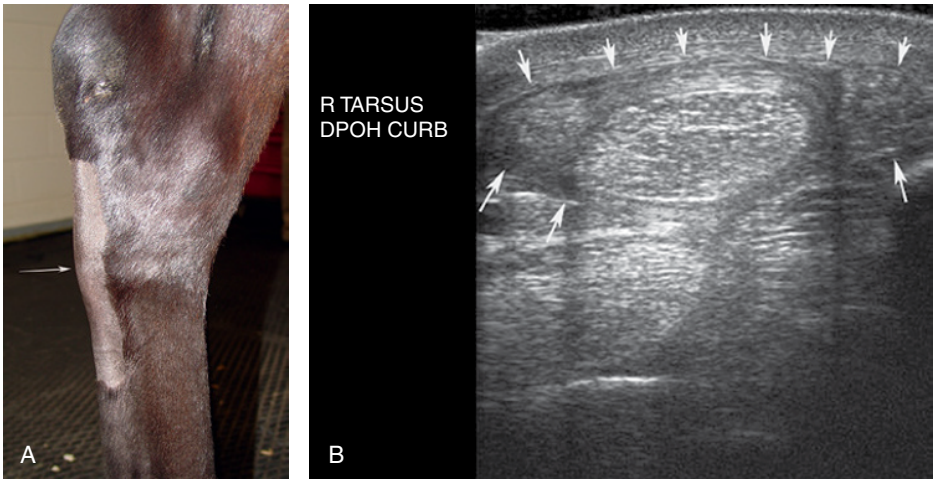


Figure 5.99. Horse with swelling along the plantar aspect of the calcaneus distal to the point of the hock (A; arrow). Often the swelling has a fluid consistency and appears sonographically as a fluid pocket surrounding the SDFT (B; arrows demonstrate a swelling consistent with an organizing hematoma surrounding the SDFT).

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Acute Tenosynovitis of Extensor Tendons

The long digital extensor tendon (LoDET) lies on the cranial aspect of the crus and courses over the dorsum of the tarsus just lateral to the MTR of the talus. A proximal, middle, and distal extensor retinaculum cover the LoDET at the level of the tarsus. The tendon courses over the tarsus within a synovial sheath from approximately the level of the LM and continues coverage through the distal retinaculum almost to the junction with the lateral digital extensor tendon (LDET). At this level, the conjoined tendons of the LoDET and LDET muscles continue distally similarly to the common digital tendon of the front limb. The LDET of the hindlimb courses distally across the lateral surface of the tarsus.

Direct trauma is the most likely cause of tenosynovitis of the extensor tendons of the tarsus. Blunt trauma to the dorsal and lateral surface of the tarsus can create acute tenosynovitis of extensor sheaths, particularly the LDET. The LDET sometimes develops idiopathic or acute tenosynovitis. The swelling within the synovial sheath may protrude proximally and distally to the retinaculum of the lateral digital extensor. Lameness when it occurs is sudden in onset and moderate in severity. Mild distension of the sheath may occur initially. These effusions often persist in spite of appropriate treatment. The LoDET is rarely affected. The distal aspect of the PT tendon can be injured as well. Diagnostic US should be utilized to assess the tendon for damage. Treatment is determined based on the specific structure(s) involved. If the tendon appears to be normal, then the treatment may be directed at the synovial sheath.

Rupture/Tendonitis of the Peroneus Tertius (Fibularis Tertius)

The PT muscle is tendinous over its entire length. It arises from the extensor fossa of the lateral femoral condyle and inserts with two distinct tendons to the tarsus. Rupture of the PT is uncommon with trauma being the most common cause.[78,86,117,132] The muscle or tendon can rupture anywhere along its course and can result in an avulsion fracture at its origin in the extensor fossa. When this muscle is ruptured, the stifle flexes, but the hock does not. Rupture of the PT is usually due to overextension of the hock joint (Figure 5.100). Entrapment of a limb with violent struggles can cause this condition. Rupture also may occur during the exertion of a fast start causing overextension of the tarsus such as in jumping. It can also occur after a full-limb cast is applied to the hindlimb. PT injury in adult horses may cause swelling of the distal cranial aspect of the crus near the TC joint (usually between the trochlear ridges), but lesions in the mid-crus seem to be more common and often are not associated with significant swelling. If the PT is ruptured, the tarsal joint can be extended while the stifle is flexed, indicating loss of the reciprocal apparatus function. There is often a dimpling that occurs within the common calcaneal tendon. If the origin of the PT fractures from the femur, femoropatellar effusion is a prominent feature, and the gait deficit is similar. On US, the acute ruptured PT is enlarged and shows multiple small focal anechoic to hypoechoic lesions. On a longitudinal scan, the normal fiber orientation disappears.



Figure 5.100. Rupture of the peroneus tertius disrupts the reciprocal apparatus allowing extension of the tarsus and fetlock with the stifle flexed. *Source*: Courtesy of Dr. Gary Baxter.

Careful monitoring of the healing process with US is necessary to prevent reinjury.

Complete rest is the best treatment. The horse should be placed in a stall and kept quiet for at least 4–6 weeks, and then limited exercise should be given for the next 2 months. Most cases heal and show normal limb action, and if properly conditioned, most horses can return to normal work. After rupture of the PT tendon, 71% of horses returned to their previous level of exercise with a mean rehabilitation period of 41.5 weeks. If a horse was a performance horse at the time of injury, it was 11 times less likely to return to its intended use. Furthermore, if an additional structure was injured at the time of rupture of the PT, the horse was 14.6 times less likely to return to its intended use.[78] Prognostic indicators previously reported for this type of injury include location and degree of rupture. Prognosis has been reported to be favorable when rupture occurs in the tibial region and poor if the rupture occurs at the point of origin from the extensor fossa of the lateral femoral condyle with any associated fracture. Performance horses have a decreased prognosis to return to their intended use after rupture of the PT.

PERIARTICULAR TARSAL CELLULITIS

Spontaneous cellulitis can develop around the tarsus that can present as an unusual cause of an acute-onset, severe non-weight-bearing lameness. One report described this condition in a series of TB racehorses.[111] Skin abrasions were identified in some horses somewhere in the more distal part of the limb, and the onset of swelling usually began on the dorsal aspect of the hock. The cellulitis was quite painful to palpation. These clinical signs were associated with a fever, neutrophilia, and hyperfibrinogenemia. Prompt, aggressive treatment

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with oxytetracycline (5 mg/kg IV SID) or other antimicrobial agents and phenylbutazone (4.4 mg/kg) for at least 5 days combined with forced walking exercise for as much as possible daily resulted in rapid resolution of lameness. The prognosis was usually favorable; 1 of 10 horses developed osteomyelitis of the third tarsal and fourth tarsal bones, although this horse ultimately made a complete recovery.

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TIBIA AND CRUS

W. Rich Redding

INTRODUCTION

The tibia is one of the major weight-bearing bones of the hindlimb. The tibia extends obliquely downward and backward from the stifle to the hock. It is a tubular bone with a triangular-shaped cross section proximally changing to an oval shape as it courses distally. The tibia has three chief centers of ossification and two supplementary ones for the tibial tuberosity and the lateral malleolus.[14] The proximal articular surface of the tibia is roughly triangular in shape with the intercondylar eminence protruding from the center of the triangle providing attachment for the patellar ligaments. The proximal articular surface of the tibia has a flat table surface with a centrally located intercondylar eminence. The tibial tuberosity contains a deep extensor groove/muscular sulcus, which is filled by the cranial tibial (CT) and the long digital extensor tendon (LoDET) muscles. The tibia is covered by muscle and tendon on the cranial, lateral, and caudal aspects. The medial surface is without muscle covering and easily palpated under the skin. Proximally the medial surface is broad where it furnishes insertion to the medial collateral ligament of the stifle and the sartorius and gracilis muscles. The caudal surface is flattened and provides attachment for the popliteus muscle proximally and for the deep flexor muscle of the digit via a series of roughened lines that fade distally. The nutrient foramen is situated among these lines and can be quite prominent. The distal articular surface contains two deep sulci oriented in caudomedial to craniolateral direction, which articulate with the trochlear ridges of the talus.

The primary axis of tension strain in the tibia occurs on the cranial surface, while the caudal surface experiences compressive strains.[26] These strains are particularly large in the proximal metaphyseal and mid-diaphyseal region. Distally the tibia experiences large torsional forces. Casting the hindlimb does not alter the strains experienced by the tibia. Casts may be effective to immobilize the tarsus and distal limb of the horse, but they do not protect the tibia from weight-bearing loads. For more information on the muscular anatomy and innervation of the tibia, refer to Chapter 1.

DIAGNOSTIC ANALGESIA OF THE CRUS

There is no practical method to use diagnostic analgesia to differentiate conditions of the tibia and crus. The distal portion of the crus can be desensitized with a peroneal and tibial nerve block. Pain from injuries involving the distal tibia and caudal soft tissues of the crus may also be abolished with this block, but the results are unreliable. Lameness of the crus becomes more likely in horses in which perineural and intra-articular techniques for the rest of the limb have been exhausted.

IMAGING THE TIBIA/CRUS

In a foal or young horse, the tibia can be radiographed easily. However, in an adult, it is better to focus on a particular region of the tibia and center the X-ray beam appropriately. Large plates should be utilized to obtain radiographs of the entire length of the tibia. Orthogonal views should be acquired, and specific oblique projections can be focused at specific areas of interest depending on the tentative diagnosis. The proximal fibula is notorious for simulating fractures. The tibial tuberosity forms from a separate ossification center and varies greatly with age. The appearance of the tibial tuberosity varies substantially with different projection angles. The parallel ridges located on the caudal and caudolateral surfaces of the tibia (termed *muscular lines*) mark the attachment of the deep digital flexor muscle and must not be mistaken for a so-called bone reaction. The opposite tibial region can be used as a normal comparison.

Ultrasonographic examination can be very helpful in evaluating the soft tissues of the crus, particularly the caudal and cranial muscle masses (gastrocnemius and peroneus tertius/CT). The patellar ligaments can be evaluated quite effectively with diagnostic ultrasound. Puncture wounds and lacerations of the crus can be carefully investigated to document the extent of soft tissue involvement with ultrasound. Clinical conditions that ultrasound may be helpful to define include gastrocnemius injury, peroneus tertius/CT tendon damage, thoroughpin (or "false" thoroughpin), calcaneal bursitis, and tarsal sheath tenosynovitis.

Nuclear scintigraphy should be considered when the lameness cannot be blocked out or cannot be confirmed/defined to a specific region with radiographic and/or ultrasound examination. Any young racehorse with an acute hindlimb lameness potentially due to a stress fracture of the tibia is a candidate for scintigraphy. Lameness conditions suspected to originate more proximally on the limb or to involve multiple sites within the limb are also candidates for scintigraphy. In older horses scintigraphy may permit detection of subtle bony changes undetected by other diagnostic methods.

CLINICAL CONDITIONS

Conditions that affect the tibia/crus can affect the large muscle mass surrounding the tibia, the shaft of the tibia, and/or the articulations of the tibia (stifle joint proximally and TC joint distally). Traumatic events are the most common cause of injuries to the tibial region. Injuries in this area can manifest a wide variety of clinical signs apparently dependent on the structures involved. In the young racehorse soft tissue injuries are not as common as injuries to the bone. In the mature sport horse, husbandry practices can increase the incidence of soft tissue injuries, particularly lacerations and

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puncture wounds. More extensive injuries may involve the tibia. Fractures can occur in about the same frequency as other long bones. Fracture types include incomplete, proximal physeal fractures (in young horses), diaphyseal fractures, and tibial crest fractures.[2–13,15–18,26,27,29,34–36] Training-related bone injuries also occur in the shaft of the tibia.[1,19–22,24,25,28,31] In racehorses stress-related bone injury occurs in the tibial cortex and often present as a subtle, high-speed lameness. In addition, enostosis-like lesions (ELLs), similar to what is seen in humans, have been described in horses and occur as focal or multifocal sclerotic lesions within the medullary cavity of long bones.[1]

Traumatic fractures of the tibia are typically catastrophic injuries (Figure 5.101). Kicks or traumatic injuries can impart enough energy to this unyielding bone to cause it to shatter.[3,6–9,13,15,16,26,29] As bone deforms, it stores the applied energy as strain energy, and this energy ultimately is released when the bone fractures. Bone loaded rapidly fails at a higher load and releases more energy than if it is loaded slowly. Unfortunately these fractures are usually associated with an explosion of bone, which can cause significant damage to the skin and increases the risk of contamination of the fracture since they become open. Younger animals are subjected to the same types of traumatic injuries as adults, but because they are actively growing, fracture configurations will often involve the growth plate(s). As in all long bone fractures in horses, maintaining the integrity of the skin and appropriately stabilizing the limb before shipping are critical to a successful outcome if surgery is being considered.[5]



Figure 5.101. Injuries that impart significant energy to the tibia often cause the bone to shatter and usually cause significant damage to the skin and surrounding soft tissues that increase the risk of contamination of the fracture.

ENOSTOSIS-LIKE LESIONS

ELLs were first described in horses as focal or multifocal sclerotic lesions within the medullary cavity of the long bones. Their role in causing lameness has been questioned. ELLs are typically recognized on scintigrams as variable intensity focal intramedullary IRUs.[1,4,23] Lesions are most often detected in the tibia and radius more commonly than the humerus and third metacarpal/metatarsal bones. Lameness severity is often correlated to radiopharmaceutical uptake intensity (Figure 5.102). The ELLs located in the humerus are felt to cause more severe lameness than ELLs in other anatomic locations. Radiographic findings tend to lag behind scintigraphic findings and when seen are not always associated with lameness. However, radiography is not a sensitive method for detection of acute bone injury. A period of bone remodeling is required before lesions become radiographically evident. ELLs have a characteristic radiographic appearance with a focal area of increased opacity usually close to the nutrient foramen and within the medullary cavity. Orthogonal views are critical to confirm the intramedullary location of the bone response as ELLs. In many horses these lesions appear to be incidental findings and often are considered unrelated to lameness.

Diagnosis of ELLs as a cause of lameness is by the exclusion of other causes in the face of positive diagnostic imaging findings (scintigraphy or radiography). The intramedullary location of the lesions makes them difficult to confirm as the source of lameness without the use of diagnostic analgesia. This can be accomplished with the use of a tibial and peroneal nerve block in the hindlimbs and a median and ulnar nerve block in the forelimbs.

There appears to be little evidence of predisposing causes for the development ELLs. Conformation seems to have little effect on the development of ELLs. However, there does appear to be a risk factor for Thoroughbreds to develop ELLs. High concussive loads associated with race training may be related to the development of ELLs. Interestingly ELLs can cause pronounced, recurrent lameness within the same animal but in different locations. Some animals have been diagnosed with ELLs annually for several years. Differentiating ELLs from stress reactions and fractures in racehorses is especially important. Identifying the location of the IRU within the tibia (cortical vs. cancellous) is critical to successfully diagnose and manage the horse's lameness. Continued training on cortical stress reaction(s) or stress fracture(s) may lead to fracture propagation and a catastrophic injury. However, horses (racehorses in particularly) with an intramedullary location are more likely to be ELLs. The presence of ELLs does not seem to have long-term adverse effects on athletic performance.

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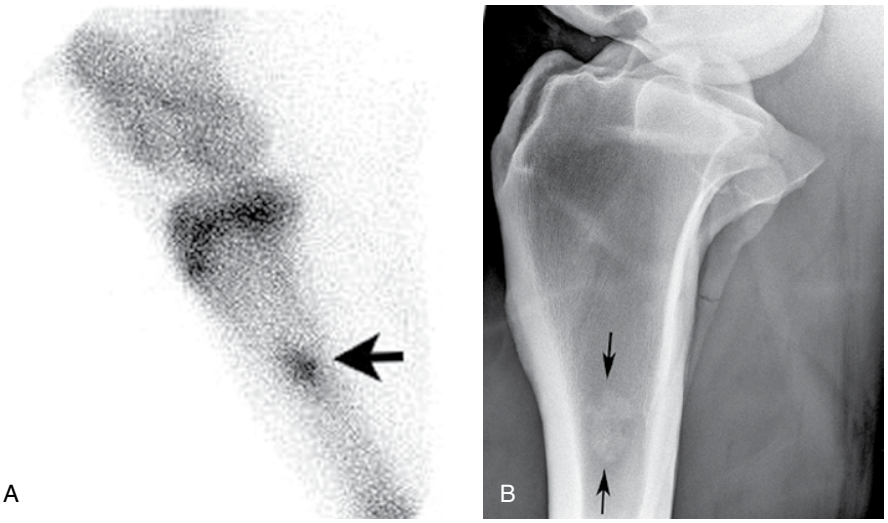


Figure 5.102. ELLs are typically recognized initially with scintigraphy (A) and appear as focal intramedullary areas of variable intensity IRUs (based on comparison of the lateral and caudal bone scan images). Lateral radiograph of the tibia of the horse in image A demonstrating typical ELL (B; arrows).

Tibial Stress Reaction

A stress fracture refers to a fracture that occurs in normal bone caused by repetitive stresses with resultant failure of the normal integrity of cortical bone. Stress fractures are a well-recognized cause of lameness in racehorses. Tibial stress fractures typically occur in young unraced but heavily trained Thoroughbreds but also occur in racing Quarter horses and Standardbreds.[19–22,24,25,28,31] The diagnosis usually involves a combination of radiology and scintigraphy. Eleven of 13 fractures were located in the tibial midshaft in Standardbreds[24] as compared with the proximal diaphysis in Thoroughbreds.[21,22,28,31] Stress fractures of the tibia occur predominantly in 2-year-old horses, while humeral stress fractures occur predominantly in 3-year-old horses and seem to occur more commonly in heavily trained animals. Horses with stress fractures of the tibia often have a history of sudden, severe hindlimb lameness that quickly resolves with stall rest. Recurrence is common often during the next strenuous exercise (speed work). This condition typically presents as a unilateral lameness with a shortened cranial phase of the stride and a stabbing hindlimb gait. Physical exam findings are often negative, but on rare occasions mild swelling and pain can be elicited by deep compression. Hindlimb flexion tests usually accentuate the lameness. Unfortunately, diagnostic analgesia is not practical except to eliminate the lower limb as a source of lameness.

Tibial stress fractures frequently involve the caudolateral cortex in the distal metaphysis or the caudal diaphysis (Figure 5.103). Proximal caudolateral fractures can occasionally be seen. Although a discrete fracture may be apparent as an oblique linear radiolucency on radiographs, more often radiographic confirmation is lacking. Oblique views may help demonstrate the presence of an endosteal and/or periosteal callus. Scintigraphic examination is a very sensitive tool to evaluate bone remodeling and is the diagnostic tool of choice. Scintigraphic findings consistent with stress remodeling are marked focal caudolateral diaphyseal or metaphyseal uptake.

Horses with tibial stress fractures should be removed from training and allowed to rest until the fracture is fully healed. In general this requires 4–6 months of rest with the horse returning to training based on follow-up examinations including either radiographic or scintigraphic examination. There is a definite risk of the tibial fracture propagating if training and racing activity is continued. Spiral fractures can develop commencing proximally in the lateral cortex and extending distally to the craniomedial cortex. Location of the IRU either within the cortex (stress fracture) or the medullary cavity (EELs) of the tibia can provide important information about the correct management of these horses. More information and images illustrating tibial stress fractures can be found in the racehorse sections in Chapter 9.

FRACTURES OF THE TIBIA

Horses with complete tibial fractures usually present with acute, non-weight-bearing lameness with moderate swelling and significant angular deformity. These fractures are typically trauma related associated with management and during pasture turnout and are not performance-related injuries. They occur in all types of horses of all different ages. Diaphyseal and proximal physeal fractures usually manifest valgus deformity or angulation of the limb laterally distal to the fracture site. Radiography is necessary to confirm the diagnosis and allows the characterization of the bone injury. Despite recent advances in fracture treatment, comminuted fractures of the equine tibia are among the most difficult to repair successfully. The integrity of the skin and soft tissues can also negatively influence the outcome of the surgical repair of these fractures. Contraction of the craniolateral muscle mass overlying the tibia abducts the distal limb and forces the fracture fragments medially. The sharp ends of the fractured bone can be easily forced through the thin soft tissues of the medial aspect of the crus and penetrate the skin. Limb stabilization is critical as continued use of the unstable limb damages

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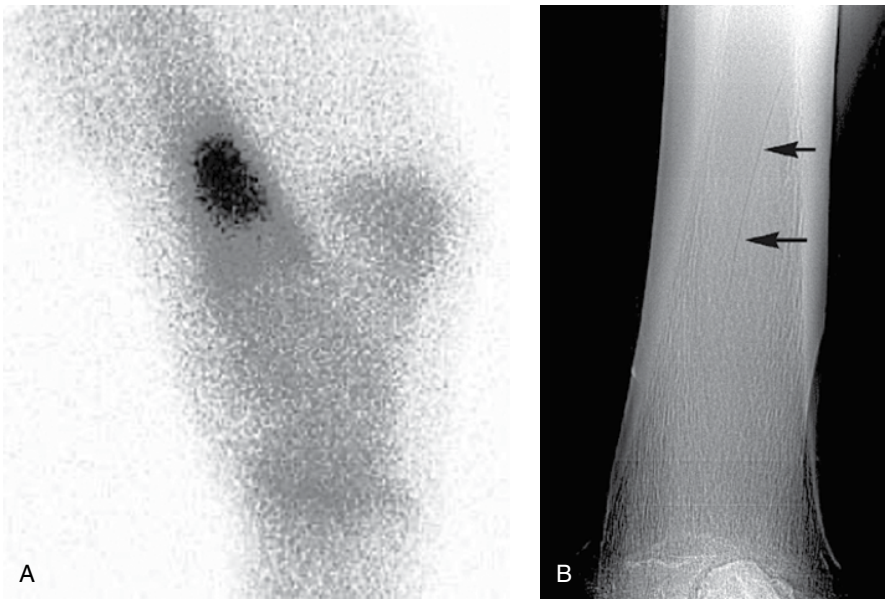


Figure 5.103. Lateral scintigram (A) and tibial radiograph (B) demonstrating an incomplete tibial fracture (arrows). *Source*: Courtesy of Dr. Gary Baxter.

the fracture ends and fracture reduction and the ultimate stability of the construct/repair can become significantly compromised.

Optimal surgical candidates need intact skin to reduce the risk of infection of the fracture repair. In most fractures stabilization prior to shipping is critical to minimize further damage to the soft tissues and the bone at the fracture site.[3,5,7,9,32] Regrettably, tibial fractures are difficult to adequately stabilize because immobilization of the stifle (the joint proximal to the fracture) is impossible. Casts and splints simply add weight to the distal limb, causing further motion at the fracture site. A Robert-Jones bandage with a lateral splint attached to the bandage with nonelastic tape should be applied to the limb. The horse should be shipped facing forward in the trailer so that as the vehicle brakes, the increase in weight-bearing can be borne through the front limbs.

INCOMPLETE FRACTURES

Incomplete fractures of the tibia often present as an acute severe lameness.[15,16] The relative stability of the tibia and therefore the limb does however allow partial weight-bearing. Radiographs are necessary to confirm the presence of and configuration of the fracture (Figure 5.103B). A complete series including oblique projections are necessary to assess the severity of the fracture. Incomplete and nondisplaced complete fractures can be managed by conservative therapy, which involves cross-tying the horse in the stall. Providing significant pain relief should be adjusted to avoid overuse of the fractured limb. In general, horses with shorter visible fissure lines (3–7 cm) are more likely to survive than those with longer spiral fissure fractures (12–15 cm). Additionally, nondisplaced fractures on the caudal compression side of the tibia tend to become unstable (Figure 5.104).[10]



Figure 5.104. Lateral radiograph of a horse with a long spiral oblique tibial fracture that exits the caudal cortex and is likely to displace further if left untreated. *Source*: Courtesy of Dr. Gary Baxter.

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Incomplete fissure fractures of the distal metaphysis of the tibia may benefit from a cast. These can be applied in the sedated standing horse in a normal weight-bearing position. Stall rest is recommended until there is evidence of fracture healing. This usually requires a minimum of 3–4 months. Maintaining a cast on the hindlimb can be problematic and may require several cast applications/resets to keep the horse comfortable on the limb. During the last 30 days of confinement, once the cast has been removed, a program that incorporates a gradual increase in hand walking is recommended. In the best of circumstances, these fractures can heal without significant complications. However, propagation of the fracture is not uncommon. Careful daily monitoring to assess the comfort level of the horse may provide an indication about the integrity and stability of the fracture. Any trend toward increasing discomfort may indicate a compromise in the stability of the fracture and a need for surgical repair.

PROXIMAL PHYSEAL FRACTURES

The bones of young horses are more elastic than mature animals and are less likely to develop into severely comminuted fractures. Young horses have an increased risk to develop fractures through the epiphyseal growth plate (physis). Physeal fractures are called Salter–Harris (SH) fractures and are further classified based on the relative involvement of the physis, metaphysis, and epiphysis.[3,12,13,33] The young horse that develops a tibial fracture will most commonly develop an SH type II through the proximal epiphyseal growth plate. A type II SH fracture involves the growth plate and a component of the metaphysis (Figure 5.105). Fracture separation begins at the medial aspect of the physis and propagates laterally through the zone of proliferating chondrocytes within the physis and breaks out into the lateral metaphysis. This is a consistent configuration of proximal tibial physeal fractures and implicates lateral to medial bending as the primary force responsible for the injury.

Blunt trauma to the lateral aspect of the stifle while the foal is weight bearing on the limb can create this type of physeal fracture. This injury can also occur when a recumbent foal attempts to stand while the uppermost limb is positioned under a stationary object or when the mare steps on the recumbent foal's uppermost hindlimb. There is usually marked soft tissue swelling on the medial aspect of the physis. These fractures can be relatively straightforward to repair with a good prognosis for healing but not necessarily for soundness. Fracture repair is best achieved by reestablishing support on the medial aspect of the proximal tibia. Screw fixation, pinning, and plate fixation have all been described for treating these fractures either alone or in combination.[3,12,13,34,35] Medial plate application is currently the most common fixation technique utilized for repair and can be significantly improved by the placement of a tension band along the tibial crest. This neutralizes the distracting forces of the quadriceps muscle that is transmitted across the stifle joint to the tibial crest via the patella and the patellar ligaments' attachment to the tibial crest.

These fractures usually heal quickly and the prognosis is good. Rapid reestablishment of the vascular system to the physis occurs with the stabilization of the fracture and allows fracture healing to occur quickly, normally in 3–4 weeks. The amount of damage to the soft tissues, growth cartilage, and the stability of the repair are major factors that can significantly affect the outcome. The amount of damage to the growth cartilage depends on the time from injury to surgery and the adequacy of preoperative immobilization. The trauma that created the

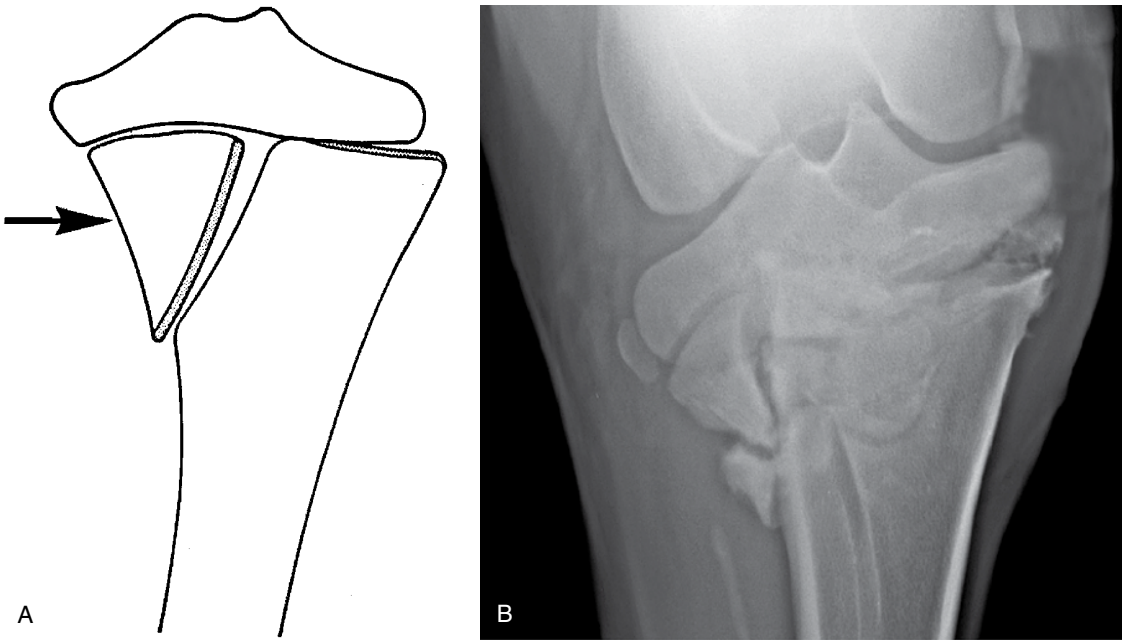


Figure 5.105. Line illustration (A) and caudocranial radiograph (B) of the stifle demonstrating a Salter–Harris type II fracture of the proximal tibial physis. The metaphyseal component is always lateral (arrow) and usually involves approximately one-third of the distance across the physis. *Source*: Courtesy of Dr. Gary Baxter.

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fracture often damages the physis leading to growth disturbances. The entire growth plate can close, which can lead to shortening of the tibia. Compensatory bone growth in the rest of the limb may counteract the shortening of the tibia. Implant removal may be considered to prevent or reduce the likelihood of premature physeal closure and limb shortening. This can be done in most instances at 8 weeks postoperatively. In general, the smaller the foal, the better the prognosis.

DIAPHYSEAL FRACTURES

Fractures of the diaphysis of the tibia are the second most common type of tibial fracture and occur in all ages of horses. Fracture type can range from simple oblique to highly comminuted.[3,6–9,13,15,16,26,29] Diaphyseal fractures are thought to occur when the tibia is loaded under considerable torque in combination with an excessive external force. Most diaphyseal fractures have a spiral configuration and many are severely comminuted. These fractures are characterized by sudden onset of non-weight-bearing lameness. Diaphyseal fractures are easily diagnosed but require radiographs to assess the fracture configuration. These fractures are often catastrophic injuries characterized by extensive comminution especially in mature horses. However, simple long oblique fractures of the mid-diaphysis do occur and are often repairable provided that they are not open. Because the tibia has very little muscle coverage on the medial aspect, fracture fragments often disrupt the skin and soft tissues (Figure 5.101). Attempts at repair of open tibial fractures are seldom practical because of the very poor prognosis. However, diaphyseal fractures in foals tend to be simple oblique or spiral in configuration and closed, making foals much better candidates for surgical repair (Figure 5.106).

The Thomas splint has been utilized but has rarely been successful as a treatment for tibial fractures in some foals. It consists of an aluminum frame with a ring that fits over the thigh and into the groin of the horse with cranial and caudal extensions reaching the foot to apply traction to the limb. Traction is applied with 1-in. white tape attached to the distal limb and pulled to the lower bar of the splint to stabilize the entire limb. Long-term management of fractures of the tibia in this manner can become quite expensive, and these splints are often difficult and tedious to apply.

Surgical repair is compromised when the location of the fracture is in the distal diaphyseal or metaphyseal location, which frequently precludes adequate implant purchase in the distal fragment. Because of the reciprocal apparatus, traction on the distal limb does not help reduce a diaphyseal tibial fracture—in fact, it causes overriding—hanging the limb in tension is not helpful. Placing the injured tibia down with the horse in lateral recumbency impedes surgical manipulation of the diaphyseal fracture and necessitates a medial surgical approach, which is not ideal because of the lack of soft tissue coverage. A cranial approach with the horse positioned in dorsal recumbency with minimal traction on the limb is the recommended approach for surgical repair. One or two bone plates are usually required depending on the fracture configuration and the size of the horse.



Figure 5.106. Lateromedial radiograph of a tibial fracture in a foal at the time of repair using a single plate placed on the cranial aspect of the tibia. The single screw was used to compress the fracture line. Note the solid caudal buttress on the caudal aspect of the tibia. *Source*: Courtesy of Dr. NA White.

TIBIAL TUBEROSITY FRACTURES

The large cranial eminence of the proximal aspect of the cranial tibia is called the tibial tuberosity. This tuberosity has a prominent groove that serves as the site for attachment of the middle patellar ligament and is flanked by rough areas for attachment of the medial and lateral patellar ligaments.[14] The cranial border of the proximal tibia distal to the groove for the middle patellar ligament is referred to as the tibial crest. The tibial tuberosity is a supplementary center of ossification that is completely ossified between 36 and 42 months of age.[14] The three patellar ligaments also serve as the site of insertion for the quadriceps femoris muscle group onto the tibial tuberosity.

Fractures of the tibial tuberosity occur infrequently.[2,4,11,17,18,27,36] They often present with severe non-weight-bearing lameness. It is not unusual to have a wound or abrasion on the cranial aspect of the stifle/tibia from the traumatic event. When they occur, it is often in mature sport horses that have sustained a direct impact of the stifle on a fence or jump.[11] Tibial tuberosity fractures may also occur due to a direct kick to the stifle area. Swelling of the cranial aspect of the stifle region can be

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diffuse and crepitus is often palpable over the tibial crest (Figure 5.107). These fractures can be articular or nonarticular with the fragment frequently being displaced proximally. Synovial effusion often accompanies fractures with articular components, but the effusion may be difficult to delineate from the overall swelling. A complete radiographic study of the stifle is necessary to accurately define the fracture. The oblique orientation of the tibial crest requires an oblique Cd35″L-CrMO radiographic view as the most useful in visualizing the fracture line and determining the fracture configuration.

The most common fracture of the tibial tuberosity is a nondisplaced nonarticular fracture. These fractures can heal satisfactorily with conservative management in the form of stall confinement and cross-tying. Careful radiographic monitoring is critical to assess fracture fragment displacement and to monitor healing. Most nondisplaced fractures with adequate stability are successfully managed and do not require internal fixation.[2] Stall confinement for a period of 60 days with serial radiographs to confirm appropriate healing is used to determine when exercise can begin. Smaller fracture fragments that are associated with wounds may require surgical removal (Figure 5.108).

Fractures that displace frequently displace proximocranially. Displaced fractures of the tibial tuberosity may be candidates for surgical repair. Stabilization should employ some form of a tension band applied cranially (Figure 5.109). The implants should stabilize the fracture while accurately reconstructing the joint surface. Most often this is in the form of a plate with the plate oriented obliquely along the tibial crest. The screws should be directed mediad and laterad in an alternating pattern to avoid aligning them in a straight line, which may predispose the underlying bone to fracture. Fractures of the tibial tuberosity have a reasonably good chance for successful repair.

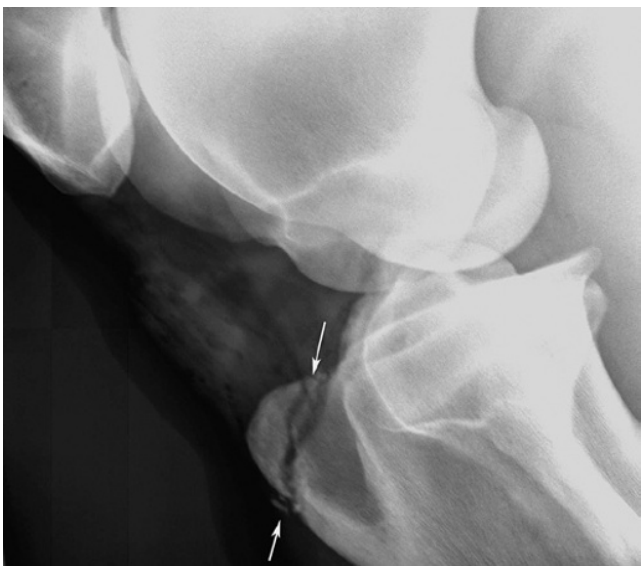


Figure 5.107. Fractures of the tibial tuberosity most often occur in mature sport horses usually due to a direct impact of the stifle on a fence or jump. Some nondisplaced fragments (arrows) may heal with stall confinement.

GASTROCNEMIUS DISRUPTION IN FOALS AND ADULTS

Disruption of the gastrocnemius muscle can cause dysfunction of the reciprocal apparatus. Gastrocnemius muscle injury is rare but can be a source of lameness in the



Figure 5.108. This small chronic fracture of the lateral tibial tuberosity (arrow) was removed because it was contributing to chronic drainage and lameness. *Source*: Courtesy of Dr. Gary Baxter.

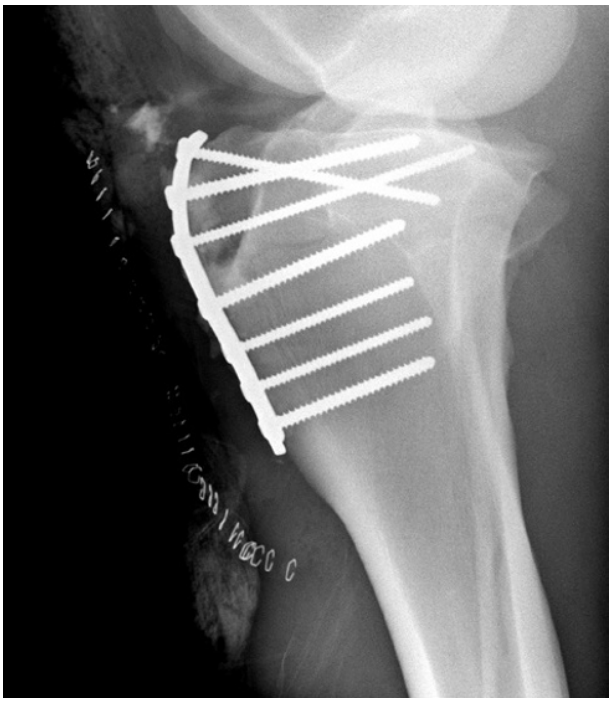


Figure 5.109. Displaced fractures of the tibial tuberosity are often best managed with tension band plating to counteract the pull of the quadriceps muscle.

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adult or the neonate. In the foal injury is usually severe and can cause recumbency and an inability to stand (Figure 10.60). Disruption occurs when the tarsus is maintained in flexion and the stifle is subsequently extended. Foals are often reported to have been through difficult births, many of which had a "hip lock" dystocia.[30]

Clinically, gastrocnemius disruption is demonstrated by hyperflexion of the tarsus when standing. The muscular injury usually occurs at the musculotendinous junction. The femoral vein, artery, and associated branches traverse the caudal aspect of the crus in close association to the gastrocnemius muscle. Disruption of these vascular structures can occur along with the muscular damage and can significantly affect the prognosis. Ultrasound can be a very useful tool to confirm the muscular damage and associated hematoma formation. Foals with moderate to severe gastrocnemius disruption are thought to have a guarded prognosis for future athletic function.[30] External coaptation can be required in some cases in the form of sleeve casts or splints. Gastrocnemius disruption in foals usually has a favorable prognosis for athletic function. This injury in foals is also discussed in Chapter 10.

Injury of the gastrocnemius muscle, often to the origin of the muscle, is an unusual case of lameness in adult horses. Forceful extension of the hindlimb may cause injury to the gastrocnemius muscle. Affected horses may have a gait abnormality characterized by lateral rotation of the point of the calcaneus and medial rotation of the toe. Nuclear scintigraphy is critical in the diagnosis and may demonstrate a focal area of intense radioisotope uptake in the caudodistal aspect of the femur in horses with injury to the origin of the gastrocnemius muscle. Roughening of the caudodistal cortex of the femur may be apparent radiographically in chronic injuries. However, radiographic imaging of this area can be difficult due to the thickness of the overlying soft tissues. A lateral radiographic of the femur (not the stifle) is necessary to reveal proliferative changes in this location. This condition can occur in mature animals but carries a poor prognosis for return to soundness. Horses with partial disruption of the gastrocnemius have a chance to return to athletic function. An extended period of rest followed by a gradual return to exercise can be successful in returning the horse to soundness. The amount of time required should be sufficient to allow the muscle to repair adequately. Some form of stabilization of the affected limb may be required. The decision to use external coaptation is based on the degree of caudal reciprocal apparatus dysfunction and the systemic status of the horse.

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THE STIFLE: FEMOROPATELLAR REGION

Gary M. Baxter and Ken E. Sullins

INTRODUCTION

The stifle is the largest and most complex joint in the horse, and not surprisingly injury to the stifle is an important cause of hindlimb lameness. The stifle consists of three synovial compartments (the femoropatellar [FP] joint, the medial femorotibial [MFT] joint, the lateral femorotibial [LFT] joint), three individual patellar ligaments, medial and lateral collateral ligaments, and a complex array of intrasynovial ligaments and menisci that are necessary to support the function of the joints. The reader is referred to Chapter 1 for more detailed anatomy of the stifle region.

Historically, stifle problems in horses have been reported to represent between 2% and 8% of horses presenting for lameness.[4,99] In one series of 553 horses with hindlimb lameness, 326 of 795 stifles that were radiographed had visible abnormalities.[87] Femoropatellar and femorotibial lesions occurred at similar rates of 27% and 32%, respectively, and there was an overall incidence of 32% with evidence of osteoarthritis (OA). In general, clinical problems involve the FP and MFT joints more commonly than the LFT joint in athletic horses. Regardless of the type of horse, stifle problems appear to be quite common in routine referral practice and may increase as more advanced imaging capabilities continue to improve our abilities to make specific diagnoses.[7,60,67]

Clinical Findings and Diagnostics

The evaluation of stifle lameness is made by visual observation, palpation of the joints, gait evaluation, and elimination of other types of lameness. The examiner should become acquainted with normal palpation and normal variations; asymmetry within the stifle usually indicates a problem. Swelling may be impressive with acute injuries, especially with extracapsular swelling, which complicates the ability to make precise anatomic characterization. Acutely painful horses usually do not bear full weight by fixing the limb in extension when walking or standing. Bruising from external trauma is common from being kicked and in horses that jump over fixed fences. Local and systemic anti-inflammatory therapy may be required to reduce the swelling before a complete diagnosis can be made, although improvements in diagnostic imaging, namely, diagnostic ultrasound, can make characterization possible.[7,27,28]

Distension of the FP joint is better observed when viewing the horse from the side and may be obvious (Figure 5.110). On palpation, distension and thickening of the FP joint capsule may be detected between the patellar ligaments. The patellar ligaments provide useful landmarks for locating the three synovial joints of the stifle. Comparison with the opposite stifle should be made; both stifles can be palpated while standing behind most horses. Some normal horses, especially athletic horses, often retain mild femoropatellar effusion without a clinical problem being present. Femoropatellar effusion can be secondary to a problem in the MFT joint, but is often less severe than with primary FP joint involvement. Femoropatellar effusion may be simple fluid distension or there may be thickening of the periarticular soft tissues. Such thickening may be edema or consist of fibrosis from chronic inflammation. With chronic lameness, atrophy of the gluteal and quadriceps muscles on the affected side may be apparent. This may be obvious, or careful comparison from the rear and side may be necessary. Hindlimb flexion, either upper limb or full limb flexion, induces a painful response in most cases.

Stifle pain causes typical hindlimb lameness. Viewed from the side, the cranial phase of the stride is shortened, and the foot is carried closer to the ground. The toe may drag when the horse advances the limb at a trot, and toe wear may be obvious. When viewed from the rear at a trot, asymmetry in pelvic movement is often observed. The duration of gluteal rise is shorter, resulting in an early unweighting of the lame limb. This often results in a pelvic rise on the lame limb, which can often be seen best when viewed from the side (the reader is referred to Chapter 2 for more details on the lameness exam). The degree of lameness varies according to the severity of the injury. Stifle lameness usually cannot be definitively distinguished from hock pain or other sites of pain in the hindlimb. In some cases the horse may

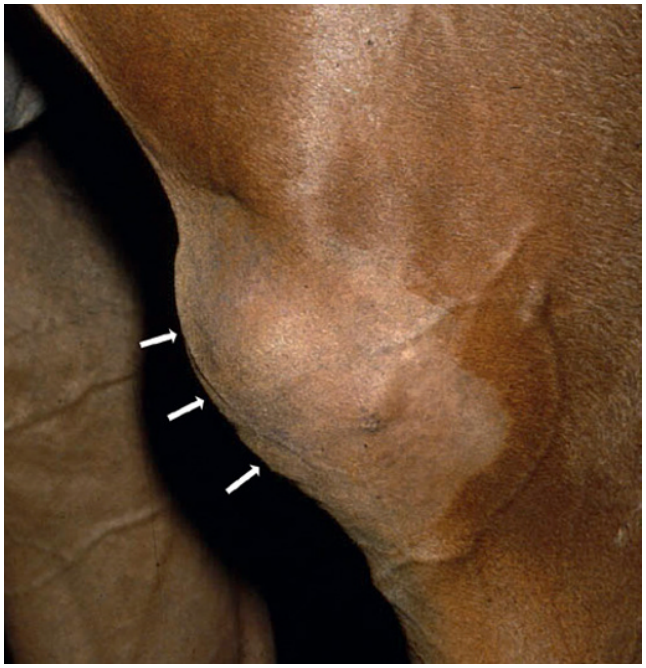


Figure 5.110. Lateral view of the stifle in a horse with severe femoropatellar effusion (arrows).

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demonstrate a subtle lameness under saddle or during specific movements.

Flexion of the hindlimb affects the digit, hock, stifle, and hip. Digital flexion is minimized if the limb is held loosely around the fetlock. However, the source of any increased pain following flexion must still be localized. The upper limb flexion (commonly referred to as the stifle flexion test) can provide the examiner with some assuredness that the region is involved if the horse reacts in a painful manner to flexion and the test exacerbates the lameness. See Chapter 2 for more information on flexion tests. However, the stifle flexion test is not specific for the stifle.

Lameness can be localized to the stifle with intrasynovial anesthesia if necessary. In some cases in which physical examination shows strong indications of stifle disease, diagnostic imaging may be performed prior to intrasynovial anesthesia in order to prevent fluid or air artifact from interfering with image quality. The number and sequence of blocking is dependent on the signalment of the horse, clinical findings, and clinician preference. For instance, a western performance horse with effusion of the MFT joint would warrant blocking the MFT joint alone. A horse with lameness that you are trying to rule out the stifle as the location of lameness may warrant blocking all three joints simultaneously. Without effusion, the MFT joint is the logical starting point if each joint is to be blocked separately, because the FP joint will usually have effusion if there is a problem, and the LFT joint is not the site of a problem as often. In spite of the frequent FP–MFT joint communication, communication between the FP joint and LFT joint is rare.[81] However, the FP joint in horses with OA occasionally communicates with both femorotibial joints.[81] None of the femorotibial joints usually communicate with their counterpart. However, pathologic changes within the LFT joint can be blocked with an MFT block, and both joints should be investigated with diagnostic imaging or surgery when needed.

A complete series of good-quality radiographs is the initial imaging tool to evaluate the stifle joints in most cases.[7] Digital radiography has made it possible to obtain very good films in the field.[10] Routine survey views include caudocranial (CaCr) and lateromedial (LM) views. The 30° lateral craniomedial oblique separates the lateral from the medial trochlear ridge, if that is needed.[35] The articular surface and sagittal integrity of the patella are demonstrated best by a flexed craniproximal to craniodistal tangential (skyline) view.[23,30,83] Some horses may have radiographically inapparent lesions or more extensive bony involvement than radiographs demonstrate.[19,67]

Ultrasonography is extremely helpful for evaluating the femoropatellar and femorotibial regions. The character of the articular surfaces, menisci and subchondral bone,[11,22,78] joint capsule,[67] synovial fluid,[60,79] patellar and intra-articular ligaments,[78] and periarticular tissues can be viewed with ultrasound.[22,67,79,80] However, the articular surface of the patella is not readily accessible.[7,67]

The benefit of scintigraphy for diagnosis of stifle lameness is variable. Some have reported it beneficial for lesions in the stifle region,[67,71] whereas others have questioned its ability to diagnose osteochondrosis (OC) or subchondral cystic lesions due to the lack of increased vascularity or increased bone remodeling required to sequester the radiopharmaceutical.[92] Normal horses have patterns of uptake highlighting the patella and caudoproximal tibia that should not be mistaken for pathologic change, especially in young athletic horses.[31]

Studies of both CT and magnetic resonance imaging (MRI) to image the stifle of horses have been reported although infrequently, since specially designed units are required to image the stifle (Figure 5.111). MRI provides the most anatomic detail available for imaging the equine stifle,[20,46,73,88] and CT can accurately image the trochlear ridges, condyles, and proximal tibia.[96] Of most value of using CT was the detail that it provided in the caudal portions of the femorotibial joints and within the intercondylar fossa.[72,96] When combined with contrast,

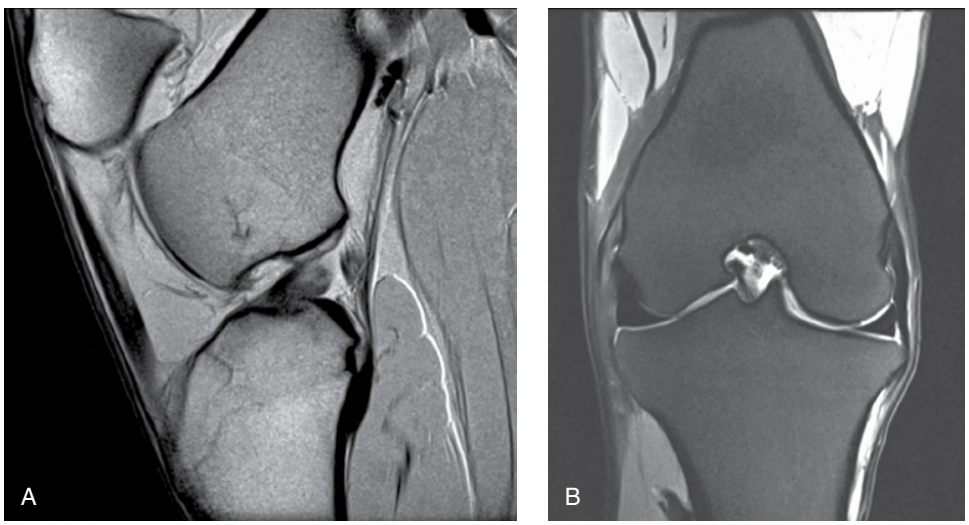


Figure 5.111. Sagittal T1 (A) and cranial to caudal PD fat saturation (B) MR images of a normal stifle joint illustrating the anatomic detail that can be obtained. Specially designed wide-bore units are required to perform MRI of the stifle in horses. *Source*: Courtesy of Dr. Kurt Selberg.

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CT offers more potential to image intra-articular soft tissues within the stifle than other techniques currently available.[9,72] The reader is referred to Chapter 3 for more information on the capabilities of CT and MRI.

Arthroscopy remains both a diagnostic and treatment tool for all of the stifle joints. Multiple different techniques and approaches have been developed, and arthroscopy remains the standard for exploration and surgical treatment of all three stifle joints without significant risk of incisional complications.[65,76] Arthroscopy is the definitive diagnostic tool for problems isolated to the FP, MFT, or LFT joints that are inapparent with other types of imaging.[35,64,65,93] Needle arthroscopy in the standing horse has been advocated to improve characterization of lesions although treatment of lesions may be limited with this technique.[36]

FEMOROPATELLAR JOINT

Osteochondritis Dissecans (OCD)

Femoropatellar osteochondritis dissecans (OCD) is a very common cause of stifle lameness rivaled in occurrence only by that in the tarsocrural joint.[51,63] In one study, Thoroughbreds were the most commonly affected of a series of 161 horses having surgery.[34] Seventy-eight percent were 2 years old and younger (most were yearlings), twice as many males as females were affected, and they were typically the better individuals in the herd.[104] Femoropatellar OCD primarily affects the lateral trochlear ridge.[34,63,64] However, lesions may be located on the medial trochlear ridge, the trochlear groove, the articular surface of the patella, or in any combination.[34,65]

Clinical Signs

Femoropatellar OCD usually causes visible joint effusion (Figure 5.110) and variable hindlimb lameness. In some cases the lesions might be incidentally visualized prior to lameness in horses that are imaged for sale. The lesion is a cartilage or osteochondral defect with separation, producing synovitis. Subtle effusion can be compared to the contralateral stifle, but at least half of affected horses have bilateral lesions.[34,63] Synovial fluid analysis is usually unremarkable. Uncommonly, horses with stifle lesions may also be affected in other joints; a complete physical exam noting effusion in other joints is indicated.[34,65] Clinically silent OC lesions may be present in other joints, and survey radiographs may be of benefit to best set expectations for clients.

The lameness may be so mild that joint effusion is the only presenting complaint; however, hindlimb flexion is usually positive in horses with clinically significant OCD. Others may be extremely lame, and some youngsters may have difficulty rising from recumbency. Chronic hindlimb lameness in foals may cause secondary forelimb flexural deformities, which could be the actual presenting complaint in some horses. The severity of synovitis depends upon the lesion itself and the amount of inflammation caused by physical activity; the degree of synovitis was not always comparable to the size of lesions in one report.[11] Uncommonly, the lateral trochlear ridge defect can be so severe that the patella may luxate laterally. Conversely, previously immobile OCD fragments may separate, causing sudden clinical signs in athletes.[27]

The usual age at presentation varies from weanling to 2- to 3-year-olds in training; more than half of a series of 161 horses were 1 year of age or younger.[34] Training may be required to cause clinical signs; presumably the osteochondral defect becomes disrupted, causing synovitis. Horses presenting after training has begun generally have less severe lesions than weanlings or yearlings that present earlier, which is probably why the older group has an overall better outcome after surgery.[34,65] Some horses have radiographic evidence of OCD without clinical signs. The clinical significance of the lesion should be confirmed by intrasynovial analgesia, particularly among horses in active training or older horses with minimal femoropatellar effusion or radiographic change. Weanlings with femoropatellar OCD may present with joint effusion, lameness, and no radiographic changes.[19] Horses with OCD lesions that appear on radiographs but are not causing clinical signs at the time should be monitored, especially during early training, with the goal of considering treatment if they become clinically significant.

Diagnosis

LM radiographs usually demonstrate a flattened defect in the proximal portion of the lateral trochlear ridge of the femur where it articulates with the distal patella (Figure 5.112 and 10.48). Ossification within the defect is variable, and loose bodies may be present.[19,34,64] Less commonly, deeper ossification defects or subchondral cystic lesions may occur, or the defect may be so large that the patella is unstable (Figure 5.113).[19,64,65] Caudolateral to craniomedial oblique films may provide more information about the severity of lateral trochlear ridge defects, which may extend over the lateral side of the trochlear ridge. Patellar changes may be due to primary OCD or secondary to irritation from a severe lateral trochlear ridge lesion, but may not be discovered until surgery is performed. However, severe patellar lesions can be observed in the LM view and typically contribute to a worse prognosis (Figure 5.114).

In one study, radiographically normal areas contained lesions detectable only by arthroscopy in 40% of 72 FP joints,[93] and more than half had lesions worse than anticipated from the radiographs.[19,93] Young foals normally have irregular contours of the proximal trochlear ridges that should not be confused with OC (Figure 5.115).[1,44]

Ultrasound should be considered as an adjunct to radiographs as the trochlear surfaces of the distal femur are readily visualized using ultrasound.[7,11] In addition, the LM extent of defects and the trochlear groove can be imaged, whereas they may be obscured on radiographs. The sensitivity and specificity of detection of lateral trochlear ridge OCD is quite good.[11] The cartilage on the lateral trochlear ridge is reported to be thicker than that of the medial ridge.[79] Underscoring the value of exploratory arthroscopy, accuracy on the proximal medial trochlear ridge is not high for ultrasound or radiography.[11,67,93] Although numbers were low, lesions in the midportion of the medial trochlear ridge were better detected by ultrasound than radiography in one study.[11]

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Figure 5.112. Loss of contour, flattening, or concavity of one of the trochlear ridges (arrow) can be one of the initial radiographic signs of stifle OCD.



Figure 5.114. Young horse with OCD lesions involving both the lateral trochlear ridge and the proximal articular surface of the patella (arrows).

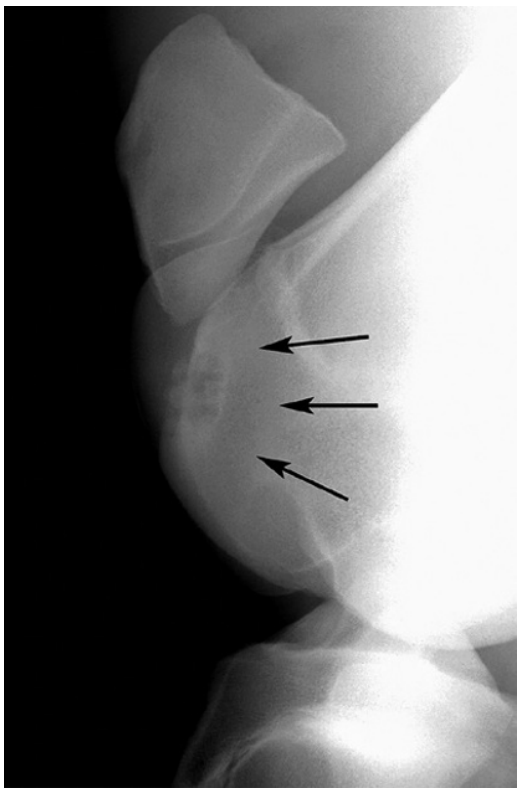


Figure 5.113. Lateromedial radiograph of the stifle of a horse with OCD of the lateral trochlear ridge. A subchondral cyst-like lesion with surrounding sclerosis (arrows) has formed beneath the surface fragments.

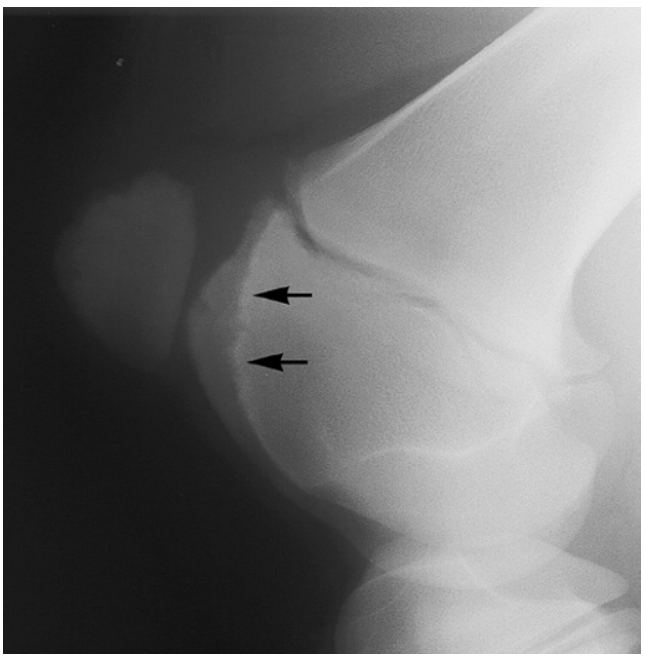


Figure 5.115. Lateral radiograph of the stifle of a young foal that demonstrates the normal irregularity of the subchondral bone along the proximal aspects of the trochlear ridges (arrows).

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In summary, a minority of horses with mild lateral trochlear ridge defects have no cartilage defect during surgery (Figure 5.116). Conversely, horses with more severe radiographic changes probably have cartilage defects at that location and possibly elsewhere in the same joint. When radiographic lesions are absent, lameness and synovial effusion often dictate the clinical significance of the OCD lesion.

Treatment

Surgical management is the mainstay of treatment for OC in young athletic horses.[76] Surgical debridement is also thought to produce better results than conservative therapy when clinical signs are present in adults.[63] However, in one study, some lesions detected up to 8 months of age were found to resolve over time,[24] and no worsening of the lesion occurred after 11 months of age. Stall confinement is recommended in very young horses with OCD lesions to protect the articular surface from disruption and to facilitate healing,[66] but surgery is indicated if the clinical signs persist after 1 year of age.[63,76]

Intra-articular and systemic anti-inflammatory therapy to minimize the chance of degenerative change and improve cartilage healing is advisable in young horses. Biologic therapies such as autogenous conditioned serum are recommended for intrarticular (IA) treatment, and polysulfated glycosaminoglycan is most often used for systemic treatment. Intra-articular corticosteroids are inadvisable where cartilage healing is needed,[15] and negative effects of steroids have been observed in immature equine joints.[39] The excess fluid can be drained at the time of treatment so that resolution of the synovitis can be monitored. In one study, half of 23 affected foals that were diagnosed early with OCD and treated with stall rest eventually raced.[66] Generally, the successfully treated horses tended to have less severe lesions.

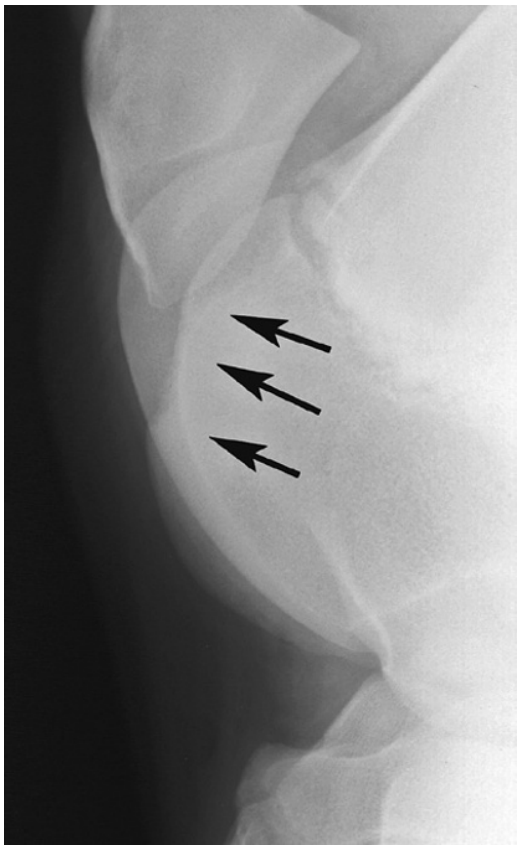


Figure 5.116. Lateromedial radiograph of a yearling with radiographic signs of lateral trochlear ridge OCD. Femoropatellar effusion was mild and arthroscopically the cartilage was slightly elevated but was intact. No debridement was performed, and as a 3-year-old, the horse was sound.

Sequential radiographs and clinical response to therapy are used to determine whether surgery is advisable. Surgery has been recommended for young horses with radiographic lesions greater than 2 cm in length or deeper than 5 mm or lesions with ossification in the defect and femoropatellar effusion.[63,65] However, the need to progress with the horse's conditioning schedule may dictate earlier surgical intervention, or preemptive removal is often requested by some owners and trainers in order to minimize the chances of the lesion affecting training and early competition.

Weanlings and short yearlings can present with effusion and lameness but no radiographic lesions.[9,66] Conservative therapy is indicated unless clinical signs persist. Subsequent radiographs may demonstrate healing or larger trochlear ridge ossification defects because the lesion has persisted while the surrounding trochlear ridge has ossified normally as the epiphysis has expanded.[19] Some believe that surgical lesions in foals and weanlings may progress, suggesting that surgery should be delayed until the lesion has fully developed.[19] This consideration will have passed after a period of conservative therapy. Medical therapy in cases without radiographic abnormalities can be diagnostic as well because a permanent response indicates that there is likely no articular surface lesion.

Arthroscopic surgery is indicated when it is obvious that the lesion will not heal with conservative therapy and in horses beyond a year of age with persistent clinical signs. The technique for arthroscopic debridement of femoropatellar OCD lesions has been well described,[63,65,68,76,101] and there are multiple different techniques and approaches.[65,68] The objective of surgery is to remove all loose osteochondral tissue and debride the lesion to leave healthy subchondral bone to fill with fibrocartilage and resolve the synovitis (Figure 10.49 in Chapter 10). A thorough evaluation of the joint is indicated as silent OCD lesions may be visible with the arthroscope that were not apparent on radiographs. It is also important to remove all the debris generated during the debridement process because it can contribute to continued synovitis.[48,68] Arthroscopic reattachment of OCD cartilage flaps has been reported in select horses but is not performed routinely.[90]

Prognosis

The prognosis for athletic activity following arthroscopic surgery for femoropatellar OCD is generally good. Of a series of 134 horses, only 16% were unsuccessful for reasons related to the OCD.[34] Slightly fewer were successful when OCD in other joints underwent surgery concurrently. Increased lesion size had a significantly negative effect upon outcome (Figure 5.117). A recent arthroscopic study also indicated that horses with larger OCD lesions had an increased risk of developing

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Figure 5.117. Horse with a very large OCD lesion of the lateral trochlear ridge seen at necropsy. Lesion size is thought to correlate with prognosis.

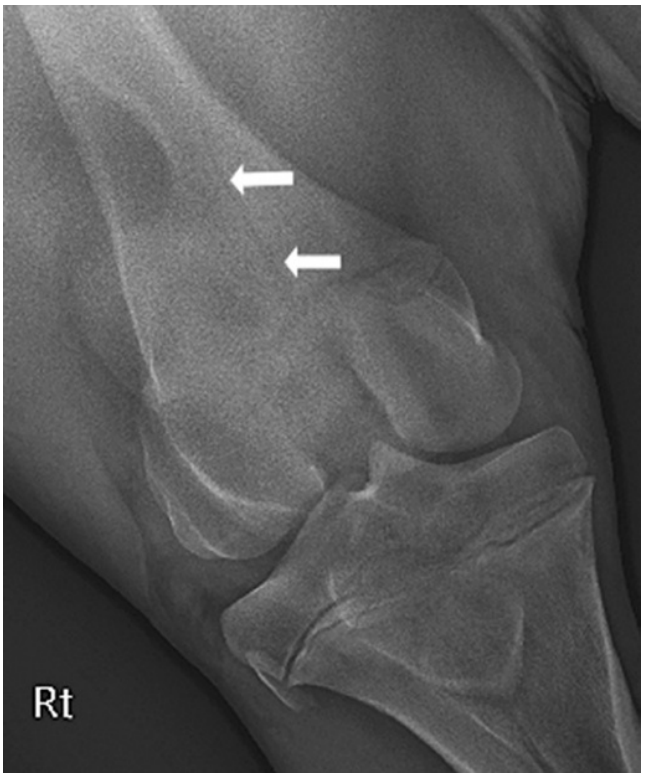


Figure 5.118. Craniocaudal radiograph demonstrating a suspected Salter–Harris type IV fracture of the medial aspect of the distal femur (arrows). This foal was lame at the walk and had significant FP and MFT joint effusion. The foal was managed conservatively.

surgical site infections, which corresponded to an increased risk of septic arthritis in the FP joint compared to other joints.[13] The overall success rate of 64% reported by Foland et al. was comparable to the starting rate of normal Thoroughbred horses in the same time period. Outcome is usually not correlated to sex, function of the horse, lesion location, uni- vs. bilateral involvement, or the presence of loose bodies.[34] However, a recent study of Warmblood stallions found that OCD lesions of the FP joint limited future performance of affected horses compared with OCD lesions at other locations.[100] Also, horses with concurrent patellar and large lateral trochlear ridge lesions tend to have a worse prognosis, and many surgeons recommend extended periods of inactivity after surgery in these horses to help minimize the development of OA (Figures 5.114 and 5.117).

Intra-articular Fractures: Femoral Trochlea

Etiology and Diagnosis

The cranial aspect of the FP joint is subject to direct blunt trauma, contributing to fractures of the trochlear ridges. Kick injuries to the stifle from other horses usually damage the patella instead of the trochlear ridges. Chip fractures have been reported in both trochlear ridges of the distal femur.[69] One horse sustained the injury by striking a jump (stone wall), and the other two occurred from punctures. Sepsis should be considered in these cases. Dyson has reported medial or lateral trochlear fragmentation in eventers after hitting jumps.[27] Separation of previously quiescent OCD fragments can also occur after direct trauma and should be considered in the differential diagnosis.[27]

SH type IV fractures of the distal femur in young horses that communicate with the FP joint can also occur from direct trauma.[103] Lameness is usually severe and femoropatellar effusion is obvious when the fracture enters the joint (Figure 5.118). The fracture may involve one or both trochlear ridges. The tangential view in addition to the CaCr and LM radiographic projections may be helpful to determine the fracture configuration.

Treatment and Prognosis

Fractures of the trochlear ridges are best removed using routine arthroscopic approaches regardless of the cause or if concurrent infection is present. Acute SH type IV fractures can generally be reduced, and fixation performed using internal fixation such as lag screws.[103] Nondisplaced fractures can also be managed conservatively. Although it may be possible to accomplish the fixation arthroscopically in some cases, the spatial orientation can be challenging. Depending upon the case, it

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may be more efficient to use the direct approach. The prognosis for these horses is usually reasonable for normal activity.

Intra-articular Fractures: Patella

Patellar fractures occur infrequently in horses, but several configurations including sagittal,[3,23,27,59] transverse,[47] comminuted,[77] basilar (proximal),[18,27,108] and distal fragmentation of the patella[62,84] have been described. Combinations of types of patellar fracture and associated fracture of the femur may also occur.[27]

Etiology

Direct trauma to the patella while the stifle joint is in a semiflexed position is commonly the cause. The patella is immobilized when the stifle is semiflexed, making it more susceptible to direct trauma.[21] Horses that jump can strike jumps and sustain bilateral patellar fractures,[59] or the fractures can occur as a result of a kick. Sudden lateral slips may cause a separation of the medial fibrocartilage.[23] The prominence of the medial trochlear ridge may be a point of contact, causing a relative higher incidence of fractures toward the medial side of the patella.

Some horses suffer fragmentation of the distal patella (FDP) when they return to work too soon after undergoing a medial patellar ligament desmotomy (MPD) and may be caused by direct trauma secondary to temporary patellar instability.[62,84] This condition is discussed in the next section.

Clinical Signs

Horses usually present with an acute onset of lameness and a significant painful swelling associated with the cranial aspect of the stifle. Femoropatellar effusion is usually profound, but significant soft tissue swelling may obscure palpation. Pain and sometimes crepitation may be found, and occasionally fragments may be palpable. Flexion of the stifle joint exacerbates the lameness and the painful response. Weight-bearing may be difficult with compromise of the quadriceps or from pain, so the horse may stand with the limb partially flexed without locking the stifle (Figure 5.119). Comminuted patellar fractures that compromise the middle patellar ligament and disrupt the reciprocal apparatus may cause an inability of the horse to support any weight on the limb. Clinical signs may diminish over a period of days to weeks of rest if smaller fragments do not affect the mechanics of the stifle.

Diagnosis

Radiographs are required to document the type and extent of the fracture. Routine LM and CaCr projections usually demonstrate transverse or comminuted fracture. The caudolateral to craniomedial oblique projection accentuates the apex (distal border) of the patella. Although swelling and pain may cause some resistance, flexed views are helpful or required to demonstrate some lesions. The flexed LM projection may accentuate some transverse fractures, and sagittal fractures, particularly the relatively common small medial fragments, require a cranioproximal to craniodistal (skyline) projection (Figure 5.120). Placing the plate proximal to the stifle with the beam traveling distoproximad separates the patella more completely from the trochlea, making it more visible.[23,77] Ultrasound is useful for identifying small fracture fragments, patellar ligament disruption, or lesions that may not be radiographically visible, such as medial fibrocartilage separation.[80]



Figure 5.119. Typical stance of a horse with a patella fracture. This horse had been kicked, and there was a wound over the cranial aspect of the stifle.

Treatment

Patellar fractures are often accompanied by severe soft tissue trauma involving the ligaments and joint capsule of the stifle joint.[28] Therefore, time and anti-inflammatory therapy may be required before treating the fracture. Nonarticular or small medial or basilar fragments may heal with rest and anti-inflammatory therapy.[27] Horses with relatively large incomplete apical fractures have become sound with extended confinement.[3,23] Some horses have returned to work with fibrous unions of the fracture.[23,77]

Horses with intra-articular fractures seldom remain sound when returned to work,[23,30,59] and stable fragments may displace when horses return to sustained work.[30] With adequate quadriceps stability, fragments approximating one-third of the patellar substance

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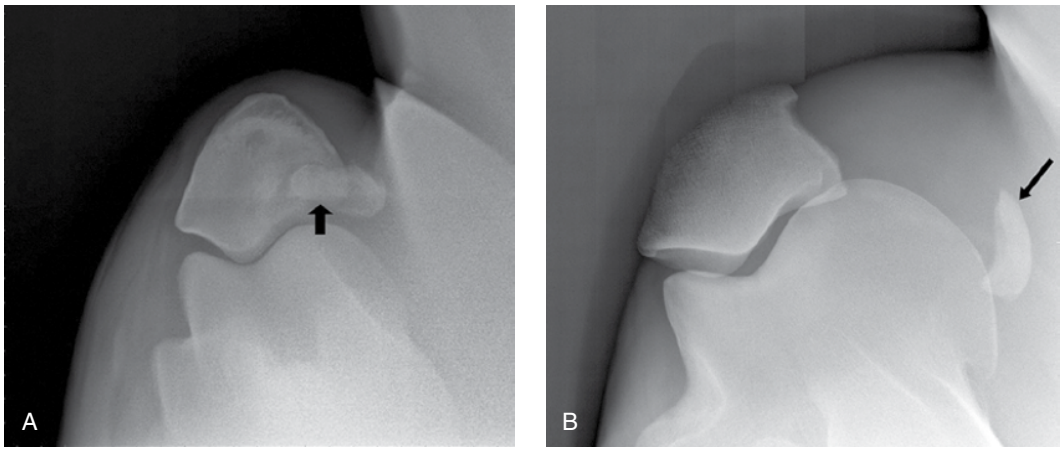


Figure 5.120. Cranioproximal to craniodistal (skyline) projections showing a typical medial patellar fracture fragment (A; arrow) and a large displaced sagittal fracture (B; arrow). Arthroscopic removal is indicated in (A). Arthroscopy was attempted in B, but an arthrotomy was necessary for removal.

can be successfully removed, and arthroscopy is recommended.[18,30,59]

Arthroscopy provides the best visualization of the fractures, facilitating complete removal of fracture fragments and exploration of the remainder of the joint without the risk of incisional complications of an open approach (Figure 5.121).[59] The fragment can be dissected from the patellar tendon with a large elevator or a motorized soft tissue resector. If the fragment is large, it can be divided with an osteotome or reduced with motorized equipment.[59]

Internal fixation should be considered for distracted or displaced fractures with sizable fragments. The patellar ligaments are the insertions of the quadriceps femoris and biceps femoris muscles. The goals of repair of larger articular fractures are integrity of the quadriceps mechanism and prevention of OA. Successful internal fixation of transverse distracted and longitudinally displaced fractures of the patella has been reported.[2,21,41,47,77] Transverse fractures require firm fixation because of the tendency of the proximal segment to become distracted by the pull of the quadriceps muscles,[21,47,77] although perfect anatomic reduction may not be required in every case. Tension band support is a good option for transverse patellar fractures.[47] Large extra-articular fractures can be removed, but it may not be necessary in all cases.[106]

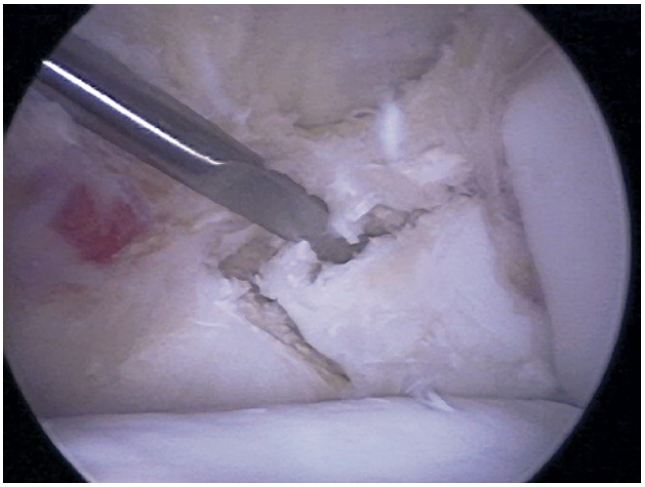


Figure 5.121. Arthroscopic view of a medial patellar fragment. The elevator is separating the patellar fragment from the soft tissue attachments.

Prognosis

The ultimate outcome depends upon retaining quadriceps function and adequate congruent articular surface; instability or persistent synovitis will result in OA. Following conservative or surgical therapy, lameness may persist for an undetermined period, while the synovitis resolves and any patellar ligament desmitis or enthesopathy heals.[23,59,79,80]

In a combined series of 19 horses from which articular medial fragments were removed, 16 returned to full work.[30,59] Failures occurred primarily because of preexisting OA, luxated patella during recovery from anesthesia, and one unexplained ensuing OA. These failures may point to the value of thorough preoperative ultrasonographic evaluation or more prolonged postoperative stall rest.

Internal fixation provides the most ideal set of circumstances for a favorable outcome for displaced and distracted fractures. Recovery from anesthesia may be the most critical point in the postoperative period. Horses with severely comminuted fractures are likely to remain lame. Some of these may attain breeding soundness with prolonged stall rest. However, if the quadriceps function is compromised or if severe OA develops, euthanasia should be considered.

Fragmentation of the Distal Patella

Etiology

FDP is an uncommon condition associated with previous medial patellar desmotomy (MPD),[40,62,84,91,102,108] and not all horses with this condition have undergone surgery.[62] Malalignment of the patella has been reported shortly after MPD,[8,90] and radiographically evident

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fragmentation was experimentally reproduced in 11 of 12 horses following MPD.[38] However, it is important to remember that fragmentation of the patella can occur from trauma or use-related injuries of the FP joint.

Clinical Signs

The clinical signs consist of variable uni- or bilateral hindlimb lameness and stiffness. FP joint effusion is common and palpable thickening of the joint capsule is often present, particularly in horses that have undergone an MPD.[62] Radiographic changes include bony fragmentation, spurring, subchondral roughening, and subchondral lysis of the distal aspect of the patella (Figure 5.122).[62,84,108] Cartilage fibrillation without radiographic abnormalities also has been reported.[38]

Treatment

Horses are usually best treated with arthroscopic surgery. The lesions observed at arthroscopy vary from flaking, fissuring, undermining, or fragmentation of the articular cartilage to fragmentation or lysis of the bone at the distal aspect of the patella. The subchondral bone is involved in most cases that had previous MPD. Fragments may be buried in the synovial membrane and tend to emanate from the more lateral portion of the distal patella.[62,102]

Prognosis

In one study, 8 of 12 horses that had a previous MPD became sound for their intended use after arthroscopic treatment of the FP joint.[62] In another study of horses undergoing MPD, 3 of 49 horses remained lame.[8] One of these horses had patellar fragmentation and did not become sound following arthroscopic debridement of the lesion. One additional horse with documented patellar fragmentation in that study was able to work with intra-articular medication. In most cases, the patellar misalignment was reported to correct within 5 months after MPD[84] and was not reported in horses 3 months after experimental MPD.[38]

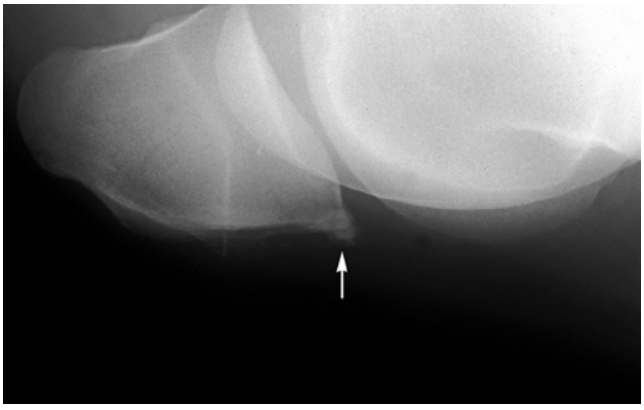


Figure 5.122. Flexed lateromedial radiograph of the stifle demonstrating fragmentation of the apex of the patella (arrow).

UPWARD FIXATION OF THE PATELLA (UFP)

Upward fixation of the patella (UFP) occurs when the medial patellar ligament (MPL) becomes caught over the medial trochlear ridge. In severe cases it becomes fixed in that position, the hindlimb cannot be flexed, and the horse assumes a posture with the affected limb extended in a caudally abducted position with the fetlock flexed due to the reciprocal apparatus (Figures 5.123 and 2.92). More commonly, intermittent UFP occurs, and a delayed release of the limb from extension is seen at a walk.

Etiology

UFP generally has been considered to occur in horses that have exceptionally straight hindlimbs; however, it can occur in horses with any conformation. Rooney considers most horses to have femorotibial angles approximating 135° and has stated that the problem occurs when the angle reaches 143°–145°.[86] Although the exact angle of occurrence likely varies among horses, it is reasonable to say that a straight-legged horse has a greater chance of reaching its individual point of injury. Furthermore, hyperextension of the limb exacerbated by walking a horse downhill causes the UFP to occur more frequently. Body type is strongly inherited; thus it is



Figure 5.123. The horse has upward fixation of the patella. The limb is locked in extension and extended caudally and slightly laterally. The digit is also fixed in the flexed position.

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likely that the tendency for UFP could be congenital. Shetland ponies are particularly affected, and UFP predisposes them to develop coxofemoral luxation.[17]

The condition also appears when the MPL becomes long enough to reach over the medial trochlear ridge in spite of normal conformation. Examples of this include loss of quadriceps muscle tone and traumatic hyperextension of the hindlimb. The ligaments may become stretched once upward fixation occurs, so recurrence is common. The MPL is thought to be weaker than the other two patellar ligaments, predisposing it to elongation.[89] Furthermore, younger horses, when they begin training, often lack the muscle tone they will acquire as they work. Upward fixation also has been observed in horses abruptly taken out of training and confined to a stall.

A recent report of 76 horses affected with UFP cites hoof balance and angle as predisposing factors, along with reduced muscle tone from seasonally reduced work.[26] Higher medial hoof wall and elongated toes were reported to cause hyperextension of the stifle and outward rotation of the limb, contributing to UFP. Corrective trimming and shoeing alleviated UFP in half of the horses and reduced the severity in an additional 20% of the horses (see "Treatment").

Clinical Signs and Diagnosis

In acute UFP, the hindlimb is locked in extension, as described above. The condition may relieve itself, or it may remain locked for several hours or even days. In other cases, there is only a "catching" of the patella as the horse walks. Hyperextending the stifle by walking down an incline may cause a jerking gait from intermittent catching of the MPL, and the horse may assume a crouched position, presumably to prevent stifle extension. Backing or moving in a tight circle also exacerbates the signs. When the MPL releases, the hindlimb usually jerks up quickly, mimicking stringhalt. Both hindlimbs usually are predisposed to the condition; however, truly unilateral cases often have a history of an inciting cause in the affected limb.

Palpation when the limb is locked in extension reveals tense patellar ligaments and that the patella is locked above the medial trochlear ridge of the femur. The horse drags the front of the hoof on the ground when it is forced to move forward with the limb locked. When the limb is in a normal position, the predisposition can be evaluated by forcing the patella upward and outward with the hand. If the limb can be manually locked in extension for one or more steps, it is predisposed to UFP; manual induction of UFP should not be possible in a normal horse.

Lameness usually is not severe or constant, but femoropatellar synovitis and distension may occur with repeated episodes. In some horses, UFP can be extremely difficult to manually reduce or correct by backing the horse. Coxofemoral strain may be associated with persistent UFP, and ponies are prone to secondary coxofemoral luxation.[17]

Radiographs of the stifle should be taken to eliminate conditions that predispose to UFP. Although uncommon, hypoplasia of the medial trochlear ridge, such as occurs with OCD, facilitates the displacement. Most affected horses do not develop radiographic abnormalities from intermittent UFP.[26]

Treatment

For a persistently fixed patella, a sideline may be applied to the affected limb so that as the limb is drawn forward, the patella is pushed medially to unhook the MPL, or downward, it often disengages the fixed patella. Backing the horse may also dislodge the patellar ligament. Personnel should take care to be out of the range of the "jerk" when the MPL releases. If the UFP cannot be reduced by manipulation, then a medial patellar ligament desmotomy is indicated.

Many horses respond to controlled conditioning to increase quadriceps strength and tone, which serves to tighten the MPL.[74] However, if the horse's fitness level drops below optimal, the UFP can recur. Obviously, rest or confinement is contraindicated. Conditioning, including going up hills without coming back down the incline, which exacerbates the hyperextension of the stifle, strengthens and tones the quadriceps.

Dumoulin et al. reported higher medial hoof walls and elongated toes along with reduced muscle tone from seasonally reduced work as predisposing factors in horses with UFP.[26] Shortening the toe and lowering the medial hoof wall sufficient to move break-over medial to the toe alleviated UFP in half of the horses and reduced the incidence in an additional 20%. Anecdotally, other farriers share this opinion.

Estrogen therapy has been used to treat intermittent UFP in horses.[29] The rationale is that estrogens can cause tendon and ligament relaxation, but it is unclear whether horses with upward fixation have overly tense patellar ligaments and if estrogen has any effect on patellar ligaments and tendons. It has been hypothesized that estrogen affects muscle cell metabolism and muscle tone leading to the anecdotal benefit sometimes reported. However, no studies currently exist examining whether this therapy is beneficial as a medical treatment. Recommended therapy is 1 mg of estradiol cypionate IM for every 45 kg of body weight (i.e. 11 mg/500 kg) once weekly for 3–5 weeks.[36] Concurrent anti-inflammatories along with exercise to strengthen the quadriceps muscles are usually recommended.

If conditioning and shoeing or medical therapy fail to halt the incidence of UFP, the MPL can be tightened with scar tissue by creating a series of longitudinal incisions in the MPL. This procedure can be performed using a scalpel blade or 14-gauge needle with the horse either standing or under anesthesia. The incisions are usually confined to the proximal third of the MPL.[49,82,97] This substantially thickens the MPL and presumably causes a functional shortening and tightening of the MPL.[82] In one study, UFP was eliminated in all seven reported cases.[97] A large advantage of this procedure compared with MPD is that the patella remains stable.

An older treatment that has been commonly used for horses with intermittent upward fixation and no palpable swelling of the FP joint capsule is the injection of counterirritants into the middle (MidPL) and MPL.[74] The injections are usually performed on the standing horse using mild sedation and nose twitch. Commonly used irritants contain 2% iodine; 1–2 mL is injected in

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six equally distributed sites along the MPL. The description includes a similar amount in the MidPL. Most horses exhibit a slight stiffness and swelling for a few days after the injections. Daily mild exercise is recommended, so there is minimal loss of muscle tone. While most horses respond well to this treatment, a few require re-treatment.

The commonly held opinion about the effectiveness of counterirritant injections into the MPL is that the ligament tightens, thereby mimicking increased tone from conditioning. In one histologic study the majority of the (severe) inflammatory reaction and all of the drug were found in the endotenon and peritenon.[12] The solutions appeared to follow the planes of least resistance. Within the dense collagen bundles, collagen fibers were disrupted without the same severe reaction, although there was fibrous tissue production and overall thickening. The mechanism of action, although still unproven, could be a reduction in the expansile ability of the fibrous MPL.

Ethanolamine oleate, a sclerosing agent, was compared with 2% iodine in almond oil for injection of MPL and MidPL in experimental horses.[98] Although both induced inflammation, 2% iodine in almond oil caused more thickening and a more significant inflammatory reaction, which would be expected to lead to more functional shortening of the ligaments. None of the horses were lame after the injections. A report of 70 horses treated with weekly homeopathic anti-inflammatory injections two to four times revealed an 82% success, with the remainder requiring surgery.[25] Possibly the number and frequency of injections may have contributed to local fibrosis.

When nothing else helps or the UFP cannot be reduced, an MPD can be performed using local anesthesia in the standing horse. Complications from that procedure should deem it the treatment of last resort.[38,50,62,84,91,102] The procedure is usually performed in the standing horse, although an alternative procedure performed with the horse under general anesthesia in dorsal recumbency has been described.[107] The ligament is usually isolated with hemostats and transected with a suitable blunt bistoury. The horse should be confined to a stall, with hand walking gradually increasing over a period of 90 days. A conservative conditioning program can then begin until regular work resumes.[102] Failure of MPD to correct the problem has been reported supposedly due to persistent tissue continuing to displace over the medial trochlear ridge.[26,45] Accidental transection of the middle patellar ligament produces a catastrophic result.[107]

As early as 1979, FDP following MPD was reported.[40] Gibson et al. observed articular cartilage fibrillation or fragmentation in all horses 3 months after MPD.[38] The patellar lesions were located distolaterally or centrally. There are also several other reports of distal patellar fibrillation, fragmentation, or subchondral lysis associated with varying degrees of lameness, femoropatellar synovitis, periarticular fibrosis, or enthesopathy at the origin or insertion of the MPL in horses having undergone MPD.[62,84,91,108]

A lateral shift in the patellar position[6] and increase in the patellofemoral angle[6,84] along with traction from the MidPL may explain the distal and lateral lesions that typically appear on the patella following MPD. Ultrasonographic evidence of MidPL desmitis was observed in experimental horses subjected to MPD and stall rested for 120 days, but no patellar fragmentation was detected.[61] Long-term MidPL desmitis and enthesopathy were discovered in a horse presented for purchase with obvious previous MPD and patellar fragmentation,[56] and 2 of 9 horses with patellar ligament desmopathy had MidPL desmitis associated with previous MPD.[30]

Although not in its original form, the MPL heals after MPD.[56] Most suggest that the risk of lameness and complications subsides after a suitable convalescence; therefore a minimum 90-day rest period has been recommended.[102] One experimental study that kept the horses stall confined for 120 days revealed positional change in the patella and MidPL enthesopathies, but neither lameness nor patellar fragmentation or femoropatellar synovitis was reported.[6]

Prognosis

The prognosis is good for horses that respond to a conditioning program and maintain that fitness level. After conditioning and ensuring correct shoeing, the logical progression is counterirritant injection, MPL splitting, and then medial patellar ligament desmotomy. The diagnosis should be confirmed before surgery is performed because more complications have appeared when MPD is performed on unaffected horses. A 90-day convalescence period following surgery is also recommended.

In one retrospective study, 46 of 49 diversely occupied horses undergoing uni- or bilateral MPD over a 10-year period by multiple surgeons became sound.[8] The authors theorized that the good results were because the surgery was only performed on horses that were actually suffering from UFP and that these horses had different stifle angles than normal horses that may prevent the development of complications. In another study of 15 horses and 6 ponies, no recurrence of UFP was observed in 19 of 21 horses following MPD.[70] However, postoperative radiographs revealed no abnormalities in the ponies but FDP was present in 4 horses and ossification at the tibial insertion of the MPL was seen in 8 horses.[70]

DESMITIS OF THE PATELLAR LIGAMENTS

Desmitis of patellar ligaments is an infrequently reported condition most often diagnosed in the MidPL (Figure 5.124).[30,79,80] Affected horses are usually athletes, mostly eventers, jumpers, or steeplechasers for which direct trauma from striking a jump is a risk factor.[27,30,79] Among event horses, the frequency of patellar ligament injuries followed only cruciate ligament and meniscal injuries in one report.[27] Many cases are diagnosed after a prolonged lameness with no recognized inciting incident.[28] Injuries to the MidPL combined with lateral patellar ligament (LPL) injury is thought to be most common followed by LPL desmitis alone and MidPL injury alone.[28] Desmitis of the LPL may accompany fracture of the tibial tuberosity.[79]

Clinical Signs and Diagnosis

Lameness is variable and usually exacerbated by stifle flexion.[28] The desmitis may not be evident upon physical

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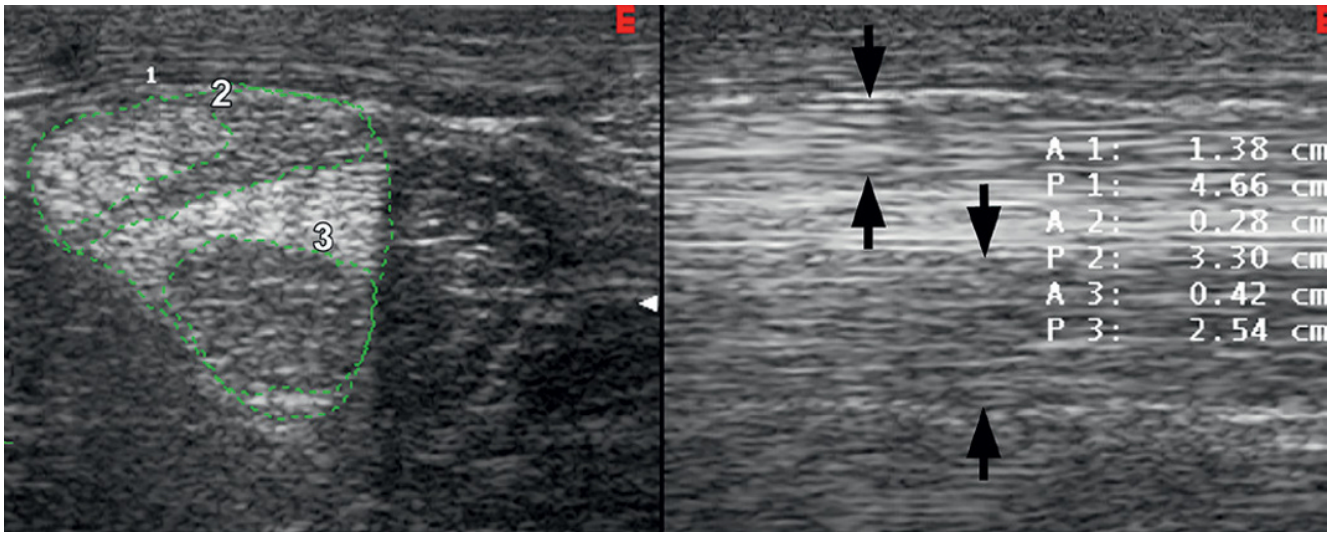


Figure 5.124. Ultrasound images of a middle patellar ligament injury. The cross-sectional view on the left demonstrates two areas of reduced fiber density (#2 and #3). The black arrows on the longitudinal view on the right outline the same areas at the same horizontal level as the image on the left. *Source*: Courtesy of Dr. Anne Desrochers.

examination, and palpable swelling or eliciting pain from a specific point may require partial flexion of the stifle.[28] More extensive lesions or ruptured patellar ligaments are usually easy to locate based on the horse's stance. Intra-articular or regional local analgesia produces equivocal diagnostic results.[28]

LPL injury was observed simultaneously with MidPL injury and less commonly alone.[28] In addition to direct trauma, MPL injury may accompany palpable patellar instability.[28] Middle patellar ligament lesions have been described as a linear lucency with fiber tearing or central hypoechoic regions similar to other desmopathies.[28,107] Some suspect that ultrasonographic abnormalities in patellar ligaments detected after injury bear more pathophysiologic significance than those reported following MPD. Proximal and distal enthesopathies may reflect bony disruption in addition to the soft tissue lesion,[79] but their role in lameness if the inciting inflammation subsides is unclear.[80,107]

The MPL is affected less commonly by primary desmitis. Similar trauma seems to cause avulsion fracture of the medial aspect of the patella instead of ligament injuries.[9] However primary MPL rupture can occur but usually heals quickly with stall confinement (Figure 5.125).



Figure 5.125. This horse developed a ruptured medial patellar ligament with an obvious palpable defect after hitting a jump. It healed completely and the horse became sound, although there were subtle radiographic changes on the distal patella.

Treatment

When the injury is confined to the soft tissue, therapy consists of rest and local and systemic anti-inflammatory therapy. Smaller enthesopathies or fragmentation also may be treated conservatively with stall rest and/or extracorporeal shockwave therapy. Injections of biologics, such as autogenous conditioned serum and platelet-rich plasma, into the affected ligament(s) may also be considered. Ultrasonographic monitoring of the healing process is advisable. Rest should be extended if MPL laxity has occurred to prevent distal patellar changes. Progressive rehabilitation exercises should also be considered to gradually strengthen hindlimb musculature and provide mobility.

Prognosis

In a series of nine patellar ligament injuries, no horses became completely sound for athletic performance, although working soundness was achieved in one.[28] These injuries may heal better with prolonged periods of restricted activity or with desmoplasty with or without regenerative medicine intervention. Pain control should be used cautiously with continued training due to the potential for patella instability leading to secondary OA.

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PATELLAR LUXATION/SUBLUXATION

Although patellar subluxation and luxation have been well documented in the literature, they are still considered to be uncommon.[33,43,52,55,57,58] The condition is usually recognized shortly after birth, but may be delayed in acquired cases. Acquired patellar luxation also occurs in older horses but even less commonly than congenitally.[58] Lateral[14,33,37,55,58] medial,[5,42,75] distal, unilateral, and bilateral patella luxations have all been described. Lateral luxation is most common. Severity can range from a mild intermittent subluxation to a complete luxation that is difficult to manually replace.

Etiology

Hypoplasia of the trochlear ridges (primary bone deformity) and hypoplasia of the ligamentous support structures have been suggested as causes.[85,95] The MPL is not as large or substantial as the MidPL or LPL.

In ponies and miniature horses, lateral patellar luxation is considered heritable.[43] The lack of adequate trochlear groove and/or lateral trochlear ridge is major factor. Congenital trochlear inadequacy is not always bilateral, at least in the pony.[42] Lateral patellar luxation can also occur in association with defective development of the lateral trochlear ridge caused by OCD.[94] When the condition occurs in the mature horse, trauma is thought to be the cause. Partial tearing of the insertion of the quadriceps femoris muscles on the proximal patella has been cited as the cause for distal patellar luxation between the femoral trochlear.[95]

Clinical Signs and Diagnosis

Foals with bilateral lateral luxation may not be able to stand or may stand in a crouched position because the displaced patella causes the quadriceps muscle group to become a flexor rather than function as an extensor (Figure 5.126). Signs vary according to the frequency, position, and degree of displacement of the patella.



Figure 5.126. This foal has bilateral patellar luxation. The quadriceps muscles cannot extend the stifle without the pulley of the patella on the trochlea. *Source*: Courtesy of Dr. Cliff Honnas.

Periods of normal ambulation occur if the displacement is intermittent. On palpation, there are varying degrees of patellar displacement and joint capsule distension. The intermittent patellar subluxations are usually readily replaced, whereas complete patellar luxation can be difficult to replace.

Persistent lateral subluxation of the patella presents much like a horse with UFP.[52] The limb is held in extension, causing the patella to be placed caudolaterad. When the limb is advanced, an awkward swinging toe-dragging gait occurs. Because some of these animals have a fairly normal gait, the condition may be missed until serious degenerative changes within the joint occur.[58]

Radiographs should be taken to confirm the diagnosis and identify the amount of degenerative changes within the joint. The flexed cranioproximal to craniodistal tangential (skyline) view is particularly beneficial to assess the degree of displacement and the depth of the trochlea. In the neonate, the normal irregular borders of subchondral ossification in the proximal trochlea should not be confused with degenerative changes (Figure 5.115).

Treatment

In one reported case of bilateral congenital lateral patellar luxation in a miniature horse, the foal overcame the crouched stance and began to walk normally by 6 months of age, although the patellae remained luxated. Most are treated surgically to minimize permanent deformity of the soft tissues and the development of OA.

When a sufficient femoral trochlea is present, stabilization of the patella in the correct position by imbrication can be performed. Release of the opposite soft tissue, including the contralateral patellar ligament, is often required to reduce tension on the repair.[33,52,58,75]

Horses with hypoplasia of the lateral trochlear ridge resulting in an insufficient trochlear groove to stabilize the patella require reconstruction by sulcoplasty and/or tibial tuberosity transposition.[5,32,37,55] Significant misalignment of the patellar tendons also requires transposition of the tibial tuberosity.[5] When OCD is the cause of the hypoplastic trochlear ridge, the cartilage surface and underlying bone are usually insufficient to sustain correction.[94,95]

Prognosis

The outlook for soundness without surgical correction is poor,[58] although pasture soundness may be possible, at least in miniature horses. The outcome is better for horses undergoing stable correction before the soft tissues contract and OA develops. Although successful cases have been reported in horses, they are rare, and miniature horses and ponies have fewer athletic expectations. In general, it appears that horses have more difficulty recovering from the repair than ponies or miniature horses in most situations.

In one study, a miniature horse requiring medial release, sulcoplasty, and tibial transposition became sound for its intended use.[5] In another report, 4 miniature foals undergoing imbrication alone to stabilize bilateral lateral patellar luxations survived to be functional, although one experienced a recurrence of unilateral lateral luxation.[33] Three of five horses that were surgically treated by release

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of the LPL and imbrication of the medial joint capsule succumbed to wound dehiscence, infection, or failure of the repair.[58] Only one horse was sound in light work 1 year postoperatively. However, the same procedure was successful for stabilizing a congenital bilateral lateral patellar luxation in a 6-week-old Shetland pony.[37]

Two of 3 horses that underwent sulcoplasty and medial imbrication of the joint capsules to stabilize lateral patellar luxations became sound 2 years after surgery.[55] In another report, a newborn Shetland pony with medial patellar luxation underwent sulcoplasty and lateral imbrication and was sound 6 months postoperatively.[42] Early recognition and correction of the problem likely contributed to the success in this pony.

SYNOVIAL OSTEOCHONDROMA IN THE HINDLIMB

Osteochondroma is a uni- or bilateral benign mass associated with cartilage growth centers. It is discussed as a distinct entity in Chapter 7. Young horses generally present with the problem, but they can remain clinically silent for long periods of time; therefore, it may be seen in older horses.[54] In the hindlimb, osteochondroma has been reported in the femorotibial bursa on the proximal lateral tibia,[54] in the plantar distal tibia within the tarsocrural joint,[53] within the tarsal canal on the fibular tarsal bone,[105] and on the calcaneus.[16] However, they can also be associated with the FP joint (Figure 7.50).

Clinical Signs and Diagnosis

Synovial effusion with or without lameness is the usual presenting complaint. The location depends upon which synovial structure is affected. Often the mass can be palpated percutaneously and may be bilateral. Radiographs usually demonstrate a focal semi-ossified variably sized mass associated with a growth plate or ossification center. The presenting signs and radiographic findings are often sufficient to make a presumptive diagnosis. Biopsy is usually better performed as excision. The radiographic appearance usually indicates that trauma is unlikely.

Treatment and Prognosis

In spite of the likelihood of it being a benign lesion, complete excision should be accomplished if possible. If the horse is actively competing, palliation of the synovitis may be accomplished by local and/or IA anti-inflammatory therapy. Routine postoperative bandaging and exercise restriction are tailored to the affected site. Although intrasynovial therapy is worthy of consideration, the prognosis for horses following surgical excision is generally good. Each case is different, and the lesions are likely to occur in more unusual locations than have been reported in the past.

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THE STIFLE: FEMOROTIBIAL JOINT REGION

Chris Kawcak

The medial femorotibial (MFT) and lateral femorotibial (LFT) joints are separate structures that usually lack physical communication except in cases of severe osteoarthritis (OA) and cruciate ligament damage and occasionally after arthroscopy in which the septum separating the two structures was perforated. The MFT joint usually communicates with the femoropatellar joint through a fenestration in the proximal aspect of the joint. This is important from the standpoint that disease in the MFT joint can manifest as femoropatellar joint effusion, sometimes leading to confusion about the true site of damage, since diagnostic anesthesia of one joint can influence the other. Consequently, heavy emphasis is placed on diagnostic imaging and sometimes arthroscopy to resolve the confusion.

Both MFT and LFT joints are each composed of a femoral condyle and tibial plateau and a meniscus between the two. A soft tissue septum separates the two compartments, which continues caudally to surround the cranial and caudal cruciate ligaments, making these structures extrasynovial in this location. The same is true for the medial and lateral collateral ligaments.

Horses with lameness originating from the femorotibial joints can vary in severity. Some cases are subtle enough to create a training issue without the presence of noticeable lameness, whereas others are severe enough to lead to non-weight-bearing lameness. In more severe and chronic cases, horses typically stand with the limb abducted, and there is usually noticeable atrophy of the hip and quadriceps musculature.

Whereas effusion in the LFT joint is difficult to palpate, effusion of the MFT joint is readily palpable, especially in the recess just cranial to the medial collateral ligament and proximal to the meniscus and tibial plateau (Figure 2.91) However, in more severe and chronic cases, significant soft tissue thickening can also occur in this area, often obscuring the ability to palpate effusion. This is referred to as a tibial buttress and is usually associated with advanced OA within the joint. The degree of effusion and swelling is usually graded as normal, mild, moderate, or severe, and any pain and thickening of the collateral ligament is noted.

There are no distinguishing characteristics to limb use in horses with stifle pain compared with those with pain at other sites in the hindlimb. All horses should be walked and trotted in a straight line. Subtle lameness is sometimes more noticeable in soft footing, especially when horses are jogged in a circle. Although most horses are more lame when the limb is on the inside of the circle, occasionally the lameness is worse when the limb is on the outside of the circle. Observing the horse under tack or in work can also improve characterization of the lameness.

Horses with stifle pain are typically responsive to full limb flexion. However, some information can be gained by attempting to isolate flexion to the stifle area. This is done by keeping the tarsal area as straight as possible and bringing the limb caudally from the tibia distally (Figure 2.108). A positive response to this flexion is not absolutely indicative of stifle pain, but it can give important information about the site of pain.

More severe cases of lameness attributable to the stifle are often obvious on clinical examination due to severe effusion, soft tissue swelling, pain on palpation, and severe response to flexion. However, diagnostic anesthesia is needed to confirm the site of pain in many horses. The approach to diagnostic anesthesia of the stifle can be complex. Intra-articular pain in one joint can be improved with anesthesia of the other joints, but the response is much better in the joint of pain.[57] Some clinicians block all three joints of the stifle at once to expedite the process, rather than starting with one joint and then blocking the other joints. Single-needle technique is preferred by the author.[22] Consequently, if all three joints are blocked at once, the clinician may need to further isolate the joints later to determine which ones contribute to the lameness.

Regardless of how stifle pain is confirmed in affected horses, accurate imaging of the joints is difficult. Improvements in radiography, ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) have helped to characterize the disease processes, but the lack of visualization during arthroscopic surgery and a minimal number of postmortem studies have prevented investigators from truly correlating imaging results to gross damage. Unlike other species, the femorotibial joints in the horse are closely packed, preventing arthroscopic visualization of most of the joints. Consequently, the ability to truly characterize and repair damage in many parts of the femorotibial joints is limited.

Diagnostic imaging has been well described in this text and others. Radiography has been most relied upon for characterization of bony changes in the joint, and the importance of proper positioning cannot be emphasized enough. There is recent evidence to show that joint space width can be accurately measured and followed with a properly performed caudoproximal to craniodistal oblique projection made at 10° from horizontal.[58] Diagnostic ultrasound is routinely used to assess lameness localized to the stifle and is helpful for characterizing meniscus and periarticular damage. However, it is limited compared to arthroscopic surgery for characterizing articular cartilage and cranial meniscal ligament damage.[1] Nuclear scintigraphy is moderately specific but insensitive for characterizing stifle lesions.[18] Computed tomography has demonstrated good correlation to radiography and ultrasound for detection of osteophytes in cadavers.[13] However, in a prospective clinical study, Nelson et al. demonstrated better characterization of cruciate disease, entheseal changes, and proximal tibial changes using CT and CT arthrography compared with

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other modalities.[38] MRI is the gold standard imaging modality for joint disease characterization in humans and is gaining some use in horses. High-field MRI has shown good correlation to soft tissue characteristics of the stifle.[11] In addition, low-field MRI has shown good correlation with gross and histologic findings[50] and has demonstrated usefulness for clinical cases.[35]

SUBCHONDRAL CYSTIC LESIONS (SCLS) OF THE STIFLE

This section focuses on the clinical signs, diagnosis, and treatment of subchondral cystic lesion (SCL) in the stifle. Although this occurs most commonly in the medial femoral condyle, it also can occur in the lateral femoral condyle and proximal tibia. See Chapter 10 for further discussion of SCLs as they relate to osteochondrosis and developmental orthopedic disease.

Etiology

Although SCLs are usually attributed to developmental orthopedic disease,[28,31] some clinicians strongly believe, and experimental evidence has shown, that it can also occur after trauma to the subchondral bone.[44,59] Ray et al.[44] have shown that creation of a lesion 3 mm deep and 5 mm in diameter into the subchondral bone created SCL in 5 of 6 horses. Furthermore, von Rechenberg et al.[45] showed that the lining of the cysts contains significant inflammatory mediators that may be responsible for enlargement and persistence of the cyst. The tissues from postmortem cystic material had significant osteoclastic function on bone. Therefore, regardless of the cause of cyst formation, persistent enlargement of some lesions may be due to a vicious cycle of inflammation.

Clinical Signs

Horses with SCL of the stifle can present in several different ways. Historically, many of these lesions are found on survey films of yearlings in which no lameness is present. They can also be found in any age horse in which no lameness is apparent. However, most horses with stifle SCLs present at or near the time that training begins when a mild to moderate degree of lameness is usually found. Effusion may or may not be present within the MFT and femoropatellar joints. When lameness is present, the horse is usually positive to upper limb flexion that is isolated to the stifle joint.

Horses with SCL in the stifle commonly improve 50% or more with intra-articular analgesia of the joints. It is important to evaluate these horses within 15 minutes of performing the intra-articular analgesia and after 30 minutes to get a full appreciation for the improvement in lameness. In older horses that have been in training and competition, it is particularly important to perform intra-articular analgesia to prove the significance of an SCL because if a horse has been working well with the presence of an SCL, other factors may be involved in the lameness. In some of these cases, in which the horse has an SCL but it is not the source of pain, the horse may have an intact subchondral bone plate.

Imaging and Diagnosis

Radiography

Radiography is standard for accurate diagnosis (Figure 5.127). In a grading scheme for SCLs developed by Howard et al., type 1 lesions are less than 10 mm in depth and typically dome shaped; type 2 lesions are greater than 10 mm in depth and either dome, conical, or spherical shaped; and type 3 lesions are flat or irregular bone surface.[25] Wallis et al. modified the grading scheme to include type 2A, which are more than 10 mm deep and have a lollipop or mushroom shape with a narrow cloaca and a round cystic lucency; type 2B lesions are more than 10 mm deep with a large dome shape extending down to a large articular surface defect; type 3 lesions are condylar flattening or small defects in the subchondral bone, usually noted in the contralateral limb to that of the clinically significant SCL; and type 4 lesions are those that have a lucency in the condyle with or without an articular defect but no radiographic evidence of a cloaca in the subchondral bone plate (Figure 5.127).[60]

A grading scheme for characterizing presale films in yearlings and 2-year-old Quarter horses has been developed that describes lesions in general.[5] In yearlings that do not show clinical signs, it is important to follow the progression of the lesions with radiographs over time to monitor the SCL, because some may resolve. Likewise, they should be monitored clinically because some may worsen and begin to induce lameness. It is not uncommon in older horses to see radiographic signs of OA such as periarticular osteophytes and joint space narrowing in addition to the cyst. It is also important to compare radiographs in both limbs because SCLs may be bilateral, especially if they are developmental in origin.

SCLs are not uncommon in the proximal tibia (Figure 5.128).[56] In horses younger than 2 years of age, tibial SCLs have been shown to occur in the cranial aspect of the lateral condyle of the tibia, and in horses older than 2 years, they commonly occur in the medial condyle of the tibia, either cranial or caudal. The author's colleagues have seen SCLs of both the medial femoral condyle and the tibia in a single horse. It is also typical in older horses to see OA changes concurrent with these lesions (Figure 5.128).

Nuclear Scintigraphy

The usefulness of nuclear scintigraphy in diagnosing SCL is questionable. It may be helpful for identifying the significance of SCL in the tibia,[56] but it has not been shown to demonstrate high specificity other than for identifying joint disease. Therefore, especially in older horses, it is difficult to rely on nuclear scintigraphy to determine the significance of an SCL because it cannot be used to discern an SCL from generalized joint disease, such as OA.

Ultrasonography

Ultrasonography helps to evaluate articular cartilage surface, joint effusion, debris, and changes that may occur in the meniscus and cruciate ligaments. SCLs

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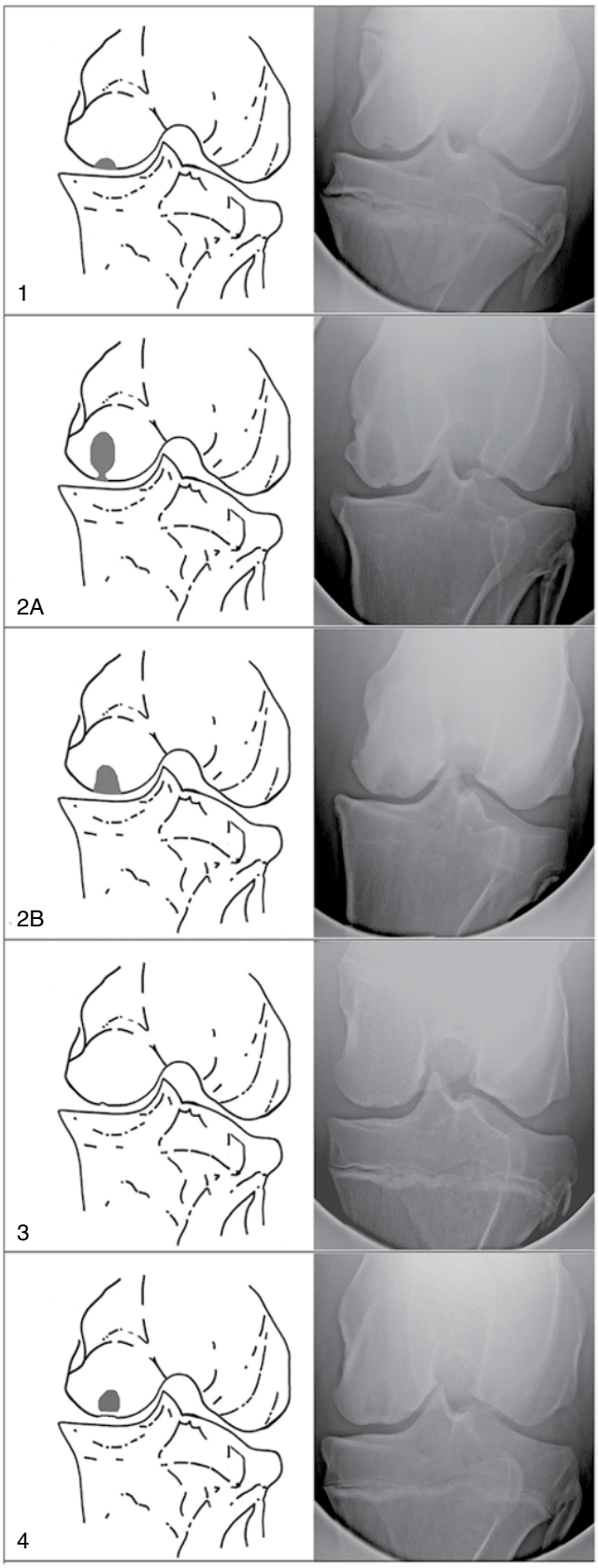


Figure 5.127. Grades of subchondral cystic lesions (SCLs). *Source*: Reprinted with permission from Wallis et al.[60].



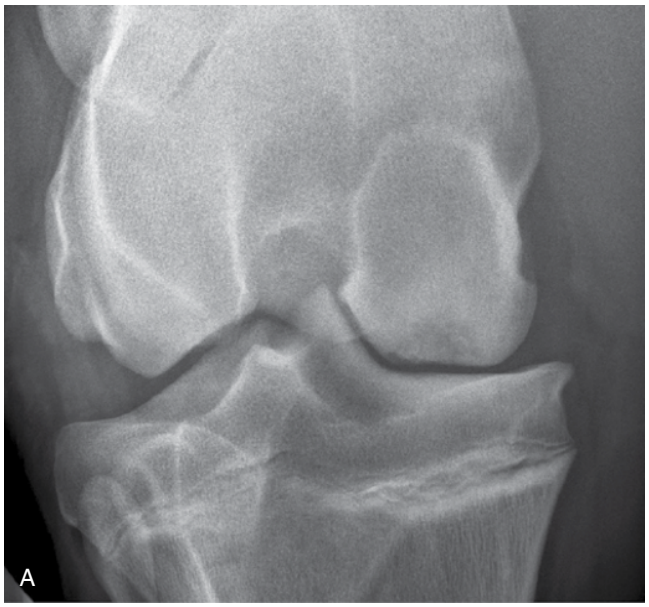
Figure 5.128. A caudal to cranial radiographic image of a proximal tibial subchondral cystic lesion with concurrent signs of osteoarthritis within the MFT joint.

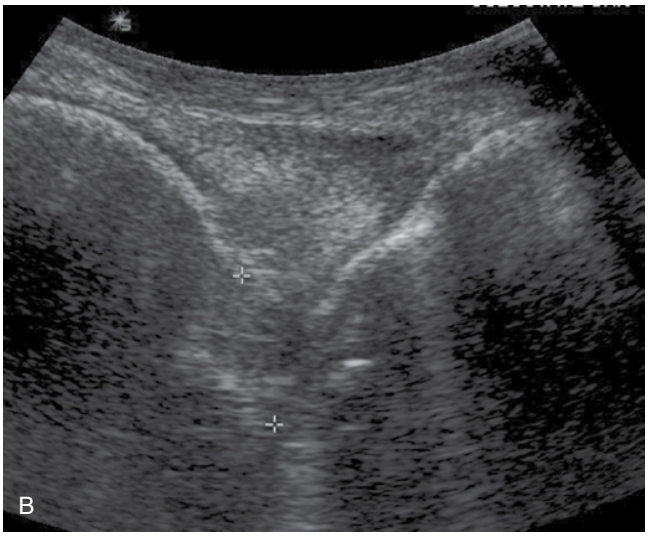
typically show an irregularity on the condylar surface and thickening of the articular cartilage (Figure 5.129). Because most cases of SCL are examined arthroscopically for treatment, the author does not routinely perform ultrasound of these cases unless there is concern about the integrity of the medial meniscus. However, if surgery is not an option at the owner's request, then ultrasound examination is recommended to obtain further information about the integrity of the surrounding articular cartilage and to later guide cyst injection. The femoral condyle becomes quite superficial beneath the skin when the limb is flexed, and the smooth, anechoic nature of the articular cartilage on the curved condyle can be easily appreciated superficial to the hyperechoic subchondral bone (Figure 5.129B). In cases of SCL, a defect is appreciated in the subchondral bone, the depths of which sometimes cannot be appreciated.[27] See Chapter 3 for further details on the use of ultrasonography to evaluate the MFT joint.

Treatment

Conservative therapy can be used in horses younger than 1 year old in which lameness may or may not be present. These horses may be treated with intra-articular medication if lameness is present. Corticosteroids in particular may have an effect on the cystic lining to prevent cyst enlargement, when physically injected into the cyst lining. Whether intra-articular administration of corticosteroids would have the same affect is unknown. Young horses with SCLs should be confined to a small

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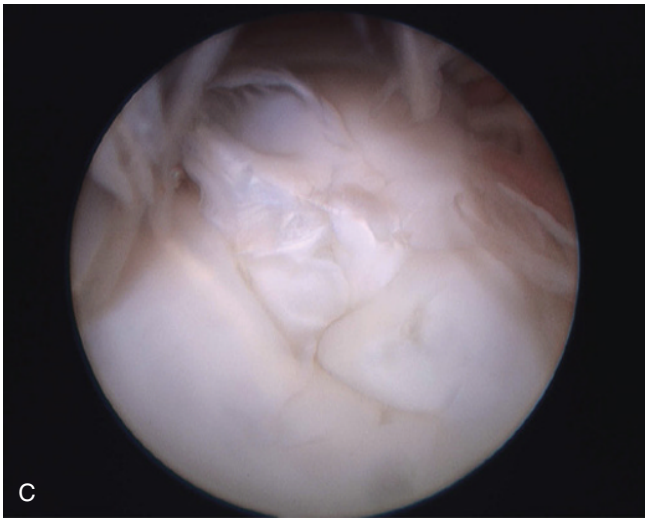


Figure 5.129. Radiograph (A), ultrasound (B), and intraoperative (C) images demonstrating an SCL that involved a large percentage of the articular surface. Note the joint space narrowing on the radiographic image (A) and the presence of the SCL as demonstrated by a defect in the subchondral bone on the ultrasonographic images (B). Thickening of the articular cartilage can be seen arthroscopically (C).

pen, and radiographs reevaluated every 4–6 weeks. If no change is present or if the cyst is enlarging, then surgical options should be considered. However, there are some horses of various ages that respond well to intra-articular and systemic medications and in some instances may be maintained for competition. Cases that respond to medical management have SCLs that are either small, have a small articular component, or may show limited communication between the cyst and the joint surface. The latter may demonstrate an intact subchondral bone plate. It is unknown whether the SCL is relatively stable in these cases, leading to adequate support of the articular surface. Rest has also been reported to be successful in treating some cases of SCL in the tibia[56].

Surgery is a common therapy for SCLs because horses that suffer lameness from this problem can rarely perform with the existence of the cyst.[31] Several methods of surgical treatment have been used with varying degrees of success, including debridement of the cyst contents, injection of the cystic lining with corticosteroids, grafting of the debrided cyst with various products, and transcystic screw placement.

Debridement of cystic contents has been shown to be effective in 56%–77% of cases that undergo surgery. In these cases, the joint is arthroscopically explored, and the contents of the cyst are removed. It is important to remove the lining of the SCL because it contains inflammatory mediators.[45] Some have advocated drilling of the subchondral bone to facilitate access of blood supply to the cyst; however, Howard et al. showed that in some of these cases, the drill holes lead to enlargement of the lesions.[25] Nevertheless, some advocate using microfracture on the cyst wall, especially when sclerosis is present. These horses are typically rested 6–12 months after surgery.

The prognosis generally is good for these lesions. Lewis has shown that 72% of horses had returned to intended use, although 14 of the 67 horses studied had occasional lameness that was managed medically.[29] Howard et al. showed that although 74% were successful,

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that the prognosis was relatively lower for Quarter horses compared with other breeds.[25] Sandler et al. showed that 77% of 150 Thoroughbreds intended for racing raced after debridement of the SCL. However, horses in which more than 15 mm of the surface of the joint was involved had a reduced prognosis compared with those with less than 15 mm.[49] Therefore, it appears that the depth of the cyst is of little importance in the prognosis but the percent of joint surface involved is important (Figure 5.129).

An osteochondritis dissecans (OCD) lesion may be present at the cyst opening and require debridement at the time of surgery.[25] Smith et al. showed that horses older than 3 years had a dramatically lower chance of soundness and return to work than horses 3 years and younger.[54] This study also confirmed that the presence of articular cartilage lesions within the MFT joint remote to the site of the SCL negatively impacted the prognosis. In addition to age and other articular lesions, SCL enlargement[25] and damage to the menisci and/or meniscal ligament can further reduce prognosis.

Some have advocated bone grafting to facilitate repair and filling in the bony defect. However, the prognosis is really no different than debridement.[26] Others have advocated the use of some form of bone graft (cancellous bone graft, tricalcium phosphate, or hydroxyapatite) with an overlay of fibrin, growth factors, and chondrocytes or stem cells to improve the prognosis.[19,33]

SCLs of the tibia that occur laterally can be approached with an arthroscope and are typically beneath the cranial ligament of the lateral meniscus. They too can be debrided. However, in some lesions, especially those involving the medial tibial condyle of older horses, an extra-articular approach may be required for debridement.[56]

Some surgeons advocate intralesional steroid therapy for treating SCLs. Based on the work of von Rechenberg et al., in which they showed the presence of inflammatory mediators within the cyst lining, this treatment makes sense.[45] However, it is important to inject corticosteroids into the cystic lining and not just into the depths of the cyst. Cyst injection is best facilitated by an arthroscopic approach[60] so that concurrent articular cartilage lesions can be debrided if necessary, and successful injection is often demonstrated by the extrusion of fibrinous material from the cyst. However, injection of the lesion can be done with ultrasound guidance of a needle into the cystic material. In the author's opinion, this technique is more difficult in assuring that the lining itself is actually being injected. Triamcinolone acetonide (10–30 mg/cyst) or methylprednisolone acetate (40–80 mg/cyst) or both are typically injected into the SCL. These horses are rested postoperatively and re-radiographed after 2 months, at which time clinical signs often improve and the horses can be put into light work.

Wallis et al. showed that this technique was successful in 67% of horses and 77% of cysts.[60] There was no significant influence of age, 90% of horses that had unilateral cysts were successful vs. 67% that were affected bilaterally, and the presence of osteophytes within the MFT joint reduced the success to 63% vs. 87% for those without.

The latest technique being advocated for treatment of SCL is placement of a screw across the cyst. In a retrospective study, Santschi et al. demonstrated that approximately 75% of horses responded successfully to the procedure.[51] Of note in this study is a reduced prognosis in horses older than 3 years, similar to other studies.

Prognosis

Overall, 56%–77% of horses returned to their intended use with surgery for SCL of the medial femoral condyle. A couple of studies have now demonstrated the reduced rate of return to work for horses over 3 years of age, regardless of treatment, and this should be taken into consideration when treatment options are given. The limited work in tibial cysts has shown that in horses younger than 2 years, 3 out of 4 returned to work. Older horses with tibial SCLs had reduced work. The prognosis was poor for those with lesions in the caudal aspect of the joint in which access was limited.

FRACTURES

Etiology

Intra-articular fractures of the stifle usually are due to acute trauma from internal forces as it is uncommon to see these problems in horses with outward signs of external trauma. They likely occur with rotation and may involve an avulsion fracture, usually of the intercondylar eminence of the tibia. However, the cruciate ligament insertion is typically lateral to the common site of fracture, and many fragments do not involve the ligament.[48,61] Intra-articular fractures of the femorotibial joints are rare, and horses with medial intercondylar eminence (MICET) fractures commonly have a history of acute lameness and traumatic event. It is hypothesized that fractures of the MICET are due to lateral movement of medial femoral condyle against the MICET.[61]

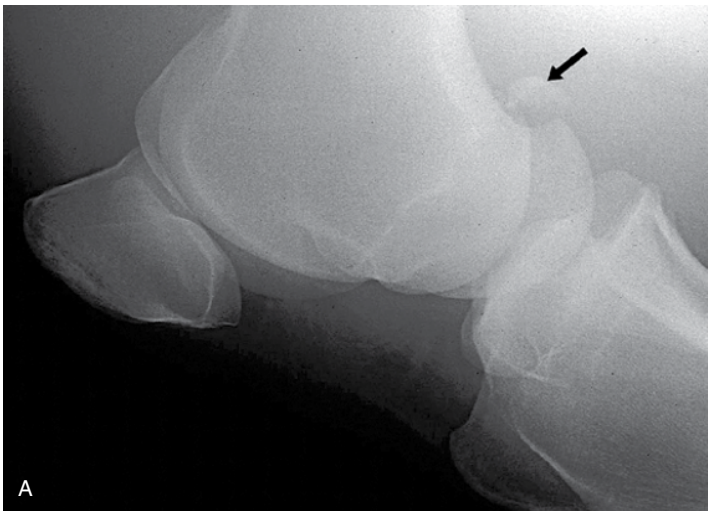
Clinical Signs

Horses typically present with significant acute lameness, often with significant synovial effusion and joint capsule swelling. They may or may not be painful on palpation, and they often show an outward rotation of the distal limb when standing. Horses are typically very painful to flexion, both passively and with movement. Horses with tibial tuberosity fractures can be painful on palpation of the area and are mentioned here because concurrent damage to the soft tissues of the femorotibial joints has been reported with this fracture.[2]

Diagnosis and Imaging

Radiographs typically demonstrate the presence of fractures. They can occur on the caudal aspect of the medial femoral condyle (Figure 5.130A),[10,21] caudal aspect of the medial tibial condyle, intercondylar eminence (Figure 5.130B and C),[37,61] Salter–Harris type III fracture of the lateral condyle,[20] Salter–Harris type IV fracture of the distal femoral physis,[20] or the origin of the long digital extensor muscle (Figure 5.131) or peroneus tertius muscle[6,24] and as loose fragments within the lateral femorotibial joint.[55]

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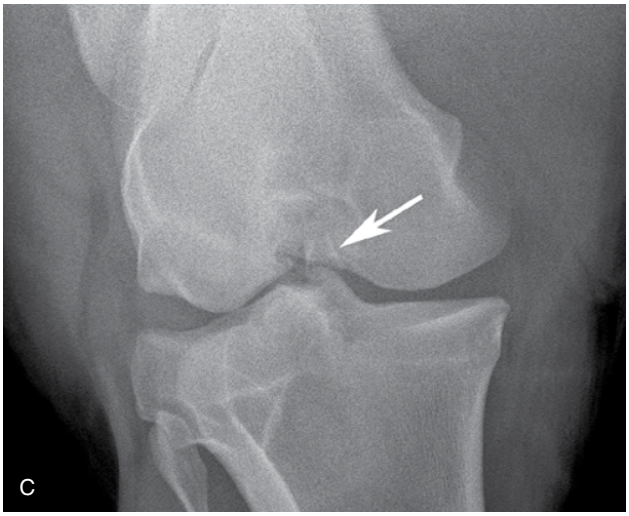


Figure 5.130. (A) Flexed lateral radiograph identifying a fracture of the caudal aspect of the medial femoral condyle (arrow). (B) A flexed lateromedial radiographic image that demonstrates fragmentation of the intercondylar eminence of the tibia (arrow). The fragment was attached to the cranial cruciate ligament, but was loose during arthroscopic examination. (C) A caudal to cranial radiograph demonstrating fragmentation of the intercondylar eminence of the tibia (arrow).

Peroneus tertius origin avulsion fractures usually induce flexion of the stifle with corresponding extension of the hock, and fragments are usually contained within the femoropatellar joint. Fragmentation of the origin of the long digital extensor muscle can induce lameness (Figure 5.131), although the author has seen these as incidental lesions on routine radiographs of yearlings. Fragments can be removed arthroscopically, and the prognosis is good. Radiographs can confirm the diagnosis, and ultrasound is sometimes useful in identifying the exact location of the fragment. Fragments originating from the MICET are thought to be avulsion fractures from the insertion of the cranial cruciate ligament; however, some fragments in this location do not involve the ligament insertion.[48] Walmsley demonstrated that the cranial cruciate ligament insertion was not involved in 7 of 12 horses with MICET fractures. Cranial ligament of MM can also be damaged. Ultrasound[48] examination is valuable in assessing prognosis, which can be worsened by significant secondary damage to soft tissues of the joints.[2]

Treatment

Small intra-articular fragments can be excised and removed arthroscopically in most cases. It is important to explore the entire joint, both front and back, because fragments and debris can migrate to the caudal aspect of the joint, and to determine if secondary damage to the soft tissues (such as the meniscus and the articular cartilage) is present. Proliferative lesions can be found in the intercondylar area of the tibia and femur, which can be debrided if they are clinically significant. Specifically, fibrous proliferation of the soft tissues can be present in chronic cases, as can bony proliferation that may impinge on soft tissue structures with movement. Large fragments in the MICET that have a stable fibrous adhesion often can be left intact. If the fragment is large and involves a large portion of the intercondylar eminence, then lag screw fixation can be used to stabilize the area.[61]

Fragment removal should be performed if there is no compromise to the weight-bearing aspect of the joint; however, internal fixation is recommended if the joint

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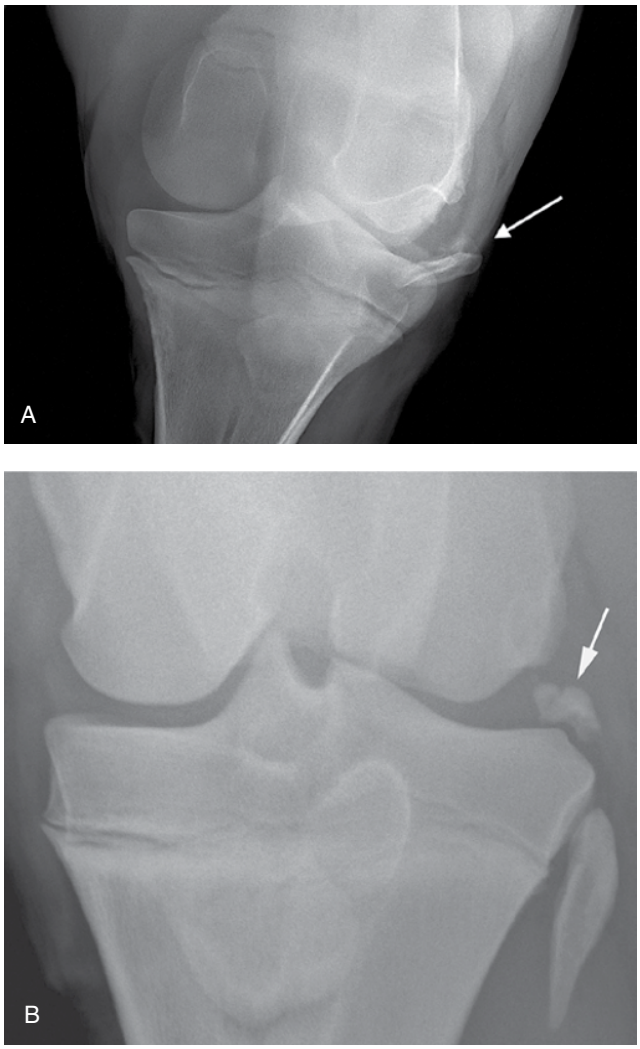


Figure 5.131. Caudal to cranial radiographic images of acute (A) and more chronic (B) fragmentation of the origin of the long digital extensor tendon (arrows).

surface needs to be reconstructed and/or vital attachments need to be retained.[61] For the condylar surface, a cannulated screw can be used to stabilize a fracture of the medial femoral condyle. Conservative therapy is sometimes best in horses with Salter–Harris fractures.[20]

Prognosis

Prognosis is often good for horses with fragmentation in which minimal soft tissue debridement occurs and minimal secondary damage is present.[48] For MICET fractures, 76% were sound after removal.[48] The prognosis is reduced for horses with larger fracture fragments involving more of the articular surfaces and for those with significant soft tissue and articular cartilage damage.[48]

FEMORAL CONDYLE LESIONS

Etiology

Femoral condyle lesions fall into two broad categories based on the age of the horse: septic or developmental

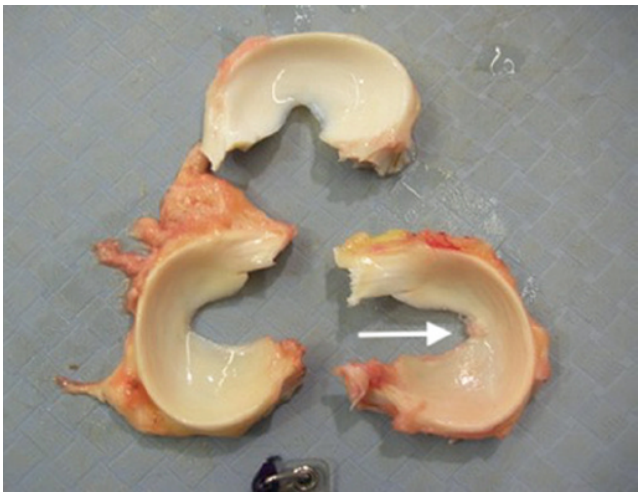


Figure 5.132. Menisci from a 5-year-old cutting horse that had chronic left medial femorotibial pain (Bottom) and a 9-year-old Warmblood with no history of femorotibial pain for comparison (Top). Both bottom menisci appear to lack substantial axial integrity, with the right meniscus showing axial damage and striation (arrow) compared with the normal top meniscus.

lesions in foals and osteochondral disease in adults. In foals, Hance et al. showed that three separate categories of femoral condyle lesions can occur.[21] Type I lesions are septic, and subchondral bone lysis is present in the face of septic arthritis and osteomyelitis. Type II lesions show osseous irregularity in less than 50% of the femoral condyle surface. Type III lesions show widespread irregularity and some have a thin osseous fragment. The authors of the study concluded that type II and III lesions may be some form of developmental orthopedic disease or vascular insult.

Osteochondral lesions of the femoral condyles in adult horses nearly always involve the medial femoral condyle. These lesions are usually considered to be traumatic in origin; however, there is growing concern that some of the lesions may be developmental in origin because abnormalities in condylar shape, such as dimpling or flattening, may predispose horses to this problem. However, a postmortem study showed no correlation of MFC flattening to OA,[13] and Barret et al. showed no correlation of flattening to performance in cutting horses at 3–4 years of age.[5] However, the effect of meniscal integrity on development of articular cartilage lesions is unknown, and considering how meniscal damage can affect prognosis for various lesions within the MFT joint and the findings in young horses on postmortem examinations (Figure 5.132), further work is needed.[14]

Clinical Signs and Diagnosis

Foals with type I and III lesions are typically severely lame, and those with type II lesions are usually moderately lame. However, all cause clinical signs that obviously point to a stifle problem. Radiographs typically reveal subchondral bone lucency indicative of the damage (Figure 5.133).

For nonseptic cases, medial femoral condyle osteochondral lesions are common in young western performance athletes; however, they can occur in any type of

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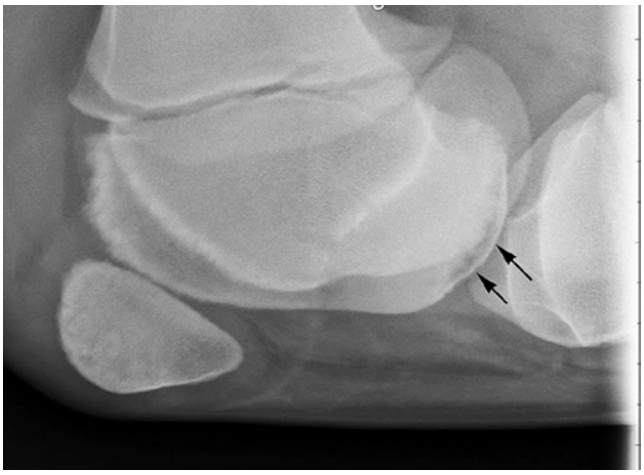


Figure 5.133. Subchondral lysis of the medial femoral condyle (arrows) was present on the lateromedial radiographic image of this foal's stifle. Arthroscopic examination revealed severe articular cartilage and subchondral bone degeneration.

horse. Adult horses with medial femoral condyle lesions may or may not have effusion, are usually positive to hindlimb flexion, and respond to intra-articular analgesia. Although these horses may not respond 100%, anything over 50% improvement is often considered significant.[29,52]

Radiographs are commonly unremarkable or only show mild OA changes in these horses. Flattening and sclerosis of the medial femoral condyle are subtle radiographic abnormalities that may suggest osteochondral lesions.[52] However, although flattening is a common finding, its clinical significance on performance is limited.[5] Ultrasound is essential to best characterize lesions, especially to the soft tissue structures such as menisci, meniscal ligaments, articular cartilage, and periarticular tissues. CT with and without contrast can be helpful to characterize lesions, and MRI is growing in use, allowing for visualization of most all tissues. Correlation of imaging findings to disease site, severity, and prognosis is contentious.

For horses with equivocal imaging results that block to the femorotibial joints, who may or may not have responded to medication, arthroscopic visualization can be beneficial to further characterize the lesions. A standing needle scope procedure has been reported and found useful for characterizing site and severity of lesions.[17] Therapeutic procedures are often limited with this approach, although it can be helpful in determining the need for further conservative versus surgical therapy.

Treatment

Arthroscopic surgery in foals should be preceded by obtaining a synovial fluid sample for culture and sensitivity. Debrided bone also may be submitted. Care should be taken during debridement because the normal subchondral bone of foals is soft and easily removed. Therefore, it is difficult to assess the extent of damage.

Arthroscopic findings in adults usually include focal or generalized articular cartilage lesions (Figure 5.134). Focal lesions are usually dimples, wrinkles, or folded pieces of articular cartilage that may or may not be detached from the subchondral bone.[52] Articular cartilage fibrillation, erosion, or subchondral bone lysis also can be involved.[9,29]

Walmsley has classified the types of articular cartilage lesions using human classification systems and in particular uses the Outerbridge classification for reporting. The Outerbridge system grades the lesions on a scale of 1–4: grade 1 includes softening and swelling of the articular cartilage, grade 2 includes partial-thickness defects with fissures on the surface that do not extend to the subchondral bone and/or do not exceed 1.5 cm in diameter, grade 3 includes fissuring to the subchondral bone and/or a lesion that exceeds 1.5 cm in diameter, and grade 4 includes those with exposed subchondral bone.

Schneider et al. reported that 6 of 7 horses with focal disease returned to intended use, whereas only 1 of 4 with generalized condylar damage recovered.[52] Lewis stated that only 6 of 20 horses returned to intended use.[29] Walmsley et al. found that in horses with Outerbridge grades 1 and 2, 78% returned to their intended use, compared with 63% of those with Outerbridge grades 3 and 4. In the study by Cohen et al., 60% of horses improved, of which 46% became sound and 37% returned to intended use. In general, however, the severity of articular cartilage lesions was an inconsistent indicator of prognosis (Figure 5.134).[9,30]

There is no consensus on how best to treat these cases. In horses with fibrillation, the fibrillated tags of tissue should be debrided. For those with partial-thickness cracks and erosions, only superficial debridement should be performed. Some surgeons have proposed using radiofrequency probes to shrink the involved areas of cartilage to cause them to recede and theoretically alleviate loading on the area. This procedure is untested, and in human medicine is contentious, because the radiofrequency procedure has been shown to cause chondrocyte death. Lesions with subchondral bone lysis can be lightly debrided. Some cases of full-thickness erosion have visible evidence of subchondral bone necrosis that require debridement. Some surgeons advocate the use of microfracture to augment healing; however, in the medial femoral condyle, this procedure could lead to the development of SCL. For the micropick procedure, care must be taken to remove all calcified cartilage as failure to do so can result in poor healing.[32] The use of mesenchymal stem cells along with the microfracture procedure has been found to be useful.[16,32]

It is currently unknown why medial femoral condyle lesions occur. Although some think that they may be developmental in nature, acute trauma and/or chronic repetitive trauma cannot be ruled out. There is also concern that flattening or dimpling of the subchondral bone may predispose these horses to this injury. Contino showed that these lesions are common in young Quarter horses,[5] but there has been no correlation in clinical studies.[5] Scott et al. found that horses with flattening or mild lysis are likely to have articular cartilage lesions that require debridement.[53] Of those horses, 7 of 9 with focal lesions were sound postsurgery compared with 2 of 6 with generalized lesions. Care must be taken, however, because overly aggressive debridement and microfracture may lead to secondary SCL formation.

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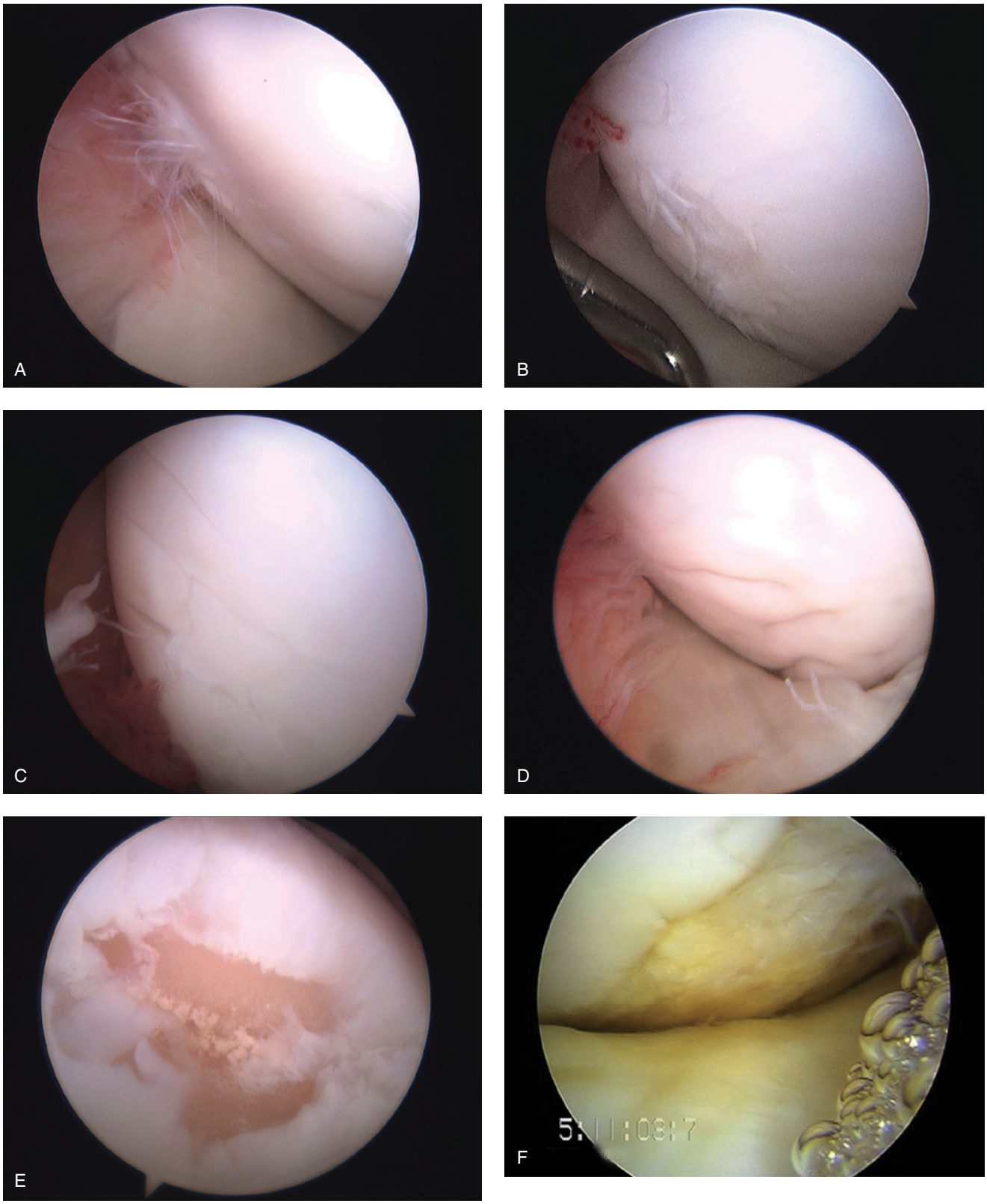


Figure 5.134. Various articular cartilage lesions that can be seen arthroscopically in adult horses. Small focal area of cartilage fibrillation (A) or partial-thickness cracking can be seen (B). More extensive cracking, some of them full thickness, can also be present (C). More extensive cracks can also contain soft, thickened articular cartilage in between the cracks (D), and in some cases, dead, necrotic subchondral bone can be present (E). In advanced cases of OA, extensive articular cartilage erosion can be seen as well as fibrocartilage healing (F).

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Prognosis

The prognosis for foals with type III lesions is guarded, even with surgical debridement; 11 of 20 horses in one study were euthanized due to poor prognosis.[21]

As noted under the treatment section, the prognosis for medial femoral condyle lesions in adults depends on the severity of articular cartilage and associated soft tissue damage and must be estimated on a case-by-case basis.

COLLATERAL LIGAMENT INJURY

Etiology

Collateral ligament injury primarily occurs to the medial collateral ligament in the femorotibial joint, usually in adults. It is not uncommon to see that other structures are involved, including the meniscus and the cranial cruciate ligament.[7] These injuries are usually acute in nature, although chronic low-grade damage to the ligament may be seen in cases of chronic intra-articular disease.

Clinical Signs

Clinical signs are usually acute and severe, but minor injuries cause more subtle signs. Often there is swelling in the medial collateral ligament area and pain on palpation. The horses are usually very positive to flexion and worsen after manipulating the limb into a valgus position. This involves placing pressure over the lateral aspect of the femorotibial joints with one hand and pulling the limb laterally to stress the medial collateral ligament. Horses with complete rupture may show significant lateral movement of the distal limb and a palpable widening of the MFT joint space on the medial aspect of the stifle.

Diagnosis

Radiographic findings often are normal; however, the MFT joint may distract on a stressed caudocranial view (Figure 5.135). Signs of OA, such as an enthesophyte, in the area of attachment in the medial collateral ligament are not uncommon in more chronic cases. Ultrasound is usually the primary method of diagnosis, which often shows fiber disorganization either in the body or the insertions of the ligament.

Treatment and Prognosis

Treatment is usually conservative in nature and may include rest, intra-articular medication, topical anti-inflammatory medication such as diclofenac acid, ESWT at both the body and the insertions of the ligament, or stem cell therapy into any hypoechoic region of the ligament. Surgical therapy has been advocated in some cases. The prognosis is often poor overall, except in horses with mild injuries to the ligament.

CRUCIATE LIGAMENT DISEASE

Etiology

Damage to the cranial cruciate ligament is usually acute in nature due to a traumatic event in which the limb is stressed in an abnormal direction, although the cause is rarely recognized. Other structures are usually involved, including the medial meniscus and the medial collateral ligament. Partial degeneration of the cranial cruciate ligament can occur in jumpers and racehorses. Horses with these lesions are less lame and tend to have a better prognosis with treatment.



Figure 5.135. Caudal to cranial radiographic image of a stifle with a ruptured medial collateral ligament. Notice the widened joint space medially on this stressed view.

The types of stresses that induce this problem are truly unknown. In an *ex vivo* study by Rich et al., in which mechanically induced tearing of the cranial cruciate ligament was assessed, 9 of 15 limbs failed in the ligament, 5 failed at the tibial insertion, and 2 failed at the femoral origin. This study suggested that there is no consistent change due to the type of injury that occurs.[46]

Clinical Signs

The degree of lameness is often associated with the degree of damage, and most horses present for an acute lameness, with significant effusion and response to flexion. However, in an attempt to mechanically assess the joint using the cranial drawer test, Prades et al. found that only 1 of 10 horses was deemed to be unstable in the standing position and 6 of the 10 were deemed to be unstable under anesthesia.[43] A tibial thrust test in the caudal direction may worsen the lameness in some horses, but this is not considered specific for cranial cruciate ligament damage.

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Diagnosis

Radiographs are usually unremarkable unless the origin of the ligament avulses from the intercondylar fossa,[15] a midbody tear shows dystrophic mineralization, or a medial tibial eminence fragment is present. However, the latter finding is not pathognomonic for cranial cruciate ligament damage because a fracture at the apex of the eminence often does not involve the ligament. Ultrasound gives variable results because the cranial cruciate ligament area is difficult to image; however, a small section of the cranial cruciate ligament insertion on the tibia can be visualized with the limb flexed. Contrast-enhanced CT has some merit if the ligament tear communicates with the joint.[38] MRI is available in limited locations and probably demonstrates the best chances of acquiring an accurate diagnosis. High-field, short-bore magnets can accommodate some horses for accurate imaging. Low-field scanners are gaining favor and have shown promise for accurate diagnosis.[35] Arthroscopic surgery can be used to explore the joint and assess the extent of damage and is currently the best method to accurately diagnosis cranial cruciate damage.[39,42]

Treatment

Osteochondral fragments from the tibial eminence can be removed and damaged tissues debrided arthroscopically. Microfracture may help stimulate endogenous repair of avulsed ligament from bone in the intercondylar space. Damaged tissue can be debrided, and although no repair technique has been successfully developed for the horse, partial tears can be augmented with platelet-rich plasma (PRP) or mesenchymal stem cells injected into the tissues or joint.[39] Other therapies to consider include intra-articular stem cell or IRAP therapy to use the growth factor properties that these two treatments are meant to induce. Overall, the prognosis depends on the extent of damage, but is often considered poor. However, some horses have returned to work.[29,39,43] Caudal cruciate ligament injuries have been reported, and most of them involve longitudinal fraying and not true ligament rupture.[3,8,36,41,47]

MENISCAL INJURIES

Etiology

Meniscal lesions can be acute or chronic. Acute damage can occur with a bad step or an accident that leads to shifting or shear forces between the femur and tibia. Lesions are often seen in other structures such as cruciate ligaments and articular cartilage. Horses of any breed and use are susceptible to these lesions. Degenerative lesions of meniscus are seen to associate positively with articular cartilage degeneration and osteophyte scores.[14] Pathogenesis is unknown. Both lateral and medial meniscal lesions occur, but damage to the medial meniscus is most common.[14]

Clinical Signs

Meniscal injuries are acute or insidious in onset and usually affect the medial meniscus. Signs of medial meniscal injury can vary. In a study by Walmsley, 40% of cases presented acutely, and 53% were insidious in onset.[65] The median lameness grade was 3 out of 10, 66% were positive to upper limb flexion, 47% had MFT joint and/or femoropatellar effusion, and 93% responded to intra-articular analgesia.[65] However, meniscal injury can be secondary to chronic OA or SCLs, and signs can include capsule thickening of the MFT joint and disuse atrophy of the surrounding musculature.

Diagnosis and Imaging

Radiographs can be normal in acute cases of primary meniscal damage. However, damage to the meniscus may lead to joint space narrowing, so it is important that these images be reassessed because poor positioning can lead to false interpretation of the radiographs.[58] In addition, radiographic abnormalities often accompany meniscal tearing in more chronic cases, as has been shown in several studies[4,12,62] Mineralization of the meniscus (Figure 5.136) and osteophytes on the medial tibial plateau (Figure 5.137) and intercondylar eminence of the tibia are common in horses with more chronic lameness. In updated results from Walmsley,[64] 23% of horses with meniscal damage had new bone formation at the medial intercondylar eminence of the tibia, and this was associated with cranial cruciate injury in only 7% of cases.[34]

Ultrasonographic findings can include characterization of an abnormal size, thickness, variability in echogenicity, location of the meniscus, prolapse of the meniscus medially, and overt meniscal tearing.[4,8,12,23] It is important when assessing for a tear that the area is imaged in both weight-bearing and non-weight-bearing stances (Figures 5.138 and 5.139). Non-weight-bearing imaging often causes opening of the meniscal tear for better demonstration of the defect. For acute severe injuries, the cranial cruciate ligament and medial collateral ligament may also be involved, but it is not uncommon to see isolated meniscal injuries. Despite its widespread use to diagnose meniscal lesions, it is difficult to validate

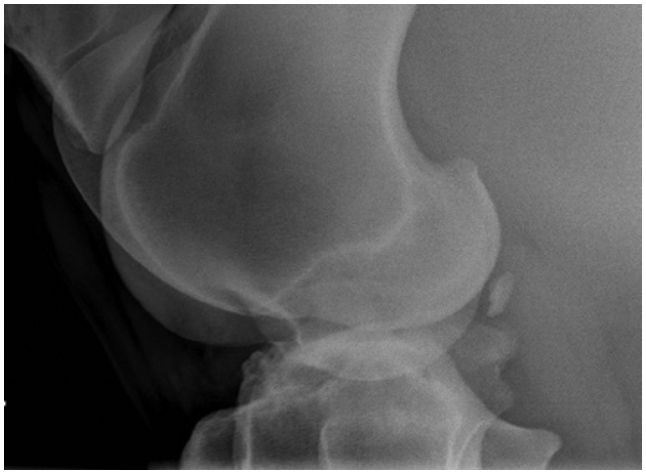
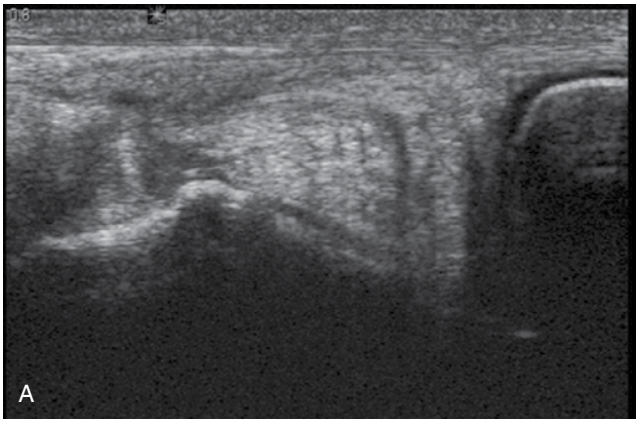


Figure 5.136. A lateromedial radiographic image demonstrating mineralization of the caudal aspect of the meniscus. Arthroscopic removal of this mineralization is rarely indicated because the OA is often advanced.

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Figure 5.137. A caudal to cranial radiographic image showing osteophytes on the medial aspect of the tibia and the intercondylar eminence, which are common in many types of diseases involving the femorotibial joints.



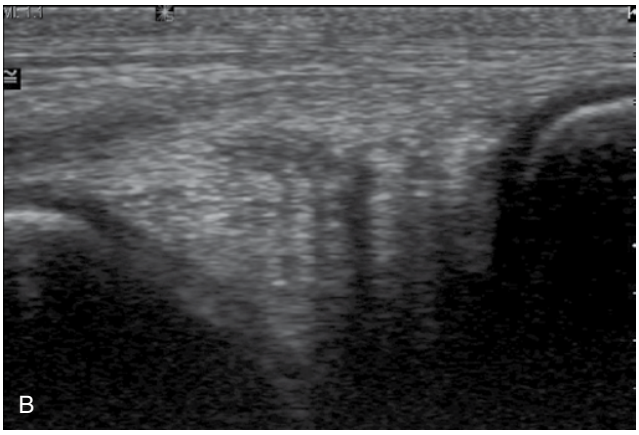


Figure 5.138. Ultrasonographic images of the medial meniscus in both weight-bearing (A) and non-weight-bearing positions (B). Notice how the meniscal tear becomes more apparent in the non-weight-bearing position.

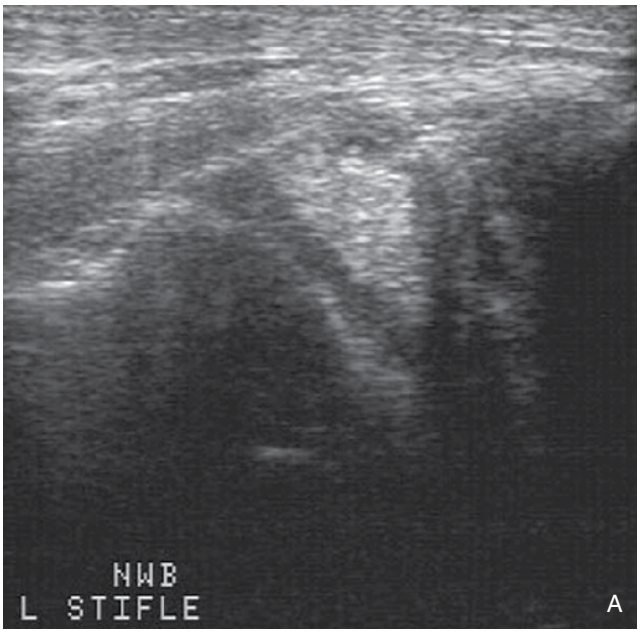




Figure 5.139. Ultrasonographic image (A) of a severe meniscal tear that correlated well with the gross appearance (B). *Source*: Courtesy of Dr. Laurie Goodrich.

ultrasonographic findings since most of the meniscus is inaccessible at surgery, leading to potential false-positive and/or false-negative findings.

Other diagnostic techniques include nuclear scintigraphy, which has poor sensitivity and modest specificity for stifle disease in general;[4,18] MRI, which is useful but limited in availability; contrast CT, which requires communication of the tear with the synovial cavity; and arthroscopy, which is an important diagnostic tool that is readily available but unable to visualize much of the weight-bearing surface of the meniscus. Therefore, the lack of findings of a meniscal lesion with arthroscopy does not necessarily rule out the absence of a lesion.

Treatment

Treatment usually involves arthroscopic surgery and debridement of the disrupted meniscal fibers that can be

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seen. In addition, it is common to test the looseness of the meniscus with a probe during surgery. The significance of this finding is unknown, but subjectively seems to be associated in some cases with damage and protrusion of the meniscus identified on ultrasound. In addition, manipulation of the meniscus permits better visualization of the tibia for evidence of secondary damage.

Walmsley developed a grading scale for meniscal injuries: grade I is characterized by axial tearing through the cranial ligament of the medial meniscus and into the meniscus, grade II is the same as grade I but with torn tissue and visible extent of the damage, and grade III is a severe tear that extends beneath the femoral condyle.[65]

In an updated review of horses with meniscal injuries, Walmsley found that of 126 cases, 111 were medial and 25 were lateral and 53% were grade I, 28% grade II, and 17% grade III.[63] In that study, 76% had articular cartilage damage, and 14% had damage to the cranial cruciate ligament. Of those with medial meniscal tearing, 45% were sound compared with 75% of those with lateral meniscal involvement. Sixty percent of horses with grade I tearing were sound compared with 65% of those with grade II and 10% of those with grade III. Overall, 51% of the horses were sound with surgery.[63] Similarly, Cohen et al. found decreasing prognosis with increasing meniscal damage, with no horses with grade III tearing returning to previous use.[9] Because most of the meniscus cannot be visualized arthroscopically, a tear or lesion in the body of the meniscus may be treated by extracapsular injection of stem cells or PRP into the lesion(s) or into the joint. Horses with meniscal lesions treated with bone marrow derived stem cells returned to work more often than those historical controls.[16] If the tear is visible arthroscopically, then various suturing and staple techniques are also available.[40] In general, suturing is preferred over stapling. The efficacies of these latter techniques to treat meniscal lesions are currently unknown.

Prognosis

As noted above, the prognosis for lesions of the medial meniscus is relatively guarded, but for the lateral meniscus can be quite good. In addition, horses with small lesions that can be debrided can do relatively well, although horses with significant tearing carry a poor prognosis. Great efforts are currently being made to provide augmentative therapies, such as stem cells, which have shown some promise.[16]

SYNOVITIS/CAPSULITIS/OA

Etiology

Horses that undergo chronic repetitive stress to the hindlimbs, such as young western performance horses, are susceptible to synovitis in the femorotibial joints, especially the medial compartment. This is similar to racehorses that develop synovitis in their metacarpophalangeal and carpal joints. A primary source of synovitis is usually not apparent in these horses. However, all of the diseases described in this section can lead to synovitis and in some cases, ultimately, OA.

Clinical Signs and Diagnosis

Horses with synovitis of the femorotibial joints may not be lame, but have mild to moderate effusion in the MFT joint. These horses may show some response to flexion and a gait that can be described as stiff. Some horses can compete well with this finding, but they should be monitored closely. As mentioned previously, all diagnostic techniques can be negative in these cases, including arthroscopic surgery.

Horses with synovitis secondary to other primary lesions within the MFT joint usually have a history of a short response period to intra-articular medication. However, trainers and owners usually note that the horse either never fully regained its level of performance or constant medication was needed. In these cases, additional diagnostics, including diagnostic arthroscopy, are often required to fully characterize the problem.

Horses with significant OA of the femorotibial joints are usually lame at the walk with loss of muscle mass and soft tissue swelling and effusion of the MFT joint. Concurrent abnormalities such as OA, SCL of the medial femoral condyle, and medial meniscal damage are common (Figure 5.140). Although a history of predisposing injury and disease of the stifle is usually noted, some horses may develop this problem insidiously without a notable injury.

Treatment

Horses with synovitis usually respond well to intra-articular medication, topical anti-inflammatories, systemic medication, and extracorporeal shockwave therapy. These medications are described in depth in Chapter 8. Horses typically respond well to rest or a reduction in training, which are often recommended. However, failure to respond to medication and recurrence of lameness are often the key findings that lead to more intensive imaging and possibly diagnostic arthroscopy.



Figure 5.140. Caudal to cranial radiograph of the stifle of a 6-year-old Quarter horse mare with multiple radiographic abnormalities within the MFT joint (arrows). Concurrent medial meniscal injuries should also be suspected in these types of horses. *Source*: Courtesy of Dr. Gary Baxter.

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Treatment of OA of the femorotibial joints is the same as for other joints. However, arthrodesis is not an option, and clinicians are often forced to try several different treatment options. Exercise in the form of small paddock turnout appears to help, although owners must ensure that another horse does not induce overstressing of the patient. Arthroscopic surgery can help to reduce the progression of OA by removing debris and fibrillated articular cartilage, but it must be stressed to the owners that this is not a cure and the disease process will most likely progress.[16]

Prognosis

Horses with synovitis are easily managed medically as long as a primary disease process is not present. However, there is concern that chronic persistent synovitis can lead to secondary damage to the articular cartilage, which may predispose to the onset of OA in the future. The prognosis for OA of the femorotibial joints depends on severity and appears no different than for other joints in the horse. However, severe OA can lead to significant lameness, making it difficult for the horse to stand, and secondary complications such as decubital ulcers.

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FEMUR AND COXOFEMORAL REGION

Nicolas S. Ernst and Troy N. Trumble

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THE FEMUR

Conditions of the femoral region that contribute to lameness are uncommon and can vary greatly in severity. For example, femoral fractures usually cause significant swelling in the region and severe non-weight-bearing lameness. In contrast, horses with fibrotic myopathy may not be lame but have a gait alteration due to fibrosis of the muscle. Careful palpation and physical examination of the femoral region are often very important in making a diagnosis because imaging of the femoral region can be difficult, especially in adult horses.

DIAPHYSEAL AND METAPHYSEAL FEMORAL FRACTURES

Fractures of the femur are relatively common in horses.[7,14,21,26,36,57] Intra-articular (IA) fractures involving the stifle and coxofemoral joints are described in those respective sections.

Etiology

Foals frequently sustain femoral fractures during initial handling or during halter breaking. Occasionally a mare will step on a foal, or the foal may become trapped under a fence. In a study of 38 horses under 1 year of age, causes included a fall or severe adduction, external trauma, or being caught in a fence.[36] In young animals, fractures often involve the proximal or distal growth plate, and diaphyseal fractures are usually oblique and spiraling.[35] Adults often sustain irreparable comminuted fractures of the femoral shaft from trauma. A common history in an adult is slipping on ice, causing the leg to splay out (adduct), transferring a large force of the body as it falls to the femur. It has also been described that femoral condylar avulsion fractures can occur in horses that had been cast with a sideline for castration without sedation.[88]

Clinical Signs

The obvious sign is non-weight-bearing lameness. When viewed from the side, the affected limb may appear slightly shortened with the hock held higher than the opposite hindlimb; a dimpling of the musculature overlying the fracture may be observed. Obvious swelling may be present, especially in horses with diaphyseal fractures (Figures 5.141 and 2.96). Uncommonly, clinically significant or fatal hemorrhage results, usually due to laceration of the femoral artery.[36,73] These horses will have pale and tacky mucous membranes, and if hemorrhage into the musculature becomes excessive, they can develop a compartment-type syndrome, making them extremely uncomfortable to any movement and palpation of the swollen region.

Diagnosis

Excessive movement of the distal limb is usually present on palpation. Fractures of the distal growth plate are usually evident because of displacement, instability, or swelling in the stifle region. For mid-diaphyseal fractures, moderate to severe swelling is often associated with the fracture site (Figure 5.141), and crepitation may be apparent. A stethoscope may be helpful for detecting crepitation, although sounds may be muffled due to the massive musculature and swelling (e.g. hematoma) or just minimal with severe comminution. Because of overriding, the patella may feel loose and may be manipulated easily in a horizontal direction. If the horse is extremely sensitive to palpation or movement of the limb, compartment-like syndrome should be considered with suspicion of femoral artery laceration.

The clinical appearance of horses with proximal femoral fractures (slipped capital epiphyses or femoral neck fractures) can be similar to those with coxofemoral luxation. If the fracture overrides proximally, the hock on

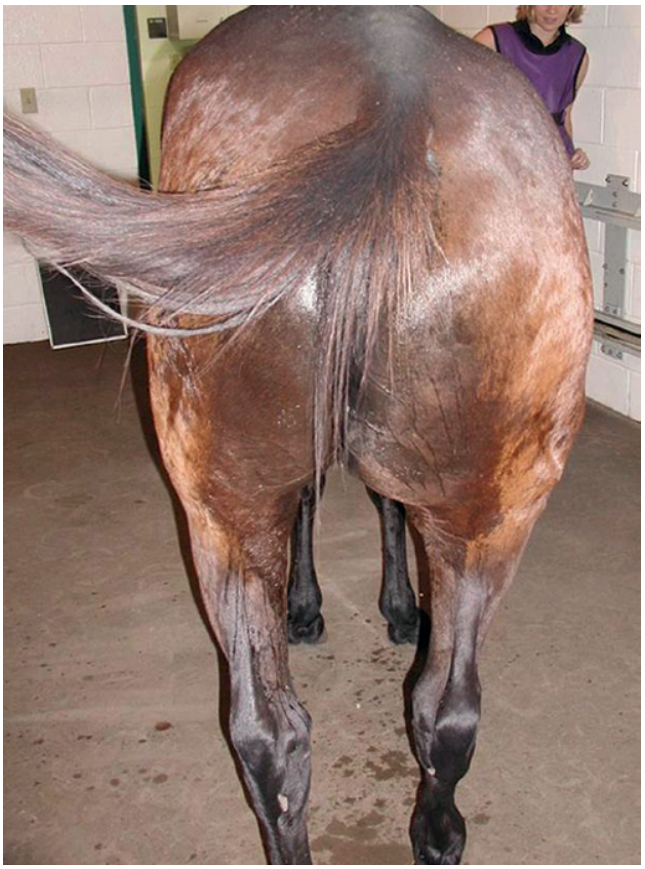


Figure 5.141. Caudal view of an adult with severe swelling of the femoral region associated with a diaphyseal femoral fracture. Notice that the horse is sweating and non-weight-bearing on the right hindlimb.

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Figure 5.142. Image of the hind end of a pony demonstrating a shorter left hindlimb than the right hind. Notice that the point of the hock is higher, the heel does not completely contact the ground, and the limb is externally rotated. Horses with slipped capital epiphyses, femoral neck fractures, or coxofemoral luxations can all have this clinical appearance.

the affected limb may be higher than the hock on the normal limb, with some degree of external rotation of the limb (Figure 5.142). Often the horse will not have any swelling present and may appear to bear weight on the limb, but often will not completely load the heel. If the fracture is several days old, swelling may be palpated on the medial aspect of the thigh. Palpation (± auscultation) over the greater trochanter while the limb is being manipulated may reveal crepitus. If the horse is large enough, a rectal exam may reveal subtle crepitation when the limb is manipulated.

Although physical exam findings may suffice to diagnose a femoral fracture, radiographs are important to make a definitive diagnosis and demonstrate the exact location and configuration of the fracture such that treatment and prognosis can be determined. For the distal one-third of the femur, standing views will usually identify the fracture, but for the proximal two-thirds of the femur, particularly in adults, a recumbent position is required to obtain quality radiographs (Figure 5.143). The most useful radiograph of the proximal two-thirds of the femur is taken medial to lateral with the horse in dorsal recumbency but tipped onto the injured limb (Figure 5.143B). Adequate penetration is difficult for the cranial to caudal projection, but the image may still be useful. A standing view of the proximal femur/coxofemoral region may suffice in certain cases.[56] Digital radiography has significantly facilitated acquisition of quality images of this region, and standing oblique projections of the femoral shaft may demonstrate a lesion almost as well as in an anesthetized horse. The quality of the images often depends on the size of the horse and swelling present; as both increase, the quality will decrease. The suspected gravity of the injury and the anticipated ability for the horse to recover from general anesthesia without further injury must be considered.

Ultrasound may demonstrate cortical disruption to prove that a femur fracture is present when radiographs are not possible or advisable.[29,71] Scintigraphy, particularly for less severe or more chronic injuries, also may localize femoral lesions not amenable to radiographic imaging (Figure 5.144).[83]

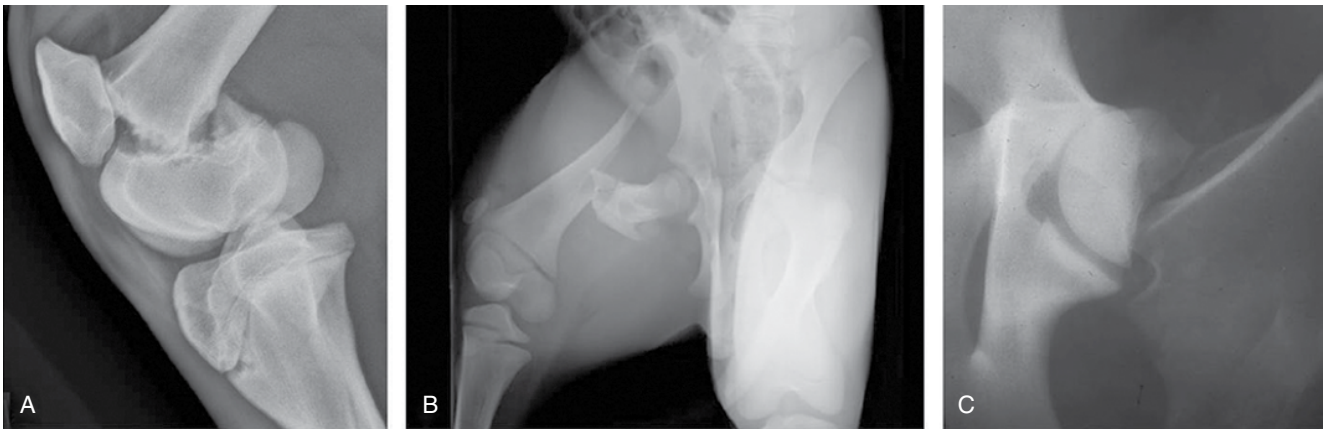


Figure 5.143. (A) Lateromedial oblique view of the stifle of a weanling taken standing, showing a Salter–Harris type II fracture of the distal physis of the femur. (B) and (C) Radiographs taken with the horses in a recumbent position under anesthesia. Image B demonstrates a closed, oblique, mid-diaphyseal fracture of the femur in a miniature foal, and image C demonstrates a neck fracture of the proximal femur (Salter–Harris type II).

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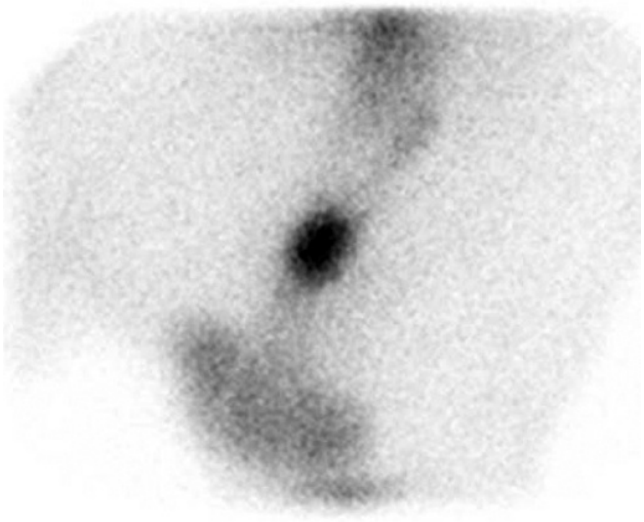


Figure 5.144. Lateral scintigraphic image of the femur in an adult mare who was chronically lame (stifle is at the bottom and coxofemoral joint at the top). Note the increased radiopharmaceutical uptake at the junction of the middle and distal one-third of the femur.

In a series of 38 foals with femoral fractures, 26 had fractures of the femoral diaphysis (Figure 5.143B), and 18 involved two-thirds of the length of the diaphysis.[36] Twelve foals had distal physeal fractures with the most common configuration being Salter–Harris type II (Figure 5.143A). A separate series of 25 foals sustained fractures of the capital femoral physis (Figure 5.145), neck (Figure 5.143C), or greater trochanter.[41]

Treatment

Treatment of femoral shaft fractures depends on the age of the animal and the type and location of the fracture. Generally, euthanasia is indicated for adults that have sustained femoral shaft fractures unless exceptional circumstances exist. If compartment-type syndrome is present, a fasciotomy should be considered to relieve pain while making diagnostic and treatment decisions.

Diaphyseal fractures have been treated with stall rest in foals weighing up to approximately 200 kg[57]; however, misalignment and nonunions are risks in young horses (Figure 5.146). Compression plating is the treatment of choice for foals that have sustained diaphyseal fractures when athletic soundness is desired. Fourteen of 15 foals in which diaphyseal fractures were plated were repaired using 2 plates, 1 lateral and 1 cranial.[36] However, single-plate application has been used occasionally.[7,36] A very stable repair can result when the fracture is midshaft and transverse.[97] More distal fractures can be repaired using angled blade plates or dynamic condylar screw plates.[11,36] The plates are not removed when there are no complications. Fractures without caudal cortical buttress remaining are at risk of an unstable repair and should be given a poor prognosis.[36]

Intramedullary pinning using the stacked pin technique or a single half-inch pin has been used successfully in young foals with femoral diaphyseal fractures.[85] However, problems with pin migration should be expected. One report in experimental fractures in donkeys (65–140 kg) reported some success.[90] However, the work was performed in adult donkeys. Creating controlled, relatively atraumatic fractures in what is really adult bone does not simulate clinical fractures in foals.

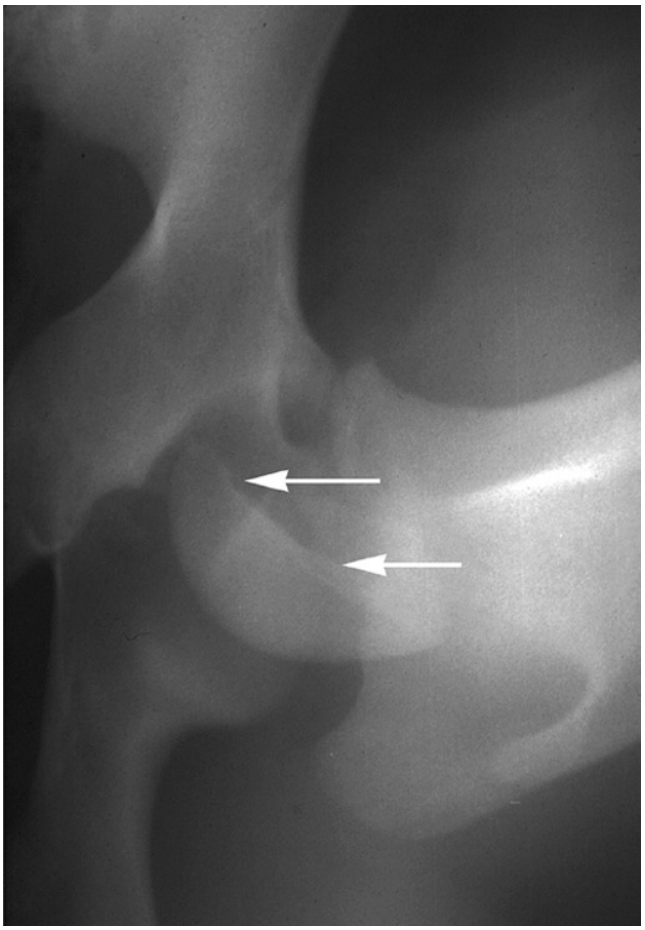


Figure 5.145. Recumbent radiograph of the pelvis of a foal with a capital physeal fracture (arrows) of the left proximal femur.

Interlocking nails (ILN) have been investigated for repair of diaphyseal femoral fractures (Figure 5.147).[58,70] In general, the less the horse weighs, the better the chance for success. Three successfully managed transverse midshaft femoral fractures in neonates have been reported using the ILN alone, along with 4 of 6 successfully managed with an ILN–dynamic compression plate combination.[99] Double-plate fixation has been reported to provide greater stiffness compared with ILN alone.[70]

Proximal growth plate fractures (slipped capital physeal fractures) are covered in the section on the coxofemoral joint. Most distal physeal fractures are Salter–Harris type II fractures (Figures 5.143A and 5.148). Minimally displaced distal physeal fractures may heal quite well with conservative therapy (Figure 5.149)[35] or with a modified Thomas splint/cast in small horses.[48] Unstable distal physeal fractures have little space for an adequate number of screws to be placed distal to the fracture, and many are not treated.[36] Options for stabilizing distal physeal fractures include an angled blade plate,[36] condylar

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Figure 5.146. Lateral radiograph of a yearling with a chronic diaphyseal fracture with a nonunion (proximal is at top of image). Notice the proximal displacement and overriding of the fracture fragments that often occur without internal fixation.

buttress plate,[67] locking compression plates,[50] a cobra head and dynamic compression plate,[46] and cross-pins or rush pins (Figure 5.148). Successful repair of a Salter–Harris type II fracture in a yearling Pony of America was reported using a condylar buttress plate.[67] A displaced Salter–Harris type IV fracture in a yearling was successfully treated using a lag screw repair countersunk into the articular surface.[20]



Figure 5.147. Interlocking nail used in this closed oblique mid-diaphyseal femur fracture of a miniature foal shown in Figure 5.143B.

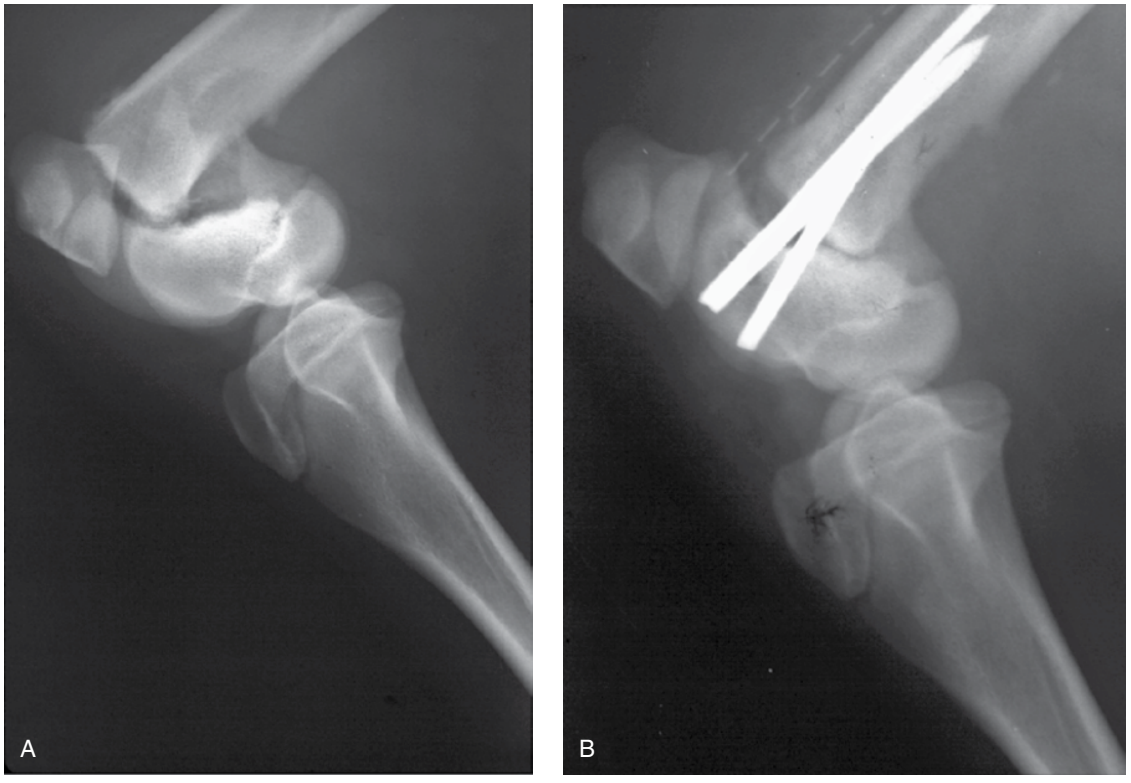


Figure 5.148. (A) Salter–Harris type II fracture involving the distal femur. (B) The fracture was stabilized with Steinmann pins placed in cruciate fashion. The fracture healed successfully, and the pins were removed 3 months after surgery. *Source*: Courtesy of Dr. Ted Stashak.

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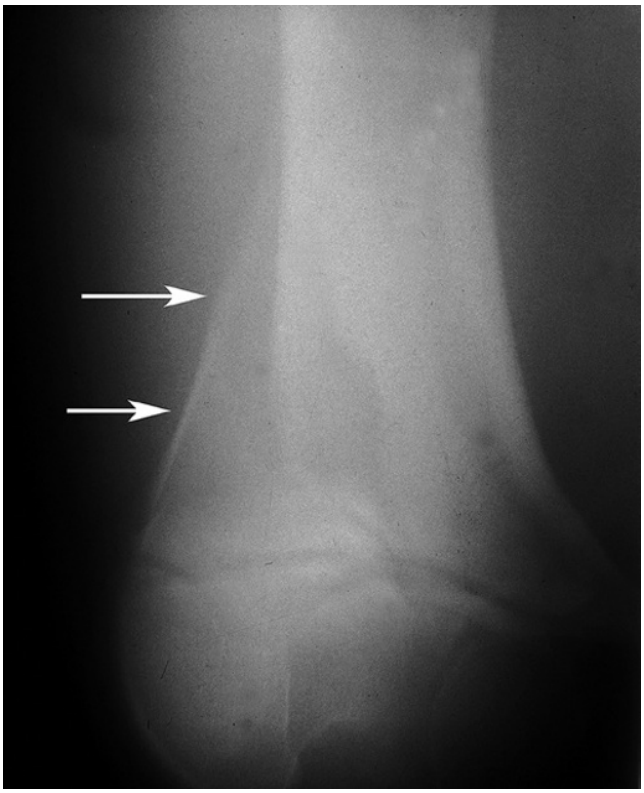


Figure 5.149. Lateral radiograph of a weanling with a type II distal femoral fracture (arrows) that was successfully treated with confinement.

Prognosis

The prognosis for fracture of the femur depends on the age of the horse, the location and configuration/type of fracture, and its intended use. Generally, femoral fractures in horses older than yearlings carry a very poor prognosis for a successful outcome. A much better prognosis can be expected in young animals with nondisplaced fractures or oblique midshaft fractures that are stabilized by internal fixation.[36] Nine of the 17 surgically repaired diaphyseal fractures healed.[7,36] Six of those were considered to have no sign of previous fracture.[36] The mean age for successfully treated foals was 2 vs. 4 months for unsuccessfully treated foals.[36] Aside from size of the foals being an influence, development of a postoperative seroma was negatively associated with success.[36] Three of 4 foals with oblique midshaft femoral fractures treated by stall rest alone became sound for breeding, but fracture disease associated with prolonged non-weight-bearing presented problems during the healing period.[57] Despite the improvement and advances in instrumentation, surgical techniques, and implants, femoral fractures are still among the ones that become infected, and those cases are less likely to be discharged from the hospital.[3,50]

In one report, only 1 of the 2 surgically repaired distal physeal fractures healed.[36] Even if successful, repair of distal physeal fractures can also lead to early physeal closure and a shortened limb.[50] A minimally displaced Salter–Harris type IV fracture treated conservatively in a yearling healed to racing status.[36] Aside from size of the foals being an influence, development of a postoperative seroma was negatively associated with success.[36]

FRACTURES OF THE THIRD TROCHANTER

The third trochanter is a prominent tuberosity that is located on the lateral aspect of the proximal third of the femur. It provides insertion to the tendon of the gluteus superficialis muscle, which is mainly a flexor of the coxofemoral joint and abductor of the limb.[30] Due to the superficial location, this tuberosity is prone to trauma causing fractures or injuries such as avulsion of the tendon of the gluteus superficialis muscle.[6] It is an uncommon cause of lameness with most horses showing severe lameness in acute cases. Clinical diagnosis can be challenging without advanced diagnostic techniques. Generally, it can be diagnosed with radiographs (50° cranial, 30° lateral caudomedial oblique, or craniolateral to caudomedial 25° oblique); however in horses with chronic displaced fractures, scintigraphy and ultrasound can help further evaluate the area.[18,76] This type of fracture is often displaced and heals with a fibrous union. Treatment consists of conservative management with rehabilitation including stall rest (1–2 months), controlled hand walking, and turnout (2–12 months).[6,76] The prognosis for return to athletic activity is good after an appropriate period of rest and restricted exercise.[6] However, another report showed 8 of 12 horses returning to athletic activity with 2 of these horses returning at a lower level.[76]

FIBROTIC AND OSSIFYING MYOPATHY

Fibrotic and ossifying myopathy is a fibrosis with or without ossification of the muscle tissue in the crus that often results in adhesions between the semitendinosus, semimembranosus, gracilis, or biceps femoris muscles (Figure 5.150).[1] It is significant because the fibrosis and adhesions limit the action of the semitendinosus muscle, causing an abnormal gait. Rarely, the condition occurs in the forelimb.[1] The largest case series reported in the United States related a predisposition for occurrence in Quarter horse mares.[52] A congenital form of fibrotic myopathy-type syndrome has been reported.[8] Foals are born with an altered gait characteristic of fibrotic myopathy in the hindlimb. On palpation, a tightening of the semitendinosus muscle was appreciated, but no firm thickening of the muscle typical of fibrotic myopathy was palpated. Because no fibrous thickening of the muscle is present, congenital restrictive myopathy may be a more appropriate description of this entity.

Etiology

Trauma causes fibrosis and adhesions of the semitendinosus, semimembranosus, gracilis, or biceps femoris muscles. It is typical for the semitendinosus to be involved, but any of the gaskin muscles can be affected. The involved muscles may be injured during sliding stops in rodeo work or from slipping and catching the hindlimb on the underside of a horse trailer, resisting a sideline, catching a foot in a halter, receiving intramuscular injections, being kicked, or cast for castration without sedation.[8,88] The lesions are usually unilateral, but a case of bilateral fibrotic myopathy resulting from a trailer accident has been reported.[8] In some cases the exact cause of the injury is not known; cumulative injury

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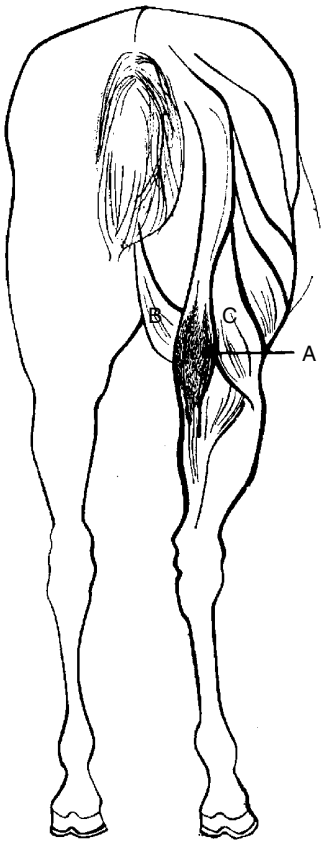


Figure 5.150. Drawing depicting the muscles in the fibrotic area in the gaskin of a horse affected with fibrotic myopathy. (A) Semitendinosus. (B) Semimembranosus. (C) Biceps femoris.

must be considered in cases such as western performance horses or those that involve any other repetitive situation. As the injury heals, fibrosis of the injured muscle as well as adhesions to surrounding musculature partially inhibits the normal action of the muscles, causing a shortened cranial phase of the stride (inability to extend the limb forward due to lack of muscular compliance). In some cases, the fibrosis will become ossified and is believed to be a more severe progression.

The etiology of the congenital form is unknown, although parturient muscle trauma has been described to cause rupture of the gastrocnemius muscle.[79] It is conceivable that similar trauma could also affect the semitendinosus under different stresses at birth or shortly after.

Three cases of fibrotic myopathy accompanied by neurogenic atrophy of the affected muscles have been reported.[98] All three underwent necropsy, where peripheral nerve and muscle degeneration were discovered. One horse had a fracture of the greater trochanter and accompanying sciatic nerve impingement. No gross lesions were found in the other 2 horses.

Clinical Signs

In the acute stages, generalized swelling and sensitivity to palpation of the gaskin area are present. As the injury starts to heal, an area of firmness with a variable pain response can be palpated over the affected muscles on the caudal surface of the limb at the level of the stifle joint and immediately above it (Figure 5.150). Most often, the main area of fibrosis can be felt over the semitendinosus. Occasionally, the lesion is deep and difficult to palpate, or it may be in the medial gaskin affecting the gracilis muscle. In the chronic stages, no pain is present on palpation.

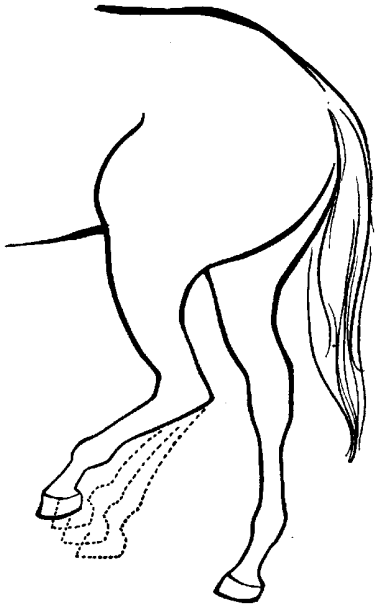


Figure 5.151. Characteristic action of the hindlimb of a horse affected with fibrotic myopathy. The limb jerks backward and downward (dotted outline of the affected foot) during the last 3–5 in. of the stride, resulting in slapping of the foot on the ground. If the gracilis muscle is affected, the swing of the hindlimb is lateral to medial instead.

Fibrotic myopathy demonstrates a gait abnormality characterized by a slapping down of the foot at the end of the cranial phase of the stride (Figure 5.151). Usually the lameness is most noticeable when the horse walks and is diminished when the horse trots. The character of the gait of a semitendinosus-affected horse has been described in detail.[13] In the cranial phase of the stride, the foot of the affected hindlimb is suddenly pulled caudally 3–5 in. just before contacting the ground (Figure 5.151). The affected hindlimb approaches the ground more vertically than normal and strikes it toe first, making more of a slap than the normal heel–toe manner of landing. Ultimately, the cranial phase of the stride is shortened with the caudal phase lengthened. If the semitendinosus is more affected, the slap is cranial to caudal. If the gracilis is more affected, the slap is from lateral to medial. Some have reported a semiflexed stance at rest with the toe touching the ground.[13,31]

Diagnosis

In chronic cases, the diagnosis is straightforward as it is based upon the altered gait and palpation of a hardened area on the caudal or caudomedial surface of the limb proximal to the level of the stifle. In making the diagnosis, stringhalt should also be considered. In stringhalt, the limb is pulled sharply toward the abdomen during the cranial phase of the stride, while in fibrotic myopathy the foot is pulled toward the ground in a caudal direction just before the foot contacts the ground.

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However, in the acute case, it can be more difficult to diagnose due to the amount of swelling and pain that can occur in the gaskin region. Nuclear scintigraphy (Figure 5.152), ultrasound, thermography, and local infiltration of the area may all be necessary in horses with acute injuries.[17]

Treatment

Treatment is often based on chronicity of the lesion. In general, the more acute the lesion, the more the clinician can attempt to minimize the formation of a fibrotic scar (Figure 5.153). Once the mature scar tissue is present, then surgery should be considered.

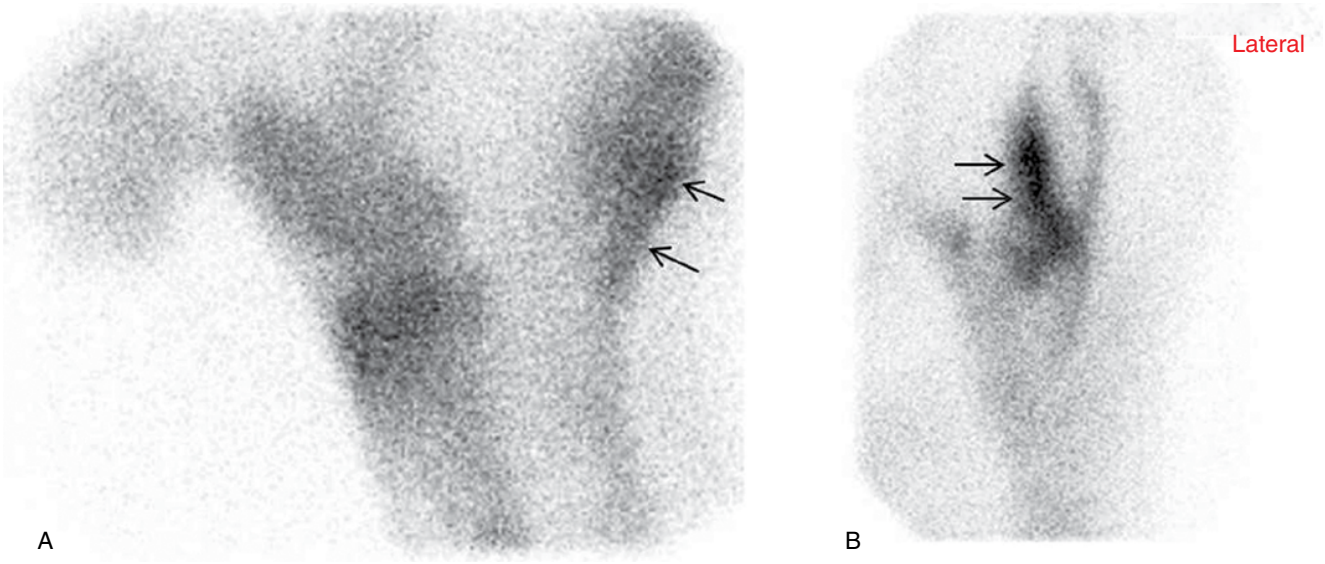


Figure 5.152. Nuclear scintigraphy performed on an adult horse that presented for an acute injury to the gaskin region secondary to trauma. (A) Lateral image showing increased radiopharmaceutical uptake in the musculature of the caudal aspect of the limb (arrows) just proximal to the level of the stifle. (B) Caudal cranial image with the lateral side of the limb labeled. There is increased radiopharmaceutical uptake just proximal to the level of the stifle (arrows) in the location of the semitendinosus muscle.



Figure 5.153. Images of the adult horse that sustained an acute muscle injury identified with scintigraphy in Figure 5.152. (A) Six months after injury. (B) One and a half years after injury. Note that the thickened area of fibrous tissue in image A (arrows) is more distal than the final fibrous tissue after 1.5 years. This horse had multiple treatments to minimize the development of scar tissue.

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In the acute stages, nonsteroidal anti-inflammatory drugs (NSAIDs) or other pain medication are usually required to help minimize the swelling and pain in the gaskin region. It is ideal to try to move and stretch the muscles as soon as the horse can tolerate it. The hope is to create as elongated and functional scar of the muscle(s) as possible. Initially, the hindlimb can be stretched cranially and caudally (Figure 5.154) working in deep massage of the affected muscle once the horse can handle it. Hand walking should start on a hard surface over the first few weeks, but range of motion exercises should be added in as soon as possible. This could include walking them part time in arena footing, followed by walking them over logs, progressing to cavaletti or an underwater treadmill, if available. The ideal water level to use in an underwater treadmill is fetlock depth.[59] In addition, as the fibrous scar develops, one can use shockwave. This can help break up the scar and make them more comfortable for moving and stretching exercises. In addition, once the scar is chronic, shockwave can periodically help if the size enlarges or new painful areas develop. Additional therapies such as therapeutic ultrasound and acupuncture could also be used during the initial phases of treatment.

Three approaches for surgical correction of fibrotic myopathy have been described; each must be adjusted if more than the semitendinosus muscle is involved. Adams originally described complete removal of the affected fibrotic/calcified muscle tissue.[1] In a retrospective study of 18 horses undergoing this procedure, all of the 11 horses available for follow-up had mild to complete persistence of the gait deficit.[96] Anecdotally, quadriceps rupture has been observed following resection of an extensive calcified mass. The failures were most likely due to recurrence of fibrosis in the defects created, but it is possible that there was more extensive involvement in some cases. A modification of this technique was reported by Janicek et al. using a Nd:YAG laser to transect fibrotic mass (fibrotomy). Advantages reported were a more controlled and complete hemostasis, which improved the visualization of the fibrotic mass and facilitated the differentiation from normal tissue.[44]

Bramlage described a semitendinosus tenotomy at the level of its insertion on the proximal medial tibia caudal to the saphenous vein.[8] The procedure is much less invasive and is expected to be effective if the problem is unaffected by other muscles. The tenotomy is performed at the insertion of semitendinosus tendon on the proximal to medial tibia with the horse positioned in lateral recumbency with the affected limb down.[8] Three of four (1 mature and 2 congenital) horses recovered completely; the fourth (a congenital) case underwent more distal transection of the calcaneal insertion of the semitendinosus tendon 2 months later, which provided sufficient improvement for race training.[8] Of 2 additional cases in mature horses, 1 experienced complete recovery and 1 improved slowly but incompletely.[31] In a more recent report in which the semitendinosus muscle was affected in 6 horses, 2 improved with no gait deficit remaining, with 3 more improving some after standing surgery.[87] Interestingly, when the surgery was performed under anesthesia in another 8 horses, none improved all the way, with only 3 improving some.[87]

Magee and Vatistas reported a series of 39 horses that underwent a standing focal semitendinosus myotomy.[52] Average wound healing time was 4.7 weeks overall. Eighty-three percent of horses were able to perform at their pre-injury level. Nineteen were considered normal and 10 had some persistent restrictive gait. The incision is made directly over the affected semitendinosus muscle, which is transected using a blunt bistoury. The incisions are left open, partially closed, or completely closed, depending upon the surgeon's preference (Figure 5.155).



Figure 5.154. Cranial (A) and caudal (B) stretching exercises being performed on an adult Quarter horse that sustained an injury to its semitendinosus muscle. These exercises are most important within the first 2 weeks after injury.

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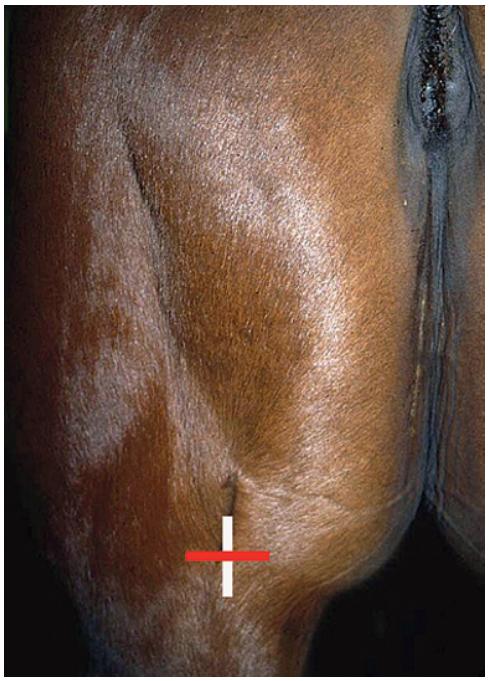


Figure 5.155. Caudal thigh region where the myotomy procedure is performed to correct fibrotic myopathy. The skin incision is made vertically (white line) and the fibrotic muscle is transected with a bistoury horizontally (red line).

No cases of involvement of muscles other than the semitendinosus were reported.

Prognosis

If it is possible to see the horse acutely, it is possible to minimize the scar tissue development and adhesions between different muscles. It is important to note, however, that although the characteristic gait will be harder to see, it will still be present. With partial myectomy, some relief is evident in all cases. After healing, some cases develop characteristic, but less pronounced, signs, although limb function is nearly normal and signs are not noticeable except in the walk. Occasionally, 3–7 days are needed for the maximum benefits of surgical correction to become evident. The semitendinosus tenotomy prognosis is fair, with horses improving more when only the semitendinosus muscle was affected compared with when the semimembranosus muscle is also affected.[87] With the standing myotomy technique, 83% of horses were able to perform at their pre-injury level, although the restrictive gait pattern did not resolve in all horses.[52] In the modified fibrotomy technique with the Nd:YAG, of the 6 horses with long-term follow-up, 4 returned to full use with no abnormalities and 1 with partial improvement.[44] A specific diagnosis of the muscles affected can help prepare owner expectations, recognizing that in those cases where more muscles are affected, a lower prognosis should be given. The prognosis for successful surgery in horses affected by neurogenic atrophy of the muscles is poor. If the muscle mass appears abnormal, nerve conduction studies or electromyography is advisable.

FEMORAL NERVE PARALYSIS (CRURAL PARALYSIS)

Paralysis of the femoral nerve affects the quadriceps femoris group of muscles. This muscle group is composed of the rectus femoris muscle, vastus lateralis muscle, vastus medialis muscle, and vastus intermedius muscle. This large muscular mass covers the front and sides of the femur and inserts onto the patella to extend and fix the stifle.

Etiology

Femoral nerve paralysis may arise from trauma or unknown causes and may be associated with rhabdomyolysis. Injury to the nerve may occur from overstretching the limb during exertion, kicking, slipping, or while the horse is tied in a recumbent position. It has also been reported as a complication of general anesthesia.[24]

Hemorrhagic neuritis has been described in 1 horse that was euthanatized due to femoral paralysis.[24] Unaffected horses had no such lesion at necropsy. The affected horse also had lesions of rhabdomyolysis, and the urinary system was affected by myoglobinuria. Postmortem studies with cadavers revealed that the femoral nerves were placed in tension by extending the hindlimbs behind the horse, whereas lifting the horse by the hindlimbs did not place tension on the femoral nerves.[24] The potential roles or interaction of femoral paralysis and rhabdomyolysis warrant further investigation.

Clinical Signs and Diagnosis

The horse assumes a crouched position with the fetlocks flexed and the toes on the ground and is unable to bear full weight on the affected limb. There is difficulty advancing the limb, but the affected horse can do so because the hock can be sufficiently flexed to pull the limb forward. After the condition has been present for some time, atrophy of the quadriceps muscles occurs, causing them to lose their normal softness and become more like tendinous structures. If rhabdomyolysis is present, the horse is more painful and less willing to attempt to stand.

The signs listed above are characteristic and are used for diagnosis. The condition should be differentiated from lateral (true) luxation of the patella, rupture of the quadriceps femoris muscles, avulsion of the tibial crest, and distal luxation of the patella. Any of these conditions could cause a similar syndrome; however, all are rare. Lateral luxation of the patella can be diagnosed by palpation of the displaced patella; rupture of the quadriceps femoris muscle also can be palpated. A radiographic examination can determine avulsion of the tibial crest where the patellar ligaments insert. Electromyography of the quadriceps femoris muscles 5 days after the first signs of femoral nerve paralysis provides a definitive diagnosis.

Treatment

No treatment is known. If the condition is due to injury of the femoral nerve, the animal should be stalled until improvement occurs. If rhabdomyolysis is present,

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intensive care and support is indicated, and repeated attempts to use the muscles should be discouraged.

Prognosis

The prognosis is guarded to unfavorable. In a report of two cases of post-anesthetic femoral paralysis, one had concurrent rhabdomyolysis and was euthanatized. The other responded to supportive therapy and was standing well 7 days after the onset.[24,51] The prognosis should be withheld until sufficient time has elapsed to determine whether any function will return.

CALCINOSIS CIRCUMSCRIPTA

Calcinosis circumscripta in the horse is a calcified mass commonly located on the lateral aspect of the gaskin adjacent to the stifle (Figure 5.156).[22,33,43,86] The condition has also been observed in other locations such as the carpus, tarsus, shoulder, and pectoral region.[22,34,86] The locations can be unilateral, bilateral, or multiple locations on the body.[22,43,86] Males appear to be affected the most, as only a few of the total cases presented in the literature are females.[22,33,43,86] The age at presentation has ranged from 1 to 13 years, with mention that the condition has not been reported in foals younger than 6 months of age.[22,33,34,43,86]

Etiology

The etiology is unknown. The description of the lesion best fits that of dystrophic calcinosis characterized by calcinosis circumscripta. Tumor calcinosis implies a metabolic derangement in calcium/phosphorus metabolism leading to hyperphosphatemia and resulting accumulation of deposits of calcium phosphate.[86] Although few have investigated the serum chemistries of affected horses, hyperphosphatemia has not been a reported feature of the condition in horses.[43,86]

Clinical Signs and Diagnosis

Because clinical signs of lameness seldom occur, horses may carry the lesion for years before presentation to a hospital. The masses are generally 3–12 cm in diameter. The skin is freely movable over the density because it lies beneath the superficial and deep fascia. They are often attached to the joint capsule of the lateral femorotibial joint. Diagnosis is confirmed on radiographs (Figure 5.156).

Once dissected free, the lesions are encapsulated in a tough, white, fibrous capsule.[22,43] The interior is composed of loculations of finely granular, gritty, white, paste-like material.[22] Histologically, the granules are surrounded by a granulomatous inflammatory reaction.[22] Although it is difficult to make a broad comparison, the lesion in humans is believed to be secondary to collagen necrosis.[91]

Treatment

The only treatment is surgical excision.[22,43] However, the lesions seldom cause a clinical problem. The location is one that tends to dehisce following surgery and must be taken seriously in that respect.[43] Also, due to the proximity to the femorotibial joint, there is a potential to create a septic arthritis.[22] If surgery is performed, a tension-relieving closure using mattress sutures and a sutured stent bandage is advised. A circumferential adherent surgical drape is helpful in preventing swelling.

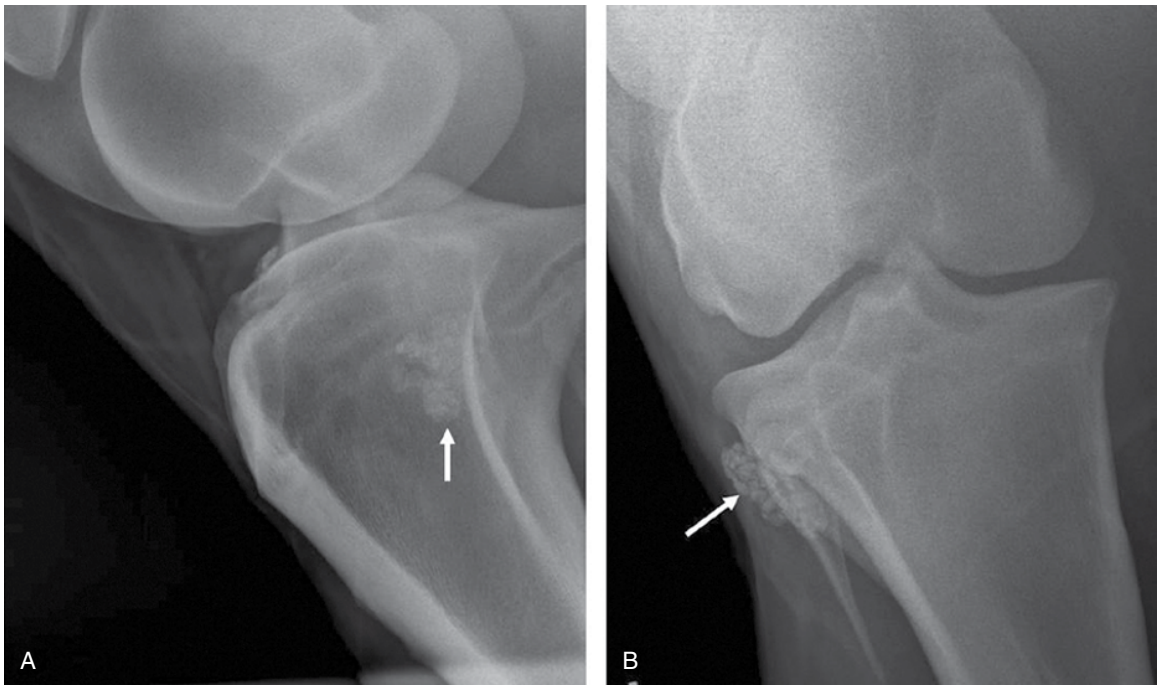


Figure 5.156. (A) Lateral and (B) caudal to cranial radiographic images of the stifle in a 5-year-old horse showing a calcified mass on the lateral aspect of the stifle (arrows).

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Prognosis

The prognosis is good for soundness and future use. Clinical problems or lameness is uncommon. The prognosis after surgery is guarded, depending upon the size of the lesion due to potential for incisional complications.[5,22,43]

TROCHANTERIC BURSITIS (TROCHANTERIC LAMENESS, WHIRLBONE LAMENESS)[81]

Trochanteric bursitis is inflammation of the bursa beneath the tendon of the accessory gluteal muscle (deep portion of the gluteus medius muscle) as it passes over the greater trochanter of the femur. The tendon of the gluteus medius muscle also may be involved, as well as the cartilage over the trochanter. The deep portion of the gluteus medius muscle has a strong flat tendon that passes over the convexity of the trochanter before it inserts into the crest, and it functions to extend the coxofemoral joint and to abduct the limb. The trochanter is covered with cartilage and the trochanteric bursa is interposed between it and the tendon.[30]

Etiology

In many cases, there is a concurrent source of chronic lameness in the same limb, particularly distal tarsitis. Chronic forelimb lameness may also contribute to trochanteric bursitis by causing the horse to place more strain on the hindlimbs.[12,37] Lameness may also be caused by bruising as a result of the horse falling on the affected side or by strain during racing or training.

Trochanteric bursitis occurs in horses racing on small tracks, where the turns are close together, and in horses working on their hindlimbs that are frequently exercised in soft, deep arenas. Short heels and long toes in the hindfeet seem to predispose to this lameness. When the etiology is severe trauma, such as a direct kick, the cartilage or the bone of the trochanter may be fractured, causing persistent lameness. It also has been found following an attack of strangles.

Clinical Signs and Diagnosis

Pain may be evident when pressure is applied over the greater trochanter. Careful interpretation is required, because some horses naturally tend to shy away when pressure is applied over the hip joint. When pushing on the greater trochanter, a normal horse should lean back into the clinician, but in cases of trochanteric bursitis, horses will move away from the examiner. At rest, the limb may remain flexed; as the horse moves, weight may be placed on the inside of the foot so that the inside wall of the foot wears more than the outside wall. This can be best seen when observing the horse from behind—the foot is carried inward and the horse sets the foot down on a line between the forelimbs. The horse tends to travel "dog fashion" since the hindquarters move toward the sound side because the stride of the affected limb is shorter than that of the sound side. After the condition has been present for some time, atrophy of the gluteal muscles occurs.

The foregoing symptoms should be used in diagnosis. The condition is difficult to differentiate from inflammation of the coxofemoral joint. The lameness, which is not common, may be confused with spavin lameness. A lameness of unknown cause is sometimes ascribed to trochanteric bursitis. Injection of a local anesthetic into the bursa is helpful in differentiating the condition from coxofemoral joint osteoarthritis (OA). Nuclear scintigraphy should identify the area as inflamed. Toth et al. compared four different techniques for trochanteric bursa centesis and concluded that centesis performed using a spinal needle with the ipsilateral foot non-weight-bearing and placed in a Hickman block positioned caudal to the contralateral foot.[93] Using the ultrasound can further increase the likelihood of successful centesis.[93]

Treatment

It is important to address any associated source of lameness. Rest and anti-inflammatory treatment may be helpful in some cases. Injection of the bursa with corticoids appears to be the most effective method of treatment. The authors have had success in treating the bursa with shockwave (1–3 treatments). Other reported treatments consist of injections of Lugol's solution of iodine into or around the bursa as a counterirritant. Hot packs applied to the affected area in the acute stages relieve some pain. Phenylbutazone therapy for 3–4 weeks may be useful. Treatment is difficult when the cartilage or bone has been damaged with fracture or periostitis. Although surgery may be indicated in these cases, no reports could be found regarding the use of surgery.

Prognosis

The prognosis is usually associated with the prognosis of a second source of lameness. If the other source is treated, as well as the bursa, the horse will usually return to soundness within 4–6 weeks. However, if there is no other source of lameness, or the injury is more severe, the prognosis is guarded to unfavorable, and the lameness may remain indefinitely or may recur when the horse is put back into training.

RUPTURED QUADRICEPS MUSCLE

Ruptured quadriceps muscle is a rare condition that causes the horse to present with a dropped stifle.[61] A single case of unknown etiology was observed in a nursing foal. The patella was prominent due to the cranial position of the stifle without the quadriceps attachment.[82] Anecdotally, quadriceps rupture has been observed following extensive resection of a calcified and fibrotic mass causing a fibrotic myopathy.

THE COXOFEMORAL JOINT

The femoral head and the acetabulum form a ball-and-socket articulation that makes up the coxofemoral joint. The heavy musculature and extensive ligament network surrounding and within the joint make the hip joint inherently stable.[23] The transverse acetabular ligament, ligament of the head of the femur (round), and

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femoral accessory ligament help to stabilize the coxofemoral joint.[23,45] In addition, the acetabulum is surrounded by a fibrocartilaginous rim that increases the bony margin of the acetabulum.

The accessory ligament is the largest and strongest ligament and is unique to equids.[45] It arises from the fovea capitis of the femoral head, passes through the acetabular notch to the pubic groove, and becomes part of the prepubic tendon. The smaller ligament of the head of the femur (round) arises in the head of the femur adjacent to the accessory ligament and attaches to the pubic groove.[45] This ligament of the head of the femur can be seen and debrided arthroscopically, but the accessory ligament is difficult to visualize.[62–64] The transverse ligament courses from the outer fibrocartilaginous rim medially across the acetabular notch.[45] The reader is referred to Chapter 1 for more detailed anatomy of the coxofemoral region.

In general, problems related to the coxofemoral joint appear to occur most commonly in foals, miniature horses, and ponies.[23,28,41,53,64] However, horses of any age may sustain damage to the coxofemoral joint from trauma. Most conditions are either developmental (osteochondrosis, osteochondritis dissecans, hip dysplasia), infectious (sepsis involving the coxofemoral joint or proximal femoral physis), or traumatic (tearing of the femoral accessory ligament, rupture of the round ligament, capital physeal fractures, hip luxation, IA acetabular fractures, and OA) in origin.

Many of these conditions cause a moderate to severe lameness and the limb is often outwardly rotated when viewed from the rear (toe-out, hock-in conformation) (Figures 5.157 and 2.97). In addition, the limb may appear straighter than the contralateral limb, and the horse may lean away from the affected limb (Figure 5.142). External swelling of the coxofemoral joint is often difficult to detect, but visual enlargement over the greater trochanter may be evident (Figure 5.158). Pain can often be elicited with direct inward pressure of this area. Rectal palpation may reveal swelling along the axial aspect of the joint but can only be performed in larger horses and gives many false-negative results. Crepitus with limb manipulation is not a common finding except with acetabular or more extensive pelvic fractures, hip luxations, or some cases of complete ligament rupture. Muscle atrophy (gluteal and quadriceps) can be found in some horses with chronic injuries to this area (Figure 2.98).

A definitive diagnosis of a hip problem can be challenging without additional diagnostics. Many horses can lack specific clinical signs such as pelvic asymmetry and/or crepitus. Arthrocentesis of the joint with or without IA anesthesia can be performed but is often difficult in large horses.[38,64] Transcutaneous ultrasound-guided arthrocentesis may be helpful in these cases.[19] In addition, it is often much easier to perform arthrocentesis of the hip in foals and ponies in lateral recumbency under anesthesia. In many cases of chronic hindlimb lameness, IA anesthesia is necessary to confirm that the hip is the site of the lameness. Rectal and transcutaneous ultrasound may be helpful to document capital physeal and acetabular fractures and hip luxations.[9,32] In foals, ultrasound provides good-quality images, but is unable to assess an accurate fracture configuration.[74] Nuclear scintigraphy has improved the potential to localize lameness to the hip region but does not provide a definitive diagnosis (high sensitivity/low specificity).[18] In many cases, radiography of the coxofemoral region using a standing technique or under anesthesia is necessary to either rule in or rule out a problem in the coxofemoral joint.[55,56,89] Geburek et al. reported an agreement of 73% between ultrasonographic and radiographic diagnosis of pelvic–femoral disorders.[29] In another report, a CT of two Warmblood fillies (270 and 210 kg) was performed under general anesthesia with good results, providing valuable information for both cases. However the authors acknowledged the cost and risk of this procedure under anesthesia.[95]

In general, the prognosis for horses with hip disease is guarded to poor for athletic use. However, this depends on the type of lesion, the extent of secondary OA, and whether the lesion is accessible for arthroscopic debridement.[62–64] Young horses with OCD lesions that can be debrided arthroscopically and foals with joint ill without secondary osteomyelitis appear to have the best



Figure 5.157. Young horse with asymmetry of the pelvis, muscle atrophy over the left hip, and a toe-out stance. The horse was lame at the walk and an acetabular fracture was present on radiographs.

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Figure 5.158. An older Quarter horse mare with grade 3 of 5 hindlimb lameness. Swelling over the left greater trochanter could be seen when compared with the opposite side, and pain was elicited with firm palpation. Radiographs revealed severe OA of the coxofemoral joint (Figure 5.163).

prognosis. Horses with complete rupture of the ligament of the head of the femur or accessory ligament, complete joint luxation, and OA tend to do poorly, regardless of treatment.

OSTEOCHONDROSIS OR OSTEOCHONDRITIS DISSECANS (OCD)/HIP DYSPLASIA OF THE COXOFEMORAL JOINT

Developmental lesions of the coxofemoral joint are rare in comparison with other joints in the horse. Malformation of the joint, hip dysplasia, osteochondritis dissecans, and subchondral cystic lesions (SCLs) have been described in the coxofemoral joint.[38,64,65,78,89]

Etiology

The cause of these developmental lesions is assumed to be the same as for other OCD-type lesions. It is unknown why there is a low prevalence of OCD-type lesions in the hip compared with other locations. Much of the coxofemoral joint is weight bearing; therefore, the development of SCLs could be trauma induced, similar to other weight-bearing joint surfaces.

Clinical Signs

Clinical signs of young horses with OCD of the hip may be similar to those of any hindlimb lameness. They may include a stilted hindlimb gait, low foot flight arc, shortened cranial phase of the stride, and dragging of the hindfeet.[65] In cases of bilateral disease, the hindlimbs may be carried very straight, and the weight shifted toward the forelimbs.[38,78,89] Physical abnormalities of the limb(s) or palpable pain may be difficult to document.

Diagnosis

Lameness in young horses may be localized to the hip by eliminating the remainder of the limb as the source of pain along with the characteristic stance of the limb. Physical examination findings may reveal pain on palpation and manipulation of the hip. IA anesthetic of the coxofemoral joint can be performed to aid diagnosis, but young horses do not tolerate the procedure very well. A definitive diagnosis is usually achieved via radiography. Better quality radiographs can generally be obtained under general anesthesia, although ventrodorsal and oblique radiographs of the pelvis can be performed in the standing horse.[55,56,64] Radiographic abnormalities consistent with OCD of the hip are similar to those of other locations and include SCLs, osteochondral fragments, abnormal contour of the femoral head or acetabulum, and shallow and irregular acetabulum (Figure 5.159A).

Treatment

Depending on the severity of the OCD lesion, conservative or medical treatment is usually unsuccessful. However, palliative treatments aimed at cartilage and joint healing may be used in young horses with the hope of joint remodeling over time. Surgical debridement of the lesion is usually the treatment of choice, especially if osteochondritis dissecans lesions are present.[64] Arthroscopy of the coxofemoral joint is more easily performed in foals and weanlings, but can be accomplished in older horses with proper equipment.[39,62–64] Surgical debridement is also the treatment of choice for affected cartilage and subchondral bone. However, access to all areas of the coxofemoral joint is not possible. In severe cases of unilateral hip malformation or dysplasia (Figure 5.159A), a femoral head ostectomy may provide a salvage procedure for breeding soundness.[80] These procedures are performed infrequently in the horse.

Prognosis

The coxofemoral joint is a major weight-bearing joint, and articular abnormalities such as OCD often lead to OA. Young animals with small OCD lesions that can be debrided arthroscopically and have minimal evidence of OA may do well.[64] However, the prognosis for most horses with hip OCD should be considered guarded to poor for future athletic use.

INFECTIOUS ARTHRITIS/PHYSITIS OF THE COXOFEMORAL JOINT

Infection of the coxofemoral joint and the capital physis of the femur are part of the joint ill complex in foals.[56,69] Infections around the hip occur less frequently than at other sites in foals and can be very difficult to diagnose.

Etiology

Joint and physeal infections in foals are hematogenous in origin, and bacteria usually gain access to the circulation through the umbilicus, gastrointestinal tract, or

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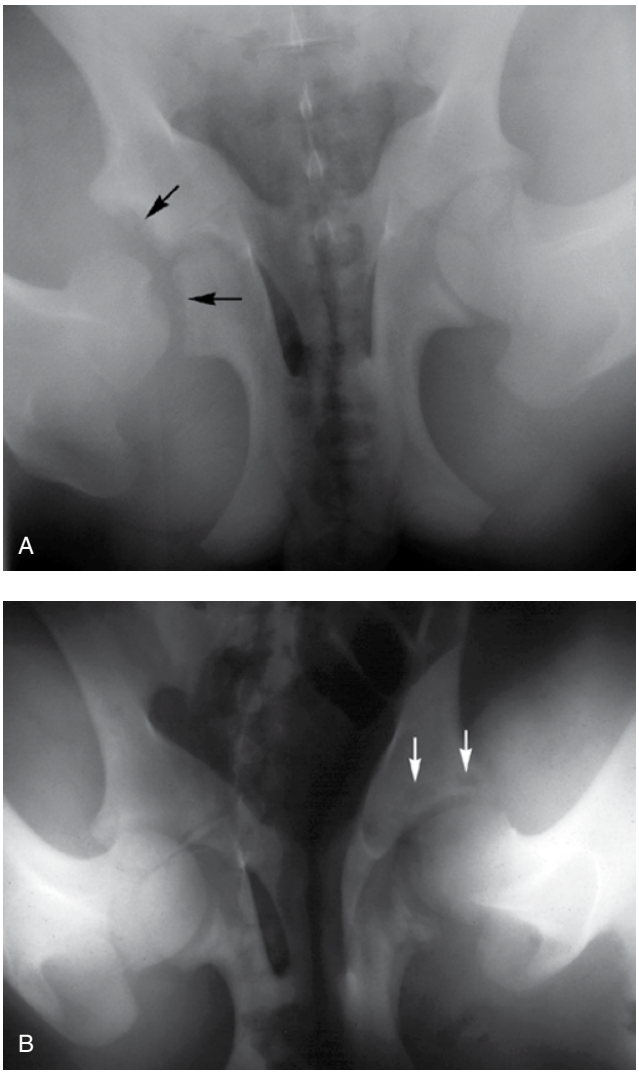


Figure 5.159. Ventrodorsal radiographs of the pelvis of two 3-month-old fillies with a history of increasing lameness. (A) Remodeling of the femoral head and acetabulum (arrows) consistent with a developmental abnormality. (B) Lysis of the left acetabulum (arrows) consistent with infectious arthritis and osteomyelitis.

respiratory tract.[54,68,84] Affected foals usually have a history of failure of passive transfer. Bacteria localize in and around the physes and joints because of the specific low-flow circulation that is present in these areas. The syndrome may be classified according to the location of the lesion: S-type involves the synovium/joint, E-type involves the epiphysis, and P-type involves the physis.[54] Most infections are from Gram-negative bacteria. Infection of the proximal femoral physis may spread to the coxofemoral joint and surrounding tissues of the hip region.

Clinical Signs

Infection of the coxofemoral joint can be a diagnostic challenge because joint effusion, heat, and pain are often not found on physical examination. Typically, these foals are less than 4 months of age and often present with a unilateral hindlimb lameness of unknown cause.[60] They may have a characteristic toe-out, stifle-out, hock-in appearance. Pain is often elicited with deep palpation over the greater trochanter and hip region. It can often be confused with trauma to the limb but the infection and therefore the lameness is often progressive, whereas a traumatic injury often improves with time. Any young foal with severe hindlimb lameness but no definable lesions in the lower limb should be suspected of having infection of the hip joint. In addition, infections of the capital physis can spread to the hip musculature, and the foal may present with an abscess in the gluteal region.

Diagnosis

A complete blood count is often very helpful to help differentiate trauma from a potential hip infection. High white blood cell counts and fibrinogen concentrations help confirm the presence of an infection.[60] However, a definitive diagnosis of infectious arthritis is made by analyzing the synovial fluid of the hip joint following arthrocentesis, with or without adjunctive IS anesthesia.[4] Synovial white blood cell counts of 30,000 cells/μL or more and total protein concentrations of 4.0 g/dL or more are consistent with infection. Physeal infections can be documented with radiographs of the hip region, where lysis around the capital physis, epiphysis, and occasionally the acetabulum is seen (Figure 5.159B).

Treatment

Treatment is similar as for any other joint/physis with a hematogenous infection. Broad-spectrum systemic antimicrobials, IA antimicrobials, and joint lavage/drainage are all important. Arthroscopy can be performed in small horses and foals to help lavage and debride these lesions, but cannula lavage alone can be very helpful.[4,39,62–64] Unfortunately, many of these infections are not diagnosed in the early stages, and significant osteomyelitis is often present, worsening the prognosis (Figure 5.159B).

Prognosis

The prognosis is usually poor because the diagnosis is seldom made before significant joint abnormalities and osteomyelitis have occurred. However, foals with coxofemoral infectious arthritis without radiographic abnormalities should respond well to aggressive treatment, and their prognosis for athletic use is similar to that of foals with joint ill at other locations.[4]

PARTIAL TEAR/RUPTURE OF THE LIGAMENT OF THE HEAD OF THE FEMUR (ROUND LIGAMENT)

A partial tear or rupture of the ligament of the head of the femur (round ligament) of the coxofemoral joint is a relatively rare condition that can affect any age or breed of horse. Nevertheless, it appears to be more common in small-breed horses (miniatures and ponies) and young horses.[64] The hip joint is usually very stable, but occasionally a partial tear or complete rupture of ligament of the head of the femur can occur without a complete joint luxation. In these cases, the head of the

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femur may have greater range of motion within the joint, contributing to synovitis, joint effusion, and lameness.[38] Subsequent degenerative changes within the joint often lead to OA. Definitive diagnosis of a ruptured ligament of the head of the femur can be difficult.

Etiology

Trauma is the most common cause of either a partial tear or complete rupture of the ligament of the head of the femur. The same type of forces/stresses that cause luxation of the coxofemoral joint may also damage this ligament without resulting in a joint luxation.

Clinical Signs

Affected horses with complete rupture of the ligament of the head of the femur often have an acute onset of lameness and a history of trauma. Visible swelling over the hip is often difficult to detect, but gluteal atrophy may be present if the condition is chronic. Horses often stand with a toe-out, stifle-out, and hock-in appearance of the affected hindlimb that is typical of problems in the coxofemoral region.[38] Unlike horses with a complete hip luxation, the limbs are the same length, and the points of the hocks are at the same height.[38] Firm intermittent pressure applied over the greater trochanter usually elicits a painful response. Limb manipulation, flexion of the hip joint, and upper limb flexion tests may also be painful. Crepitation over the joint may be present because of the excessive motion of the femur but can be difficult to document.[2]

The clinical signs associated with partial tearing of the ligament of the head of the femur are not as clear as those seen with complete rupture of the ligament. The hock-in and stifle and toe-out appearance is generally not observed, and these horses may present with a chronic hindlimb lameness. Varying degrees of gluteal muscle atrophy may occur in chronic cases, but this is not a consistent finding. Limb manipulation, particularly hip abduction and flexion of the joint, may be present but less so than with complete ligament rupture. Intermittent firm pressure applied externally to the greater trochanter usually elicits a painful response.[2]

Diagnosis

A presumptive diagnosis of a complete rupture of the ligament of the head of the femur is based on a history of trauma combined with an acute lameness with clinical signs characteristic of a hip problem. However, several other hip-related injuries (capital physeal fractures, other ligamentous injuries, acetabular fractures) may present with similar histories and clinical signs. Hip luxations usually can be ruled out based on the limb length.[38] Although the clinical signs often localize the problem to the hip region, intrasynovial anesthesia may be required to prove that the joint is affected. Aspiration of hemorrhagic synovial fluid during arthrocentesis is consistent with joint trauma. See Chapter 2 for additional information regarding the technique for intrasynovial anesthesia of the hip joint.

Radiography or ultrasound of the hip region is necessary to rule out other possible problems. Ventrodorsal and oblique radiographs of the hip can be performed with the horse under general anesthesia or standing. Radiographs of the hip may be normal with rupture of the ligament of the head of the femur and avulsion of the insertion of the round ligament,[66] or an abnormal position of the femoral head within the acetabulum may be identified.[2] Subluxation of the coxofemoral joint may also be diagnosed with dynamic ultrasound (weight-bearing and non-weight-bearing views) of the hip region in standing horses.[9] An abnormal position of the femoral head seen on radiographs or with dynamic ultrasound together with hemarthrosis of the hip identified with arthrocentesis is highly suggestive of an acute ruptured round ligament. If the condition is chronic, radiographic signs consistent with OA are often present. These include osteophytes on the cranial and caudal rim of the acetabulum and at the capsular attachment on the femoral neck.[64]

Diagnostic approaches used to identify a partial tear of the accessory ligament may include scintigraphy, radiography, and arthroscopy. Although not diagnostic for a partial tear, scintigraphy may show a mild to moderate radionucleotide accumulation in the affected hip, particularly if secondary degenerative joint changes are present.[38,49,63,64] This is often recognized as being able to clearly recognize the entire ball of the proximal femur on the scan. Radiography may identify changes associated with OA of the hip joint but is not specific for damage to the ligament of the head of the femur. Subluxation of the coxofemoral joint due to partial tearing of the ligament of the head of the femur was diagnosed in 2 horses with dynamic ultrasound and should be considered in these cases.[9] Arthroscopy of the coxofemoral joint also may be used to diagnose and potentially treat (debridement) partial and complete ruptures of the ligament of the head of the femur.[62–64]

Treatment

There is no effective treatment for complete rupture of the ligament of the head of the femur of the hip joint. Affected horses often develop OA and remain lame.[64] However, arthroscopy of the hip joint has been used successfully to debride partial tears of the round ligament and to perform a synovectomy.[62–64] Ligament tearing in small breeds of horses, particularly miniature horses, can be adequately debrided, and a return to soundness is possible.[64]

Prognosis

The prognosis is poor for horses with complete rupture of round ligament, regardless of treatment. Secondary OA of the hip joint appears to be a likely sequela. However, the response to debridement of partial tears in small-breed horses has been favorable in a limited number of cases.[64]

COXOFEMORAL SUBLUXATION AND LUXATION (DISLOCATION OF THE HIP JOINT)

Subluxation or luxation of the coxofemoral joint is an uncommon condition in horses compared with cattle because of the numerous ligaments and heavy musculature surrounding the joint. The femoral accessory ligament

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and the round ligament both attach to the femoral head and provide the primary stability to the hip.[23] The fibrocartilaginous rim around the acetabulum further stabilizes the head of the femur within the joint. In horses, the ilium tends to fracture before the hip luxates.[38] Foals, miniature horses, and ponies are most frequently affected.[23,38,53,69,72] Coxofemoral subluxation and luxation are generally unilateral, and the head of the femur nearly always becomes craniodorsal to the acetabulum.[10,23]

Etiology

Both the accessory and the ligament of the head of the femur (round) must rupture for a luxation to occur.[38] Trauma is nearly always the cause. Violent overextension and falling on the point of the stifle with the femur in a vertical position occasionally produce fracture and/or subluxation or luxation of the coxofemoral joint.[10,38] A tethered horse that catches its foot in a rope or a halter may dislocate the hip in the struggle to free itself.

Because the acetabulum is deep and the head of the femur is large, excessive trauma is usually necessary to dislocate this joint. Fractures of the dorsal rim often accompany the luxation because of the deep acetabulum in the horse.[38] Additionally, absence or partial tearing of the ligament of the head of the femur may predispose to subluxation and luxation with or without associated trauma.[2,9,38]

Coxofemoral luxations may be complicated by upward fixation of the patella.[16,28,38] The upward patellar fixation may potentially contribute to the luxation or occur because of it. If it precedes the luxation, the secondary hip luxation is believed to occur as a result of the violent contraction of the quadriceps muscles trying to flex the limb while the stifle is locked in extension. The net result is that the head of the femur luxates out of the acetabulum rather than the stifle flexing.[38] The patella usually becomes unfixed at the time of the luxation. Alternatively, the patella becomes locked following the luxation because the craniodorsal position of the femur causes the limb to become much straighter, predisposing to upward fixation of the patella (Figure 5.160). Luxation of the hip also may occur secondary to wearing a full-limb hindlimb cast, especially in foals.[94]



Figure 5.160. Horse with luxation of the hip and concurrent upward fixation of the patella. The craniodorsal positioning of the femoral head straightens the limb, contributing to upward fixation.

Clinical Signs

A history of trauma resulting in a severe non-weight-bearing lameness is common; other horses present with a chronic history of lameness of varying degrees at the walk. Gluteal and quadriceps atrophy can be a common feature in many horses. Some horses may toe touch when walked because the affected limb is shorter than the opposite limb due to the craniodorsal position of the femur.[23] In the case of luxation, the limb may "dangle" somewhat because of shortening and the point of the hock on the affected side is higher than that of the opposite limb (Figure 5.142). The toe and stifle turn outward and the point of the hock turns inward (Figure 2.97). In cases of subluxation, such findings are usually not observed. Affected horses generally have a limited cranial stride because of limb shortening and a more prominent greater trochanter of the femur. Soft tissue swelling may make this prominence difficult to see in the early stages. Crepitus with limb manipulation may be present as a result of the femur rubbing on the shaft of the ilium.[38] This may also occur with acetabular and pelvic fractures. Pushing the greater trochanter in a caudal to cranial direction may displace the femur further cranially than normal when a hip luxation is present.

Diagnosis

A presumptive diagnosis can often be made based on the history and clinical signs. Radiography confirms the diagnosis and also rules out other possible causes of the lameness such as pelvic fractures, acetabular fractures, and capital physeal fractures (Figures 5.161 and 5.162).[55,56] In cases of the subluxation, radiographs should be performed with the limb weight-bearing in order to identify the problem (Figure 5.161).[100] Ultrasound is being used in the ambulatory and hospital setting to examine the pelvic region and may be able to confirm subluxation or luxation of the femoral head.[10,32] A low-frequency (2.5–5.0 MHz) curvilinear transducer should be used, and the area should be evaluated with the horse in a weight- and non-weight-bearing position.[10] The evaluation of the coxofemoral

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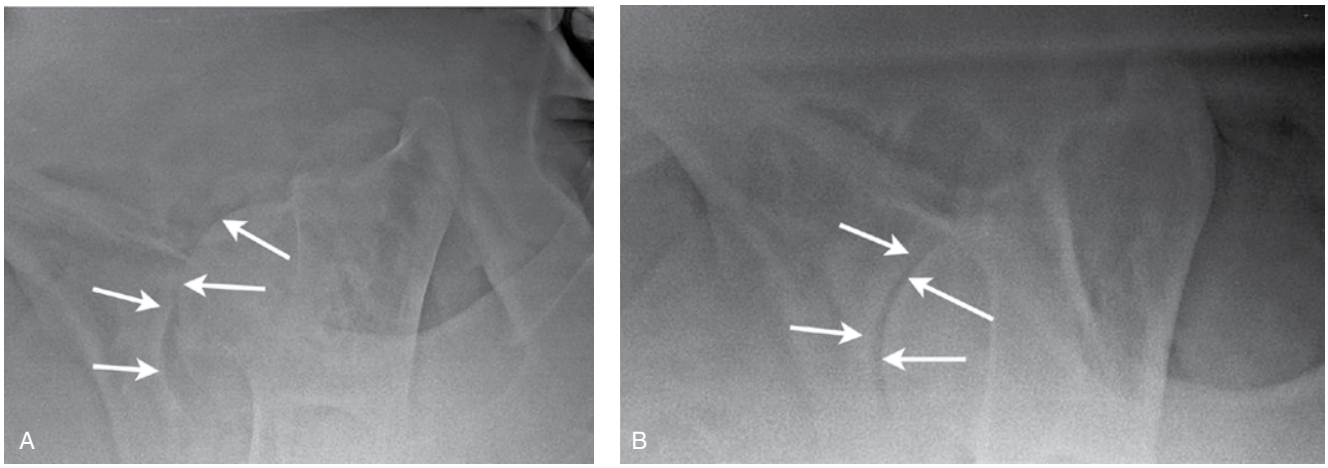


Figure 5.161. Standing, weight-bearing, lateral oblique radiographs of the pelvis in an adult horse confirming a subluxation of the femoral head (A; arrows) compared with the normal opposite limb (B).

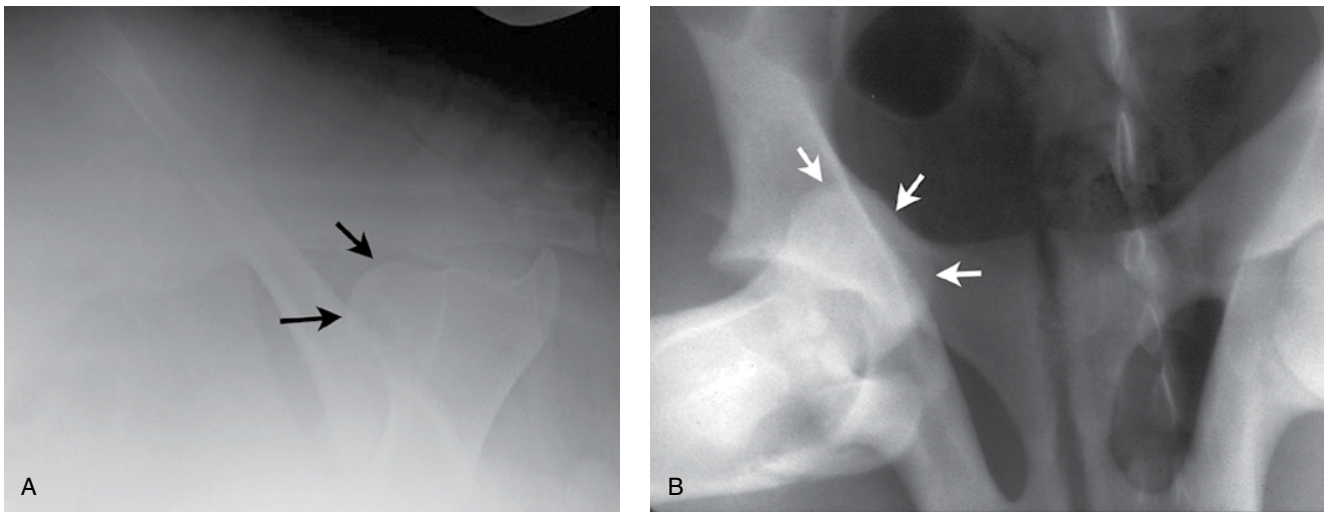


Figure 5.162. Lateral standing radiograph of the pelvis in a miniature horse (A) and ventrodorsal radiograph under general anesthesia in an adult horse (B) confirming craniodorsal luxation of the femoral head (arrows).

joint during weight-bearing is very important in the ultrasonographic diagnosis because the subluxation might only be seen in this phase. The differentiation with luxations can be based on the ability of the head of the femur to return to its normal position when horses rest the affected limb.[10] In both subluxation and luxation of the coxofemoral joint, there is usually craniodorsal displacement of the femoral head from the acetabulum (Figures 5.161 and 5.162). On ultrasound severe OA, acetabular rim fractures and severe effusion and synovitis (cranioventral recess) may also be seen.[100]

Treatment

In general, treatment of hip luxation is problematic, especially in adult horses. Many horses with this condition are euthanized because of the often unsuccessful treatment options and the poor prognosis. Closed reduction of the luxation is usually the best treatment option, but it can be very difficult to perform in adult horses and re-luxation is common. The likelihood of re-luxation after closed reduction increases if the dorsal rim of the acetabulum is fractured. If the luxation is chronic, the acetabulum may be filled with fibrin and granulation tissue, preventing a successful closed reduction. In a recent report of 17 cases of coxofemoral luxation, 35% had other orthopedic injuries, and no form of closed or open reduction was successful.[53]

Horses with an acute luxation with no secondary fracture of the dorsal rim of the acetabulum are the best candidates for closed reduction. General anesthesia with or without muscle relaxation is required. The luxation may be reduced by manual traction and

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manipulation if good muscle relaxation is obtained.[38,53] Traction also can be applied by using a hoist with the horse placed in dorsal recumbency or with a calf jack if lateral recumbency is selected. Traction combined with external rotation and adduction of the limb followed by internal rotation as the traction is reduced completes the reduction. Reduction can be appreciated when the hip clicks or pops into place. Closed reduction is often difficult, and even when it is successful, re-luxation may occur within a few days.[28,53] An Ehmer sling was used to prevent re-luxation after the second closed reduction in a pony and successfully improved the outcome.[16] However, this is only possible in foals or small-breed horses.

Several surgical approaches have been described for cases that re-luxate or those that cannot be corrected with closed reduction. However, these are most applicable for foals or small-breed horses. They include open reduction alone, transposition of the greater trochanter, femoral head and neck resection, toggle pinning, or augmentation of the lateral joint capsule with synthetic sutures attached to screws.[2,23,28,47,69,72,92] A combination of toggle pinning, synthetic capsular repair, and trochanteric transposition was used to successfully repair a hip luxation in an adult miniature horse.[28] In a case report, a hip arthroplasty was performed in an 8-week-old dwarf Friesian filly with a history of traumatic subluxation of the coxofemoral joint. Initial short-term success was reported; however the horse died 5 days after the surgery.[40] Open surgical approaches should be performed soon after the luxation occurs; otherwise, contraction of the musculature will make reduction difficult. In addition, excision of the femoral head and neck and dorsal rim of the fractured acetabulum should be considered in smaller horses to aid in the development of a pseudoarthrosis.[27,38,80]

Prognosis

In cases where there is a combination of acetabular fractures and subluxation, the prognosis is poor. In cases where the subluxation is the only problem, the prognosis can be better.[10] In horses with complete luxation, the prognosis is typically guarded to poor because successful closed reduction is not always possible. Conservative treatment is not an option and euthanasia is recommended. A minority of horses may return to complete soundness after the head of the femur is replaced, but this is the exception.[38] There is usually a better chance of maintaining permanent reduction if the femur stays in place for approximately 3 months.[38] Most horses can become sound enough for breeding purposes if the reduction can be maintained.[38] Surgical correction may be warranted if the horse is valuable and small, but is performed infrequently.

OSTEOARTHRITIS (OA) OF THE COXOFEMORAL JOINT

OA of the coxofemoral joint is a sequela to all of the conditions described in this section. It can also occur from any type of soft tissue trauma to the hip region that does not necessarily result in a ligament injury or IA fracture. Trauma to the joint capsule and surrounding musculature can contribute to the development of OA. It is seen most frequently in older animals and must be considered in any horse with chronic hindlimb lameness.

Etiology

Any hip-related traumatic injury can lead to OA. Reported causes of coxofemoral joint OA include idiopathic infection,[15] abnormal development of the coxofemoral joint,[78,94] joint ill, and trauma.

Clinical Signs

No clinical signs are specific for coxofemoral joint OA. However, hip OA should be suspected in older horses with chronic hindlimb lameness problems that have a history of trauma (Figure 5.158). Many of these horses have significant lameness (grade 3–4 on a scale of 5) and have a low arc of foot flight and a reduced cranial phase of stride. Horses with hip pain tend to move with the limb rotated externally and carry the limb abducted during advancement. Firm swelling over the greater trochanter and hip area may be present in chronic cases, and the characteristic toe-out, hock-in stance may or may not be observed. In horses with bilateral disease, the hindlimbs are often carried very straight with a shift of weight onto the forelimbs.[38]

Diagnosis

A presumptive diagnosis of severe hip OA often can be made based on the history and clinical exam. Milder cases of OA can be a diagnostic challenge. IA anesthetic of the coxofemoral joint is the best method to localize the lameness to the hip. Nuclear scintigraphy may be helpful to localize the problem to the hip but is not very sensitive nor specific for coxofemoral joint OA.[18] Radiographs are often necessary for a definitive diagnosis and usually reveal evidence of bone remodeling and osteophyte production in chronic cases (Figure 5.163). Good-quality radiographs of both hips (to permit comparison between the two) are necessary to diagnose mild to moderate hip OA. Ultrasound can also be used; however it is challenging to determine whether the ultrasonographic changes noted are result of the progression of OA or whether they are there from the initial injury.[100]

Treatment

Treatment of hip OA is usually palliative because there is no cure. Horses with unilateral mild or moderate OA may benefit from coxofemoral joint arthroscopy to debride cartilage lesions and determine the inciting cause. Focal femoral head cartilage lesions have been debrided with good results in a limited number of cases.[64] Horses with hip OA usually respond well to oral NSAIDs and/ or IA treatment with hyaluronan and

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Figure 5.163. Ventrodorsal radiograph of the pelvis of the horse in Figure 5.158 demonstrating severe OA of the coxofemoral joint. The OA was reportedly secondary to trauma to the area from a previous fall.

corticosteroids. However, IA injections are often needed every 4–6 months. Horses with mild or moderate hip OA may benefit from medications directed at joint healing such as IV hyaluronan, IM polysulfated glycosaminoglycans, and oral nutraceuticals.

Prognosis

There are no long-term reports on the prognosis of horses with coxofemoral joint OA. In general, any treatment is a temporary fix, and the prognosis for athletic use in horses with severe OA is thought to be poor. However, many of these horses can be used for breeding or maintained as pets because they tend to do well at the walk. Horses with mild or moderate OA may respond well to treatment and may be used at a reduced performance level.

CAPITAL PHYSEAL FRACTURES OF THE FEMORAL HEAD

Fractures of the capital physis of the femoral neck occur commonly in foals under 1 year of age.[25,41] They are usually a type I Salter–Harris physeal fracture (Figure 5.145), but types II (Figure 5.143C) and III are also observed.[25,41] Another term that has been used to describe them is a "slipped capital physis" (Figure 5.145).

Etiology

Traumas such as violent falls, struggles, and kicks are the cause of these fractures. In particular, falling on the greater trochanter is thought to cause shearing forces across the physis, resulting in displacement between the epiphysis and metaphysis.

Clinical Signs

There is usually a history of trauma with an immediate severe lameness. However, some foals are still able to bear some weight but often stand with a toe-out, hock-in appearance. Swelling and pain over the hip, pelvic asymmetry, gluteal muscle atrophy, and crepitus with hip manipulation may be present. However, these signs were found inconsistently in a report of 25 foals with capital physeal fractures.[41]

Diagnosis

Because of the inconsistency of clinical signs, radiography of the hip is necessary for an accurate diagnosis. Separation of the epiphysis and metaphysis of the femoral neck confirms the diagnosis (Figures 5.143 and 5.145). However, other radiographic abnormalities are often found, such as comminution of the epiphysis, greater trochanter or acetabular fracture, or coxofemoral joint luxation/subluxation.[15] Concurrent radiographic findings often make potential surgical repair unlikely. Ultrasound has also been used to diagnose a gap between the femoral head and neck with varying degrees of displacement. Careful interpretation in the appearance of the cartilage and physes is important when ultrasounding foals. The contralateral limb should be evaluated for comparison.[74]

Treatment

Nonsurgical management of these fractures is not recommended because malunion, avascular necrosis, and secondary hip OA can lead to debilitating lameness.[41,97] However, surgical treatment is difficult and is not recommended in foals with concurrent radiographic abnormalities. Surgical treatments that have been used to repair capital physeal fractures include the use of cancellous or cortical bone screws, IM or Knowles pins, and an interfragmentary compression system.[41,42,50,77,97] Small foals with a Salter–Harris type I or II physeal fractures are the best candidates for surgery.

Prognosis

Unfortunately, the majority of foals with this condition are euthanized because of the poor prognosis with nonsurgical treatment and the questionable results with surgery. If the epiphysis can be reduced and maintained until healing, athletic soundness can be achieved.

INTRA-ARTICULAR ACETABULAR FRACTURES

Acetabular fractures can occur independently or together with other fractures of the pelvis. They may be nondisplaced but more frequently are comminuted and displaced. They appear to be more common in young horses and frequently result when the horse slips or splits (spread eagle).[38]

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Etiology

Trauma is the cause in nearly all cases. Stress or fatigue fractures occur elsewhere in the pelvis such as the ilium, but are usually not confined to the acetabulum.[23]

Clinical Signs

Horses with acetabular fractures present with similar clinical signs as horses with pelvic fractures or hip disease. They usually have a significant unilateral lameness and may have gluteal muscle atrophy if the condition is chronic. Swelling, pain, and crepitus may be palpable over the greater trochanter, and there is often pelvic asymmetry if other pelvic fractures are present (Figure 5.157). A rectal examination may also reveal swelling axial to the coxofemoral joint, but it may be difficult to detect with acetabulum fractures alone.

Diagnosis

Nuclear scintigraphy can be valuable in identifying that the cause of lameness is in the hip,[18] but ultimately radiography or ultrasound is necessary to document an acetabular fracture and rule out other possible problems in the hip and pelvis (Figure 5.164).[23,32,55] Nondisplaced acetabular fractures may be difficult to visualize with ultrasound. However acetabular rim fractures are easier to diagnose with ultrasound. Both transcutaneous and transrectal ultrasound should be performed to evaluate the area. Concurrent fractures of the pelvis and hip are not uncommon.



Figure 5.164. Ventrodorsal radiograph of the coxofemoral joint demonstrating a minimally displaced acetabular fracture. Intra-articular fragmentation was not identified, and the horse was treated with stall confinement.

Treatment

Conservative treatment with stall confinement is often the treatment of choice with acetabular fractures. Euthanasia may be indicated with severely displaced fractures because there is no known surgical treatment. Small IA fractures from the cranial and caudal perimeter of the acetabulum may be removed from the joint with the arthroscope to potentially improve the prognosis.[64] However, removal of large fragments appears to rarely be successful. Minimally displaced acetabular fractures heal very well with confinement, and horses often can achieve their full athletic potential (Figure 5.164).

Prognosis

In general, horses with fractures of the acetabulum have a worse prognosis for soundness than those with fractures elsewhere in the pelvis.[23,72,75] However, most acetabular fractures respond better to conservative treatment than many other hip problems such as capital physeal fractures, luxation, and round ligament rupture. Approximately 20% of horses with articular fractures are capable of athletic performance.[76] However, this greatly depends on the fracture configuration and degree of displacement.

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6

CHAPTER

Lameness Associated with the Axial Skeleton

THE AXIAL SKELETON

Rob Van Wessum

Back pain is increasingly recognized as contributing to or even causing equine lameness.[1,4,8,16,19] Previously back pain was generally considered to be a secondary problem, with the primary lameness of a limb causing changes in movement and affecting the spine. In recent years, more and more primary spine injuries have been acknowledged.[2,8,10,11,19] To understand the function of the vertebral column, its attached tissues, and their relationship to the physics of equine locomotion, some basic anatomical and biomechanical knowledge is necessary.

ANATOMY AND BIOMECHANICS OF THE VERTEBRAL COLUMN

There are approximately 260 joints between the vertebrae; the vertebrae and ribs, the sacrum and pelvis, and the head and the first cervical vertebra all contribute to mobility in the entire spine. In most of these joints, the total range of motion is limited in both direction and degree, but the overall effect of all of the joints within the spine creates a significant amount of motion in the entire spine.[3,9] Because of its connection to the limb, a major function of the spine and the attached muscles is the positioning of the limbs. Some of the muscles controlling limb position have their origin at the spine and their insertion on an extremity; other muscles with both origins and insertions on the spine regulate mobility in the spine itself.

Joints of the spine allow mobility dorsally, ventrally, and laterally, and some allow rotation. In particular areas of the spine, the vertebrae have specific ranges of motion within the three basic axes: lateroflexion, dorsi- and ventroflexion, and rotation. Any specific change in the posture of the horse causes a change in the direction in one or more of these three planes. The cervical intervertebral joints allow a relatively large range of flexion dorsiventrally as well as laterally, while the intervertebral joint between the first and second cervical vertebra permits rotation. In the thoracic vertebrae the mobility is mainly lateral flexion, with some minor dorsiventral motion. In addition, the mobility of the costovertebral joints is necessary for breathing. In the lumbar spine the main mobility is in dorsiventral flexion with some rotation. In the sacral region there is a small amount of dorsiventral flexion, while in the tail there is considerable dorsiventral and lateral flexion but no rotation.[3,4]

The pattern of motion of the spine also is gait specific. The support and the positioning of the spine are a specific function of each gait, enhancing the range of motion of the extremities by facilitating optimal positioning of the limb relative to the spine and the other limbs. The axial muscles can function as a stabilizer of the spine and hold certain portions of the spine during locomotion. The epaxial muscles are symmetrically arranged between the transverse and dorsal spinous processes. The spinalis muscle, semispinalis muscle, and multifidus muscle form the most axial and deepest epaxial muscle group. The multifidus muscle consists of a large number of separate muscle units, each covering only two to six vertebral segments along the entire vertebral column. The middle epaxial muscular layer, called the lateral system, extends from C1 to the pelvis and sacrum. The iliocostal muscle forms the most abaxial part of this system, and longissimus muscle forms the

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medial part, which is very well developed in the lumbar region. The splenius muscle in the cervical and cranial thoracic region forms the most superficial layer of epaxial muscles.

Walk

In the walk, the main motion of the spine can be described as a "snakelike" motion. A sinusoidal pattern of motion is present in the spine with lateral flexion in the thoracic region, some rotation in the lumbar spine, and dorsiventral flexion in the cervical region, all contributing to support the walk.[5] Electromyographic evaluation has shown the action of the splenius muscles is to elevate the head and neck and facilitate forelimb protraction by the elongated brachiocephalicus muscle. The sternocephalicus muscle contributes to the stance phase of the front limb. The longissimus dorsi muscle acts during the stance phase of the hindlimb, whereas the rectus abdominus muscle is not active at all but limits vertical acceleration of the abdominal visceral mass. The multifidus lumborum muscle is active in the intermediate part of the stance phase of the ipsilateral limb, and the obliquus externus abdominal muscle shows intermittent activity.[4]

Trot

At the trot there is only modest dorsiventral flexion in the thorax and the neck. The maximal thoracolumbar extension occurs in the mid stance phase due to the visceral inertia. The splenius muscle acts before and during the first part of the stance phase of each forelimb to limit lowering of the neck, and the sternocephalicus muscle controls neck elevation. The brachiocephalicus muscle acts to achieve protraction of the forelimb. The rectus abdominus muscle limits the passive thoracolumbar extension caused by the visceral mass inertia during the stance phase. The longissimus dorsi muscles act at the end of the stance phase and during the suspension phase to induce lumbosacral extension to facilitate hindlimb protrusion. In the trot, most muscles act to stabilize the spine, not to move it.[5,6]

Canter

There is more lumbar and sacral motion in the canter than in any other gait, with lumbar rotation and dorsiventral flexion in the lumbar, thoracic, and sacral spine.[7] In the neck, the splenius muscle is active during the trailing diagonal stance phase. It limits the neck to lower and causes neck extension during the leading stance phase, and the sternocephalicus muscle shows reciprocal activity. The brachiocephalicus muscle is mainly active during the stance phase. The longissimus dorsi muscles are active during the suspension phase and during the trailing hindlimb stance phase. The rectus abdominus muscles act reciprocally during the support phase of the non-leading diagonal to support the visceral mass and initiate thoracolumbar flexion. The sublumbar muscles, psoas major and minor, are active during the suspension phase and are part of the propulsion forces in the canter.[4]

Table 6.1. Mobility of the spine.

		Part of the spine			
Gait	Axis	Cervical	Thoracic	Lumbar	Sacral
Walk	DV	++	+	+	+
	Lat	+	++	–	+
	Rot	+–	–	+	–
Trot	DV	+	+	–	–
	Lat	–	–	–	–
	Rot	–	–	–	–
Canter	DV	++	+	+++	++
	Lat	+–	+	–	–
	Rot	–	–	++	+

DV = dorso- and ventroflexion, Lat = lateroflexion, Rot = rotation, – = not present, + = minor mobility, ++ = major mobility, +++ = maximum mobility.
Source: Reprinted with permission from van Wessum,[19] p. 469.

Table 6.1 is an overview of the degrees of mobility and their relationship to the three gaits. It is easy to understand that this relationship can make a lameness examination more of a challenge. Some common symptoms are associated with specific parts of the spine, as shown in Table 6.2.

History

As in every examination, investigating the horse with back pain starts with the history.[12] Taking time for a good interview with the client is the first step to better comprehend the problem. Listening to the information provided by the client is a good start. Asking guiding questions such as whether there are time-related changes in the quality of the gaits or difficulties in keeping the correct lead can provide information about the gait in which the complaints are more evident. Common complaints from owners of horses with back problems include resistance to bending to one direction more than to another; not accepting the bridle; kicking and bucking (especially in one gait, most commonly the canter); poor-quality canter, cross canter, or switching leads; or generalized stiffness.[1] The poor-quality canter may be a no-sound, 4-beat canter, more "bunny-hop-like" with less separation between the subsequent footfalls of the hindlimbs.

Some complaints are specific to the discipline. Complaints indicative of back pain in dressage horses include difficulties in the walk and canter pirouettes, a poor quality or a lateral walk, and unsatisfactory collection and "coming under" (carriage of the hindquarters). In jumpers, common complaints are unsatisfactory bascule (balance above the fence), lack of

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Table 6.2. Symptoms and locations.

	Location within spine			
Symptom	Cervical	Thoracic	Lumbar	Sacral
Gait abnormalities				
Lateral walk	Rare	Sometimes	Often	Often
Bunny hop canter	Rare	Sometimes	Often	Often
Cross cantering	Rare	Sometimes	Often	Often
Toe dragging behind	Rare	Rare	Often	Often
Narrow behind	Rare	Rare	Sometimes	Often
Wide behind	Rare	Sometimes	Often	Rare
Hip lowered	Rare	Rare	Sometimes	Often
Tail fixed to one side	Rare	Rare	Sometimes	Often
Head out on circles	Often	Rare	Rare	Rare
Front limb lameness	Sometimes	Sometimes	Rare	Rare
Reduced stride length, behind	Rare	Sometimes	Often	Often
Reduced stride length, front	Sometimes	Sometimes	Rare	Rare
Behavioral issues				
Downward transition difficult	Rare	Often	Often	Often
More complaints when ridden	Sometimes	Often	Often	Often
Refusal to accept the bit	Often	Sometimes	Sometimes	Sometimes
Refusal to jump	Sometimes	Often	Often	Often
Poor lateral bending	Sometimes	Sometimes	Often	Often
Poor collection	Sometimes	Rare	Often	Often
Resistance when tacked up	Rare	Often	Sometimes	Sometimes
Collapsing in hindlimbs	Rare	Rare	Sometimes	Sometimes
Rearing	Rare	Rare	Sometimes	Rare
Poor engagement of hindquarters in trot	Rare	Often	Sometimes	Sometimes
Poor engagement hindquarters in canter	Rare	Rare	Often	Often
Bucking	Sometimes	Often	Often	Often

Source: Reprinted with permission from van Wessum,[19] p. 471.

propulsion and power, unwillingness to jump and/or turn, and landing in the wrong lead canter. The quality of the lope is a complaint in Western pleasure horses. In racing horses, back pain is often located in the lumbar region, which can limit dorsiventral flexion, thus decreasing propulsion and reducing the efficiency of the canter and gallop and ultimately leading to poor performance.

Other usual lameness issues also may be mentioned. Forelimb lameness can be a result of thoracic problems because the attachment of the scapula to the thoracic spine causes painful mobility in the thoracic vertebrae.[4,13,15] A reduced stride length of a hindlimb or lack of propulsion can be caused by pain in the lumbosacral region,[10,11,14,15] and a lowered hip can be a sign of back pain as well as an indication of hindlimb lameness.

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Clinical Examination

Clinical examination of the horse is performed as with any lameness case, with more emphasis on observation of lateral bending in walking serpentines and circles at the walk, trot, and canter. The quality of the walk (4-beat) and canter (3-beat) can give essential information about location of a lesion within the spine due to the specific loads that are generated by these gaits to specific parts of the spine (Table 6.1).

In the walk and trot in hand on a straight line, observation of the gait and the position of the hindlimbs in relation to the front limbs and the position of the tail can assist in assessing for the presence and location of back pain. For example, do the hindlimbs track the forelimbs, and are the haunches more to one side? Because there is more mobility of the lumbosacral spine in the canter, lumbosacral pain is more obvious at this gait. By contrast, thoracic pain appears more as stiffness in the trot and resistance to going downhill, or in making downward transitions from canter to trot or trot to walk. More detailed signs and the locations they may identify are presented in Table 6.2.

Information gleaned from palpation of the back can be confusing. Most horses with any kind of back pain exhibit some muscle tension in the epaxial muscles. It is nearly impossible to differentiate between muscle tension caused by guarding spinal structures and primary muscle tension due to muscle problems. See the section on muscle diseases in Chapter 7.

Sensitivity to touch or pressure is another symptom that can be difficult to interpret. Some horses initially show a defensive reaction to every contact with their back, even when there is no pathological condition present. To make an initial differentiation, touch or apply pressure very gently and when the horse resists, maintain it and wait for the initial reaction to fade. In behavioral issues, when the pressure persists without correction or change, quite often the initial resistance will diminish. With pain, the same reaction will be shown over and over again.

Manual compression of the muscles in the withers area can give information about sensitivity of this region, which can indicate saddle fit issues (see the section on saddle fit in Chapter 10), as can spots of white hairs in this region. Bald spots or spots with damaged hairs are another indication of poor saddle fit as a cause for back pain and related muscle tension.

Specific Tests

Passive mobility tests should be performed to assess the mobility of the spine and the range of motion within every part of the spine.[18] Symmetry of range of motion is very important because muscle tension, which is a manifestation of pain, impedes symmetrical movement.[17] Gentle bending of the neck to the left and right, with one hand of the examiner on the head of the horse and the other fixing a specific cervical vertebra, provides information about the intervertebral mobility as well as the range of motion to the left and the right (Figure 6.1). A carrot or other palatable treat used to tempt the horse to move its head can indicate the willingness of the horse to bend its neck laterally and dorsoventrally. Lifting the head allows assessment of the amount of dorsiflexion as well as the symmetry of the muscles and location of anatomical landmarks on the ventral aspect of the neck (Figure 6.2).

In the thorax, a similar manipulation (pulling on the tail with one hand and placing the other arm as a fulcrum at an individual horizontal process of a specific vertebra) provides information about the range of motion and intervertebral mobility in the lateral plane (Figure 6.3). Palpation of the withers, dorsal processes of the thoracic vertebrae, and associated muscles indicate pain in this region that is very susceptible to discomfort caused by poor saddle fit. Triggering the dorsiflexion reflex by pressure with a pen in a paramedian motion



Figure 6.1. Flexion of the neck to the right. The left hand of the examiner is at C6, while the right hand gently asks for lateral motion.



Figure 6.2. Extension of the neck. The examiner lifts the head of the horse, evaluating the extent of stretching of the neck and symmetry of the cervical vertebrae in a ventral aspect.

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Figure 6.3. Lateroflexion of the caudal thoracic spine. The examiner pulls on the tail with the left hand to gain lateral excursion of the spine, while the right hand functions as a fulcrum on the transverse process of T17 so the mobility between T17 and the more caudal spine can be evaluated.

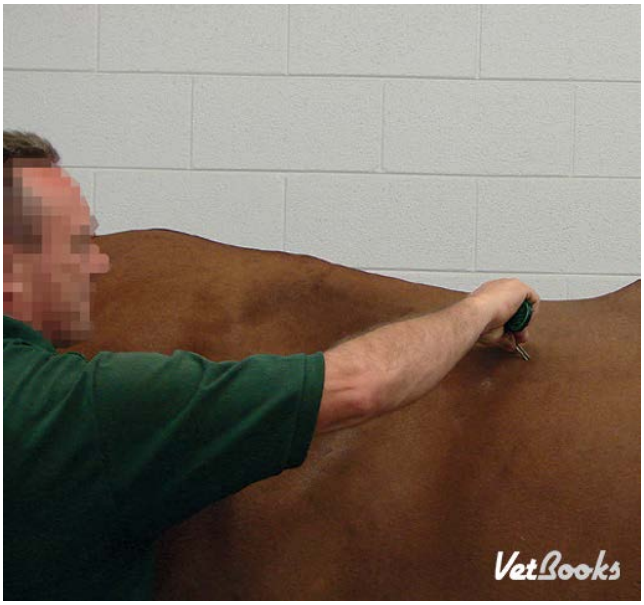


Figure 6.4. Moving a key on the back from cranial to caudal in a paramedian line checks for the reflectory movement of the horse with dorsiflexion and some lateroflexion.

from cranial to caudal (Figure 6.4) gives information about dorsal mobility in the thoracolumbar spine as well as symmetry in these reflexes. The same technique applied to the ventral aspect of the thorax and abdomen provides information about ventral mobility. An evasive reflex to these procedures is normal and absence of such reflexes can be neurological in origin (lack of skin sensation) or a consequence of pain that occurs when the spine is in motion.



Figure 6.5. Lateroflexion of the lumbar spine, as in Figure 6.3, now with the hand as a fulcrum on the transverse process of L3.

Specific manipulation techniques in the lumbosacral region can yield information about the mobility of the spine. Lateroflexion of the lumbosacral spine and rotation of the lumbar spine can be tested by pulling intermittently on the tail base with one hand while fixing the spine at the tuber coxae with the other "fulcrum" hand (Figure 6.5). Information is obtained about the mobility of individual intervertebral regions by moving the fulcrum hand more cranially to the lateral process of each lumbar vertebra. Introducing a wavelike motion in the spine by gentle but powerful manipulation between tail base (pulling) and the tuber coxae facilitates evaluation of tension in all epaxial muscles. This wave can be observed to move over the spine to the head in a horse with relaxed muscles. Abruptly halted motion of this induced wave is a good indicator of higher muscle tension in that part of the spine.

Specific tests to localize pain include manual pressure on the individual dorsal processes of the thoracic and lumbar vertebrae (Figure 6.6) and on the tuber sacrale (Figure 6.7), as well as testing for sacroiliac pain by lateral manipulation of the pelvis. The latter provocation test introduces rotation and friction in the sacroiliac joints, so a positive response to the test is indicative for sacroiliac pain. The tests are executed with one hand on the tuber coxae and the other hand on the tuber ischium to manipulate the pelvis in cranial, ventral, dorsal, and caudal directions to cause friction and rotation in the sacroiliac and lumbosacral regions (Figures 6.8 and 6.9). Finally, standing on a step stool can facilitate inspection of the entire spine. The higher position permits more thorough evaluation of the spine for potential deviations that have not been noticed previously.

PRIMARY VS. SECONDARY BACK PAIN

A common topic in equine back pain discussions is whether the back pain is primary, caused by a primary lesion in the spine, or secondary, due to altered use of the

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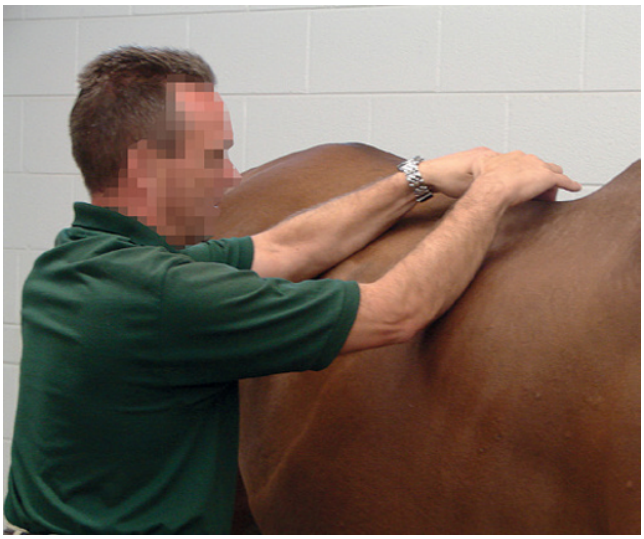


Figure 6.6. Digital pressure on the dorsal process of T16. Both index fingers are located on the dorsal process with strong downward pressure to place load on the intervertebral ligament, facet joints, and the intervertebral disc.

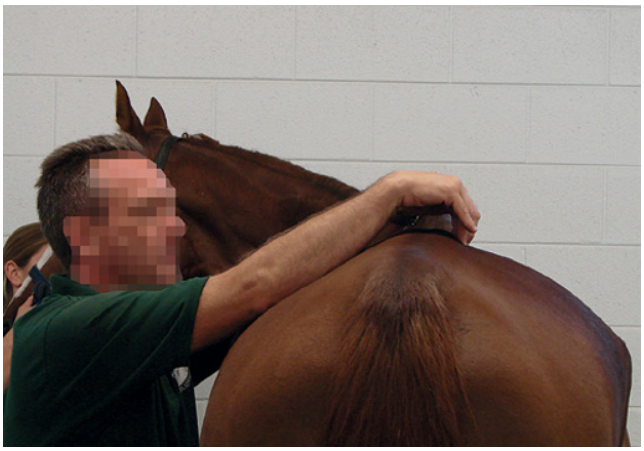


Figure 6.7. Digital pressure on the right tuber sacrale. Pressure is placed downward and slightly to the median so pressure is placed on the bone, dorsal sacral ligaments, and sacroiliac joint.



Figure 6.8. Provocation test for sacroiliac joint pain. The left hand of the examiner is on the right tuber ischium, and the right hand is on the tuber coxae, making rocking motion in the cranial direction.

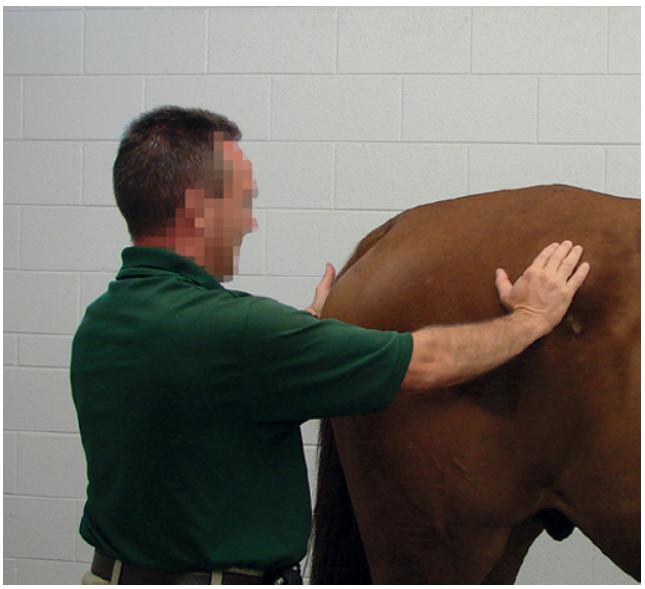


Figure 6.9. Pain provocation test for sacroiliac joint pain, making a rocking motion in the caudal direction.

spine that stems from limb lameness. In humans, contrary to the belief that secondary back pain is very common, there is little literature on this topic other than some papers on the influence of leg amputation or secondary back pain due to tumor metastasis in the spine. One can assume that specific changes in gait in humans can eventually lead to (over)compensation in the spine, resulting in secondary pathology in the spine. However this requires a considerable duration of time to occur because this phenomenon typically presents in people in their 50s. One could question whether secondary pathology of the spine has time to develop in horses, since most performance horses are much younger, between 4 and 16 years of age.

In the author's experience, in cases with clear pathology of the spine such as facet joint osteoarthritis, pathology of the intervertebral disc, desmopathy of spinal ligament structures, and sacroiliac disease, as well as cases with fractures of spinal structures, very often in the history, there is an incident that indicates significant trauma to the spine, resulting in later symptoms of back pain. In the author's opinion, these are clear cases of primary back pain. When the pathology is located more in the soft tissues of the spine, with strain of spinal ligamentous structures or muscles, it is more difficult to assess whether this is primary or secondary back pain.

Complete physical examination is necessary to evaluate the complete condition of the horse and assess potential sources of coexisting limb lameness (primary or secondary). In the author's experience, when no clear pathology is found in the spinal structures and coexisting limb lameness is present, back pain is secondary. When the coexisting limb lameness is treated, the accompanying back pain diminishes or dissolves.

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THE PELVIS

Rob Van Wessum

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FRACTURES OF THE PELVIS

Fractures of the pelvis are rather uncommon, ranging from 0.9% to 4.4% of all equine lameness cases.[15] Most reports in the literature are from before 2000, and in the last few years no real new information about treatment of pelvic fractures has emerged. Therefore, the emphasis in this chapter is given to diagnosis and imaging modalities.

Diagnosis of pelvic fractures can be a challenge in practice. In most cases, therapy focuses on stall confinement and adjuvant therapy rather than invasive surgery.[16,26] However, for prognosis it is important for the veterinarian to have a confirmed diagnosis. Some pelvic fractures have a favorable prognosis for return to an athletic career; others only guarantee a breeding career. Some fractures are accompanied by severe pain and invalidation of the animal; therefore, euthanasia is a favorable option.

Etiology

Most pelvic fractures are traumatic fractures; common causes include falling, slipping, fighting, and accidents with cars or in trailers. The history of a sudden onset of the lameness is usually a clear indication of a traumatic event. Often the event is observed, but simply finding the horse very lame in the field is a common finding in the history.

However, ilial wing fractures are an example of common stress fractures caused by repetitive overloading of certain portions of the pelvis. These occur in young, immature Thoroughbred racehorses. The lameness is usually less severe with a more gradual onset than that of horses with a traumatic injury.[2,6,7,9,18,22,23,30]

Clinical Examination and Signs

Clinical examination of the horse with a suspected pelvic fracture starts with a complete inspection of the horse, emphasizing symmetry of the hindquarters. Important observations include muscle atrophy, position of the tuber sacrales, limb length, and pelvic height (height of the tuber coxae). The tuber coxae height is easier to determine when standing behind the horse and palpating both tuber coxae, left and right, to exactly determine their position. For accurate inspection, the horse should be taken out of its stall and made to stand completely square on a firm, level surface. This may be difficult when the horse is severely lame.

Particular attention should be paid to limb position; an abnormally straight limb may reflect luxation of the coxofemoral joint and secondary upward fixation of the patella. Toe-out, hock-in position is more often found with pelvic fractures. The muscles of the lumbar and pelvic regions should be assessed carefully to identify abnormal muscle tension and pain as well as swelling or hematoma. Firm manual or digital pressure on bony prominences such as the tuber sacrale, tuber coxae, and tuber ischium should be applied to identify pain or defensive reactions. Keeping contact with the hand on these bony prominences when the horse is asked to walk can assist in detecting crepitation or abnormal (and asymmetrical) motion of the pelvic region. Auscultation during motion also can reveal crepitation.

Careful examination of the pelvic canal per rectum is indicated to assess the aorta and iliac arteries; psoas muscles; and the caudal aspect of the ilial shaft, pubis, and ischium. Some authors advocate rectal examination, while the horse is rocked or even walked, but this can be dangerous for the horse (risk for rectal tear) and examiner (when horse acts up or falls). This author does not recommend such examination.

Most horses with pelvic fractures have a significant lameness, depending on the duration of the fracture. Most are lame at the walk, and gluteal muscle atrophy tends to occur more quickly in horses with pelvic fractures than with other severe lameness conditions of the hindlimb. Horses often stand with the affected limb outwardly rotated (toe-out, hock-in stance) and often lean away from the affected limb. Pelvic asymmetry, muscle atrophy, and pain and crepitus on manipulation of the pelvis are common clinical findings. The exceptions are horses with stress-type fractures or other types of nondisplaced fractures.

Diagnosis

Diagnostic imaging involves radiography, ultrasonography, and scintigraphy. Particularly when the horse is reluctant to move and transportation to the clinic is difficult or dangerous, on-site imaging is needed to make decisions about first aid and follow-up examination. Standing radiography with mobile equipment is possible[8,20,22] but does not always give a final diagnosis. More complete radiography in dorsal recumbency under general anesthesia may be needed, with all of its associated risks during recovery.

Ultrasonography can be performed on site and is capable of imaging ilial wing fractures, ilial shaft fractures, and acetabular fractures with percutaneous probe placement. Transrectal ultrasonography can be used to diagnose acetabular and pubis fractures.[1,13,14,22,31] Due to the risks associated with radiography under general anesthesia and the potential for ultrasonography to confirm a tentative diagnosis of pelvic fracture, radiography has fallen out of favor as the first choice of imaging of horses suspected of having a pelvic fracture, in the author's opinion.[6,12] Ultrasonography has the advantage of not adding to the risks associated with transport when the horse must be taken to a facility for radiography. Most portable ultrasound equipment, in the hands of skilled clinicians, can confirm the diagnosis of pelvic fracture (PA Pease and MB Whitcomb, personal communications). When radiography is available at the location where the horse is presented for evaluation, it is still

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very useful, especially when the procedure can be performed in the standing horse.

On-Site Management

Because of their traumatic origin, many of the pelvic fractures occur during a workout, in competition, in a race, or in the pasture. With the more serious injuries, such as ilial shaft and acetabular fractures, the horse may be down or impossible to move. In these cases it is good practice to establish a preliminary diagnosis with a thorough clinical exam, which should at least include a rectal and/or vaginal exam. Crepitation and major swelling in the pelvic region at least can provide a severe suspicion for pelvic fractures, but not confirmation. Transportation to a clinical facility to perform more diagnostics can be contraindicated or at least undesirable. In the field, ultrasonography can be a perfect technique to confirm the diagnosis of a pelvic fracture and make transportation unnecessary.[1,13,22,31]

Treatment and Prognosis of Pelvis Fractures

Treatment for pelvic fractures is conservative, including with anti-inflammatory medication (NSAIDs).[6,15,21,24,26,29] Strict stall rest for at least 30 days is most important. Some clinicians advocate tying the horse in its stall for periods of up to 30–60 days to minimize the chance of a complete fracture when getting up. In general, any horse will lie down after some time, tied or untied. Therefore, tying the horse may increase the risk of other injuries associated with trying to lie down or getting up when tied. Furthermore, stress levels of horses that are tied up, or the public opinion about the possible stress this procedure can evoke in the horse, decrease the acceptability of this procedure.

Normal bone healing of pelvic fractures takes 2–3 months, so motion must at least be restricted during this period. Brief periods of hand walking (with the horse under control) can assist in keeping muscle tension and development more progressive than when complete stall rest is recommended. The use of hot walkers or treadmills must be avoided, because there is no permanent control with these types of rehabilitation equipment. Use of this equipment risks the chance of a brief moment of joy or fear, in which the horse can damage itself severely. Ultrasonographic examination can be a good technique to monitor healing and determine when to increase the workload.[1,31]

It is important to realize that complete healing of pelvic fractures (e.g., tuber coxae or tuber ischium fractures) and the adaptation of the adjacent structures to a changed anatomical conformation can take several months to a year. Sometimes it is possible after this period to give a final prognosis for the return to an athletic career. It is a good practice to communicate this at an early stage with the owner and/or trainer so they are aware that the final outcome after such a long period of treatment and rehabilitation may be unfulfilling. A recent paper however showed a better prognosis for Thoroughbreds with pelvic fractures than previously reported, so it is wise to incorporate these findings in your communication with clients.[21]

SPECIFIC TYPES OF PELVIC FRACTURES

Ilial Wing Fractures

Ilial wing fractures are most commonly found in the young Thoroughbred racehorse, age 1–2.5 years, with an immature skeleton. Lameness is sudden in onset and can vary in severity from 2 to 5 on a scale of 5, according to the AAEP grading scale. The horse has the tendency to plait with the hindlimbs or cross over the hindlimbs in a trot. The severity of the lameness diminishes or decreases in 24–48 hours. Profound muscle atrophy can occur within the first 2 weeks after injury, which can give the horse a very asymmetric appearance when the injury is unilateral. When a complete fracture is present, the ipsilateral tuber sacrale can be displaced. Manual pressure on the tuber sacrale (see the section on clinical examination above) can be painful, as when sacroiliac injuries are present (see the section on the sacroiliac region below).

The major risk in these cases is when an initial incomplete fracture extends to a complete fracture. When this is the case bilaterally, the horse can become recumbent with the chance for adjacent severe neurological damage or a catastrophic injury during workout or race. Therefore, correct diagnosis is important.[26,28,30]

Ultrasonography is the first choice for a diagnosis in a practical field situation.[1,13,22,31] With a 3.5–5 MHz transducer, nearly all commonly available ultrasound equipment will facilitate imaging of the ilial wing. Moisturizing the skin and coating it with alcohol is all that is necessary to prepare the site for imaging. A better image can be obtained by clipping the coat before imaging. Following the contour of the ilial wing with the probe in a craniocaudal position and moving the probe from tuber sacrale out to the tuber coxae provides an outline figure of the ilial wing, and steplike changes can be observed at the fracture site (Figure 6.10).

Scintigraphy is very useful for evaluating incomplete stress fractures (Figure 6.11) as well as evaluating the adjacent structures of the tuber sacrales and sacroiliac joints for involvement. In most cases, scintigraphy is the most sensitive technique 5–10 days after initial injury.[10,12,20] Prognosis in most cases is good for a full recovery to an athletic career when no involvement of the sacroiliac joint(s) is present. When sacroiliac joint involvement is present and not diagnosed correctly, osteoarthritis of the sacroiliac joint(s) can be career limiting at a later stage. See the sacroiliac region section.

Tuber Coxae Fractures

Tuber coxae fractures are nearly always associated with trauma, such as running into a door post or fence, fighting, and kicking, and trailer accidents.[15] In cases of high-energy impact to the tuber coxae (e.g., high-speed collisions), adjacent structures in the sacroiliac region may be involved and require attention later. See the sacroiliac region section. Tuber coxae fractures are associated with moderate to severe lameness that decreases to mild lameness in 24–48 hours.

These fractures are fairly easy to recognize because the contour of the tuber coxae is changed (Figures 6.12 and 6.13). The fractured portion moves cranioventrally due to traction of the internal abdominal oblique muscle.

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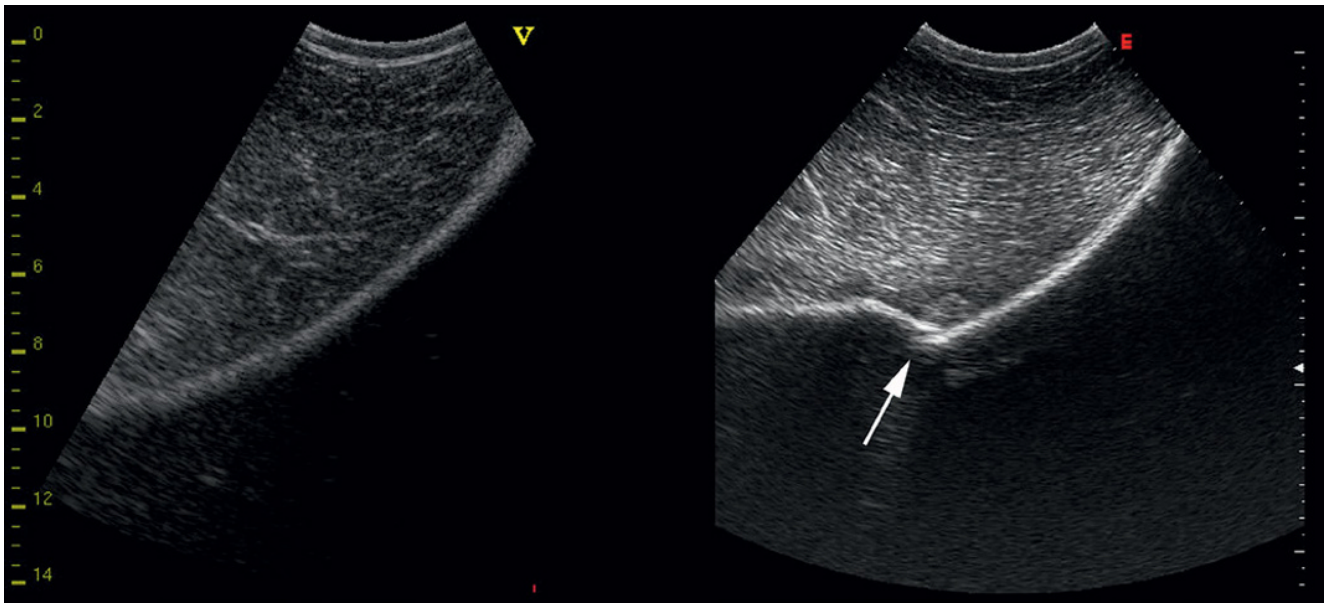


Figure 6.10. Ultrasonographic image of an ilial wing fracture. Left, the sonogram of a normal ilial wing. Right, an ilial wing fracture (arrow). *Source*: Courtesy of Dr. Mary Beth Whitcomb.

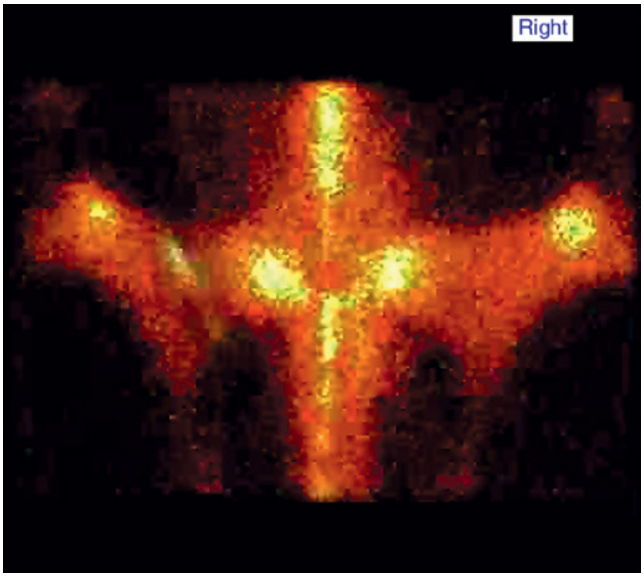


Figure 6.11. Scintigraphy image of a stress fracture of the left ilial wing. Increased radiopharmaceutical uptake (IRU) in the left ilial wing, as well as IRU at both tuber sacrales and the right tuber coxae, is indicative for trauma to these structures.

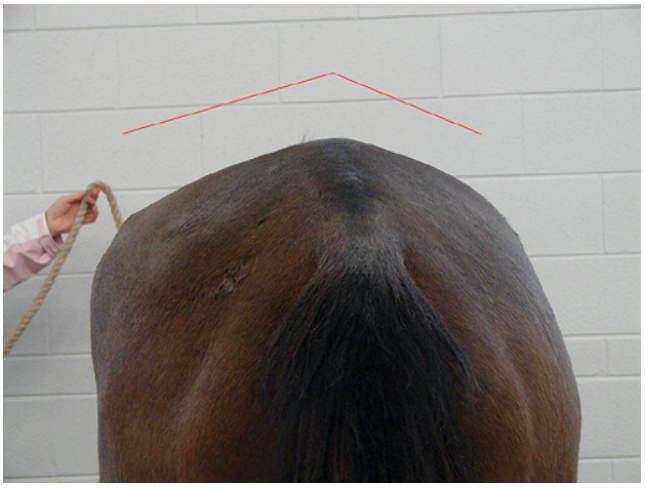


Figure 6.12. Fracture of a large part of the right tuber coxae. Note the shorter distance between the right tuber sacrale and tuber coxae compared with this distance on the left.

The edges of the remainder of the tuber coxae are palpable through the skin. In rare cases the sharp edges of the ilium can penetrate the skin and cause a fresh laceration. In a later stage, sequestration can cause draining tracts or nonhealing wounds, and the sequestrum must be removed.

Ultrasonographic examination is again the first choice for imaging, because it can show the irregular bone edge of the ilium and the displaced part of the tuber coxae (Figure 6.14).[1] Ultrasonography can identify a draining tract when present and provide a guide to the sequestrum. Radiography in this region can be disappointing; however, sometimes a mediolateral oblique projection can show the fragmentation of the tuber coxae.[8,18,20,26] A recent study described a dorsomedial–centrolateral 50° oblique radiographic view in the standing horse as being very helpful.[8]

Scintigraphy is a very sensitive technique to show involvement of the tuber coxae[12,20] but is unnecessary in most cases, at least in the initial diagnostic procedure. Later, when sacroiliac problems are suspected due to the initial trauma, scintigraphy is the first choice for imaging. The prognosis is favorable in most cases. A fibrotic and functional union between the dislocated

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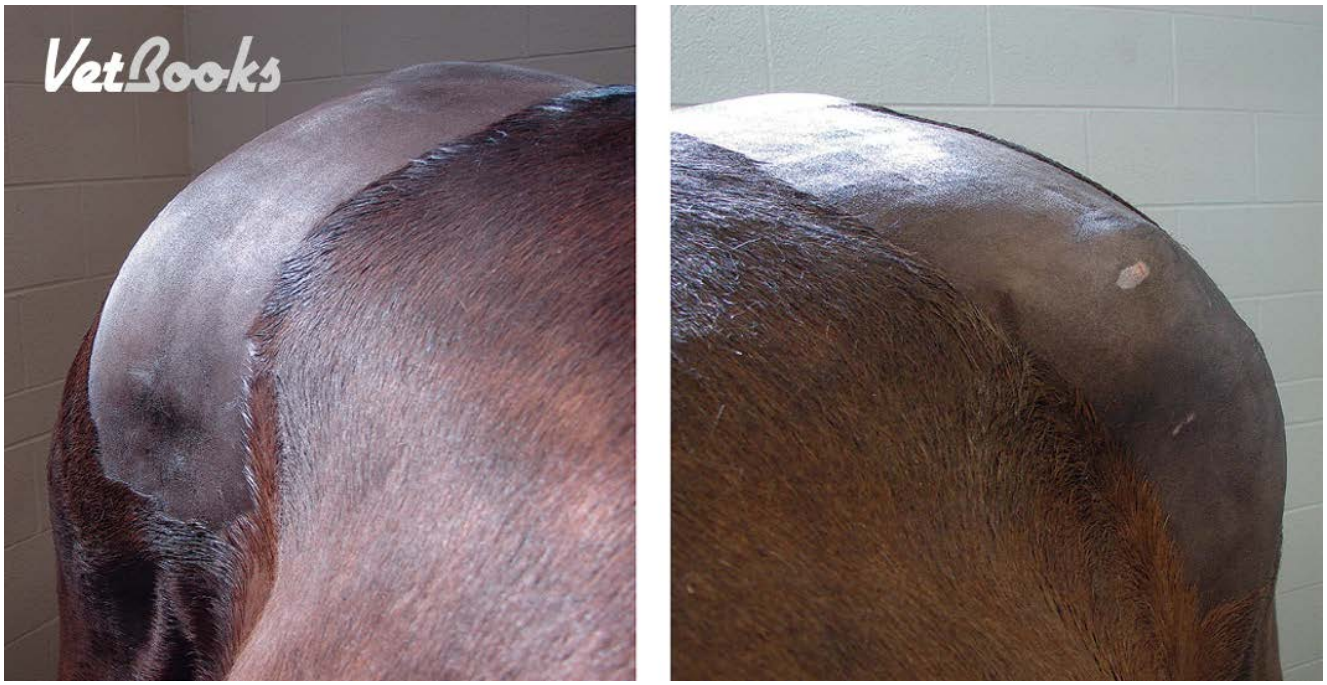


Figure 6.13. Images of the horse in Figure 6.12, right and left side views. Note the rounded aspect of the right tuber coxae and the atrophy of the quadriceps muscles.

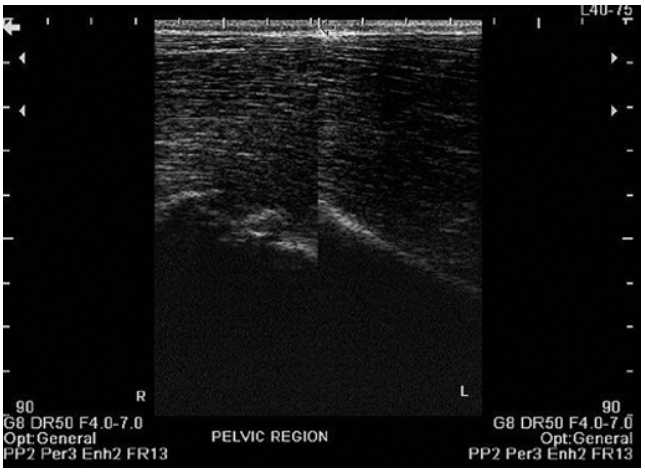


Figure 6.14. Ultrasound image of the same horse as in Figure 6.13. Note the disrupted contour of the ilial wing just medial to the normal position of the right tuber coxae.

fragment and the adjacent structures facilitates recovery to full athletic performance. In a recent study, up to 93% of the horses returned to athletic performance.[8] For higher level dressage, however, prognosis is less favorable due to the asymmetric position of the pelvic region. The horse appears to be asymmetrical and gives the judge the impression that it cannot do certain exercises. Some minor gait asymmetry (altered motion pattern of the hindlimb(s) and slight change in stride length and path) can affect scores at the higher levels of dressage competitions, when judging is more strict and sensitive to small gait irregularities.

Fractures of the Pubis and Ischium

Isolated fractures to the pubis and ischium are rare. In cases of severe trauma to the pelvic region with high-energy impact (e.g., fall, car accident), fractures to these structures can be complementary to fractures of other pelvic structures. When they are isolated fractures, they often cause unilateral hindlimb lameness combined with clear swelling of the thigh area. After a few days, the swelling can progress downward due to gravity and cause remarkable swelling around the stifle, femur, or hock.

This descending swelling can lead the examiner away from the initial injury and place suspicion at the stifle or hock as being the initial injury site.[5] With palpation it is often quite difficult to feel crepitation because of the muscle spasm that accompanies the injury. The injury is very painful, and muscle spasm is very common, sometimes leading to an asymmetric position of the tail or even the entire pelvic region. Sweating can be noticed, sometimes in localized patches, especially when nerve involvement is apparent.

Scintigraphy can be used to diagnose these types of fractures (Figure 6.15). Again, this technique is very sensitive but should be done 1–2 weeks after the initial trauma. Ultrasonography can be used to identify displaced fragments of the tuber ischium (Figure 6.16), often after scintigraphy has identified the region of interest. Prognosis for isolated fractures of the tuber ischium is favorable for a return to an athletic career. The exceptions are horses competing in upper level dressage, as mentioned above in the tuber coxae fractures section, for the same reasons.

Ilial Shaft Fractures

Ilial shaft fractures are associated with non-weight-bearing lameness.[24] The horse is extremely painful, with sweating, and has severe muscle spasms. When the fracture ends of the

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ilial shaft damage the internal iliac artery, shock and exsanguination can occur. When the fracture is complete, the tuber coxae will be asymmetric, and the affected limb will appear to be shorter. Rectal or vaginal examination reveals a possible hematoma and crepitation. Many of these fractures are multiple fractures that involve the pubis, ischium, and ilium. Prognosis is unfavorable, and often euthanasia is performed.

Acetabular Fractures

Acetabular fractures are traumatic in origin (e.g., falling, slipping, trailering accidents) and very painful. In most cases the horse is non-weight-bearing lame and reluctant to move at all. Some horses are found non-weight bearing standing in the pasture or paddock without any clear signs of the origin of the trauma. Crepitation can be found with a rectal or vaginal examination, especially when the horse is rocked a bit during the examination. Profound swelling can also be felt with the rectal or vaginal examination. Sometimes swelling is noticeable around the stifle and thigh area. Ultrasonography[1,31] (transcutaneously or transrectally) can assist in the diagnosis when there is a complete fracture (Figure 6.17). Radiography under anesthesia can confirm acetabular fractures but adds the challenge of safe recovery afterward. Scintigraphy is the most sensitive technique but should be performed at least 7–10 days after the initial cause; otherwise, the

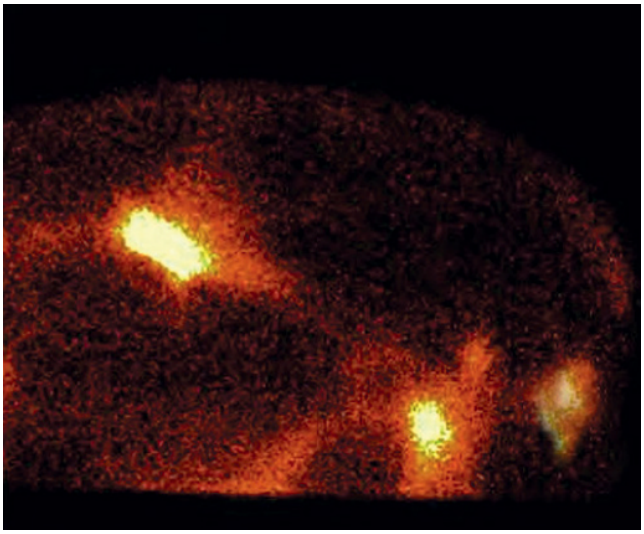


Figure 6.15. Lateral scintigraphy image of the hindquarters. Increased uptake is visible in the region of the tuber ischium.

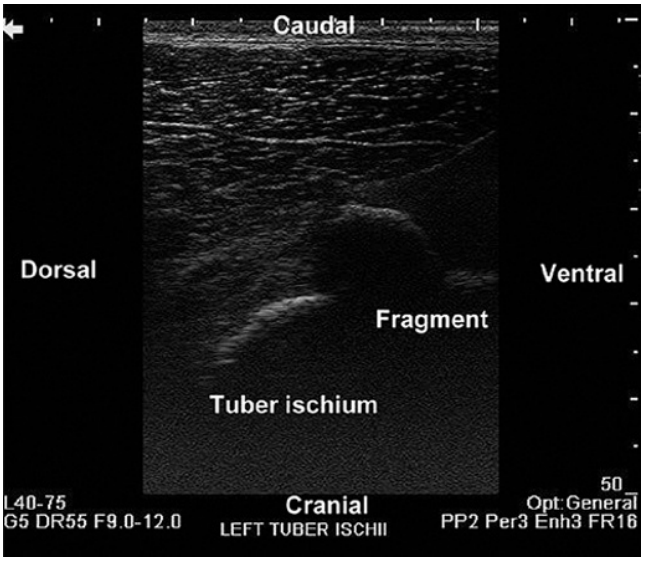


Figure 6.16. Ultrasound image of the tuber ischium of the horse in Figure 6.15. A large fragment that is avulsed from the tuber ischium is visible and is displaced ventrally. Note the attached muscle pulling it ventrally.

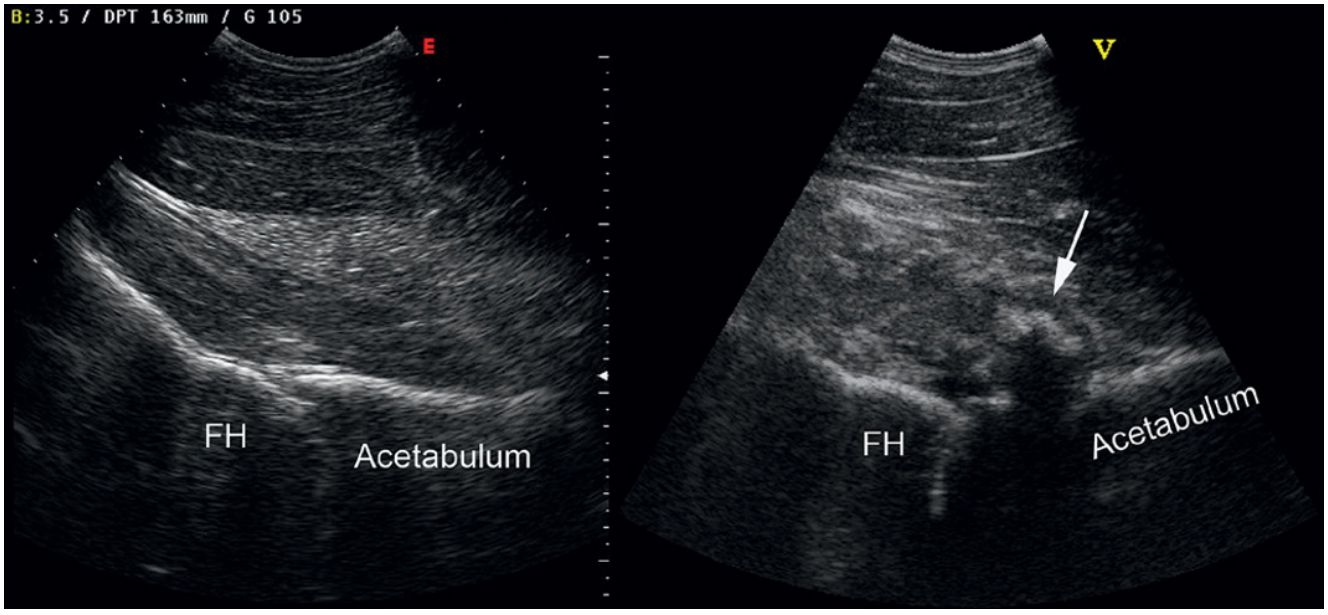


Figure 6.17. Ultrasonographic image of an acetabular fracture. At the left is a normal image, whereas a fracture of the acetabulum (arrow) can be seen on the right. FH = femoral head. *Source*: Courtesy of Dr. Mary Beth Whitcomb.

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Figure 6.18. Radiograph of a horse with bone proliferation between the coccygeal vertebrae 1 and 2 at the dorsal aspect as the result of an avulsion fracture of the intervertebral ligament.

chance of false-negative findings is great. Prognosis is unfavorable because of the chance of development of osteoarthritis of the coxofemoral joint, leading to permanent and progressing lameness.[22]

Fractures of the Sacrum and Coccygeal Vertebrae

Fractures of the sacral bone (primarily dorsal processes) or coccygeal vertebrae are rare but can cause lameness. When the proximal coccygeal vertebrae are involved, the normal locomotion pattern at the walk (the snakelike motion) and the canter can be influenced by pain in this region. Backing up (lowering the tail region) may be painful, and the horse may be reluctant or completely refuse to back up. Often the owner regards this as a dominance problem more than a physical issue. Some owners and trainers may think the horse's unwillingness to back up is showing the horse is not respectful to the handler and they do not understand there is a physical cause for this behavior (many disciplines use backing as an obedience test). Clinical examination can show enlargement of the soft tissues in the sacral or coccygeal region, and passive mobility can be painful. Radiography can be used to image the fracture site when coccygeal vertebrae are involved (Figure 6.18), as can a lateral image for sacral dorsal process fractures. Ultrasonography is also a good tool for imaging this region, but scintigraphy is the most sensitive.

Treatment can focus on anti-inflammatory therapy and reducing the workload. When nerves are involved, caudal epidural steroid administration has been used to treat this condition successfully.[17] Methylprednisolone acetate (200 mg) mixed with 10–20 mL of saline can be used to perform an epidural injection. Surgery may be indicated when fracture fragments are displaced or sequestration occurs. The prognosis is generally good for a return to athletic performance. Sometimes coccygeal fractures are a coincidental finding during a prepurchase examination, because they do not cause clinical symptoms.

THROMBOSIS OF THE CAUDAL AORTA OR THE ILIAC ARTERIES

Etiology

Thrombus formation in arteries of the limbs can cause blood supply interruption. Specific locations for these thrombi are the caudal aorta, iliac arteries, femoral artery in the hindlimb, and very rarely the brachial artery in the front limb. Circulatory problems in the muscles (and later in all tissues) can cause pain, resulting in lameness. The etiology has been described as being caused by the migrating larva of *Strongylus vulgaris*, but it is not clear why only these locations are affected. It is more likely that any damage to the intima of the blood vessels can cause a thrombus to form.

Clinical Signs and Diagnosis

This lameness is specific in that it occurs after or during work, is sudden in onset, and can be very severe (grade 3–5 of 5) but can diminish or disappear within minutes to hours after ceasing work. Because of the severe pain, horses may sweat, show anxiety, and be very restless, which can make the condition look like azoturia or even colic. Blood chemistry and a clinical workup for colic are negative.[11,16]

Clinical examination shows lower perfusion in the affected limb, lower temperature (thermography can be useful in these cases), and absence or lower pressure of arterial pulse. On the nonaffected limb, the veins can show clearly, while they are indistinct on the affected limb. Ultrasonography can show lower blood flow in the blood vessels of the affected limb (using Doppler).[25] With transrectal ultrasonography it is sometimes possible to find the thrombus and measure its size,[32] but with a rectal examination one can determine the decrease or absence of arterial pulse or fremitus in the iliac arteries.

Treatment and Prognosis

Treatment is focused on medication: anticoagulants (heparin 100 IU/kg sc SID, aspirin 5–10 mg/kg po SID, phenprocoumon 0.08–0.16 mg/kg IV initial bolus, gradually reduced), nonsteroidal anti-inflammatory drugs, and antimicrobial drugs (metronidazole 25 mg/kg PO BID and thiabendazole 4 g/45 kg PO once weekly for 3 weeks or ivermectin or moxidectin) to act against *S. vulgaris* larvae when they are expected to be the cause.[16]

Successful surgical removal of the thrombus has been performed with a thrombectomy catheter,[4] but this requires that the exact location of the thrombus be determined by ultrasonography or scintigraphy.[3,19,27] Prognosis for thrombosis of the aorta and the iliac arteries is guarded, especially when the underlying cause is not known.

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SACROILIAC REGION

Rob Van Wessum

Diseases of the sacroiliac region are recognized more and more as a cause for low-grade lameness or lack of performance.[2,3,20] Recent studies show that the sacroiliac joint may not always be involved, but the soft tissue structures adjacent to the joints in this region can be the cause of sacroiliac disease.[19,23,24]

The skeletal structures involved in sacroiliac diseases include the pelvic bones and spine, especially the sacroiliac joints where the ventral aspect of the ilium and ilial wing come into close contact with the sacral bone. At this junction there are two synovial joints, one at the ilial facet with a thin layer of fibrocartilage and one at the sacral facet with hyaline cartilage (Figure 6.19).[9,12,15]

At the ventral aspect of the sacroiliac joint, support is provided by several ligamentous structures, the capsule of the joint itself, and a package of small fibrous ligaments known as the ventral sacral ligaments.[12] On the top of the pelvic bones, ligamentous structures called the dorsal sacral ligaments, originated from the tuber sacrale. These include a long ligamentous branch, called the long part, that adheres to the fibrous structures of the pelvis (the sacrosciatic ligament), and a shorter portion, called the short part, that adheres to the sacral bone and the coccygeal vertebrae and ligaments (Figures 6.20–6.22). From the ventral aspect of the ilial wing, the interosseus sacroiliac ligament supports the sacroiliac joint (Figure 6.23).[9,12]

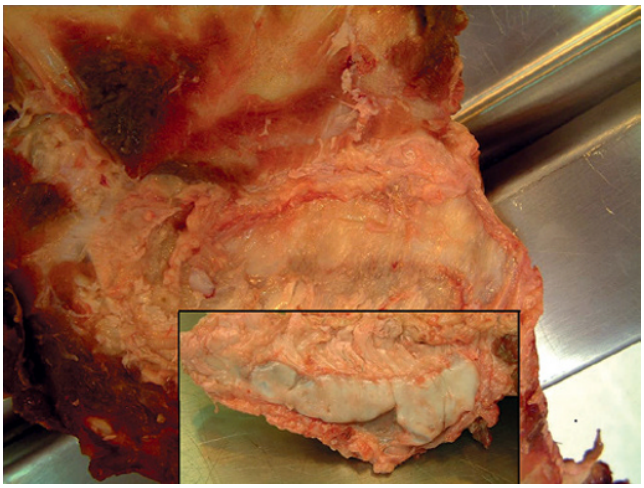


Figure 6.19. Anatomical specimen of the sacroiliac joint, ilial wing joint surface, and fibrocartilage. Insert: Sacral bone joint surface and hyaline cartilage.

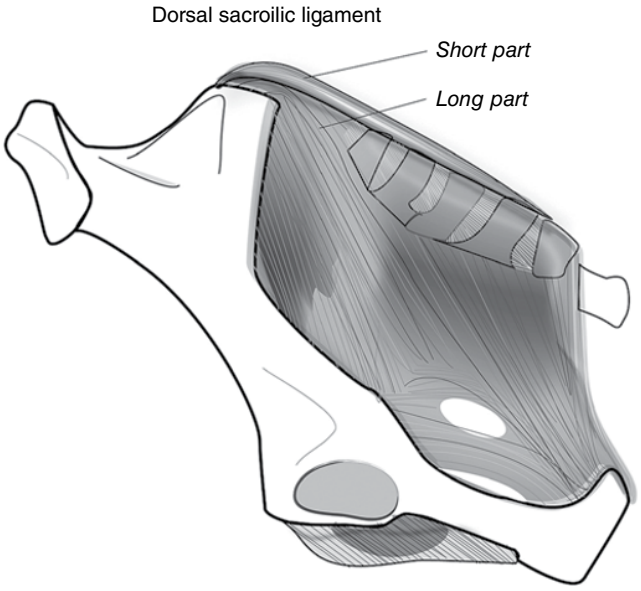


Figure 6.20. Schematic drawing of the dorsal sacral ligament, including the short part and long part. *Source:* Drawing by Maggie Hoffman. Reprinted with permission from van Wessum[23], p. 484.

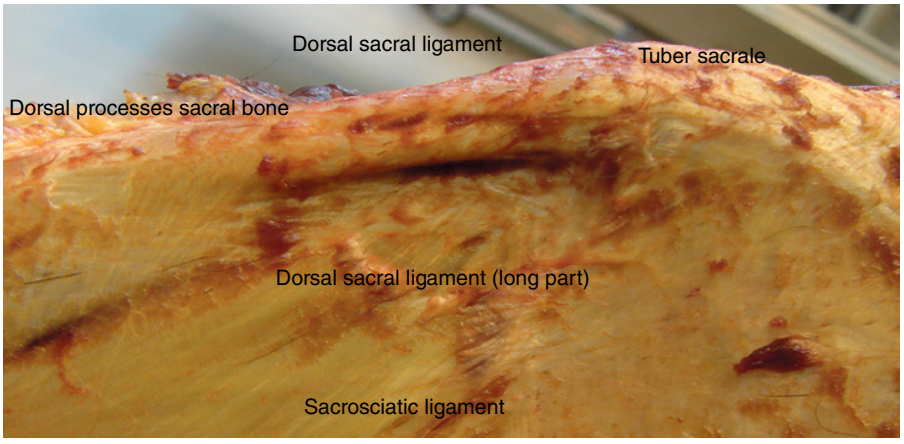


Figure 6.21. Anatomical specimen of the dorsal sacral ligament, longitudinal view.

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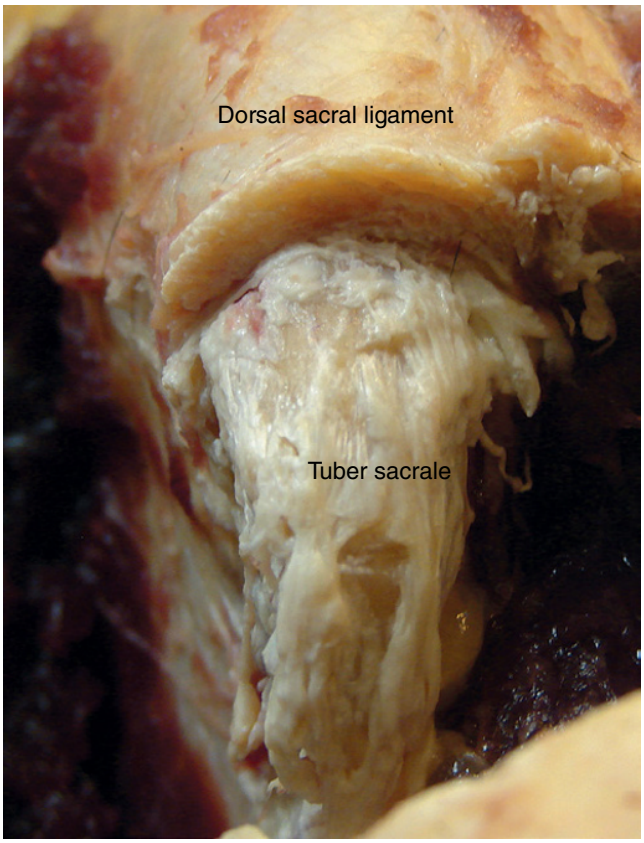


Figure 6.22. Anatomical specimen of the dorsal sacral ligament, cross-sectional view.

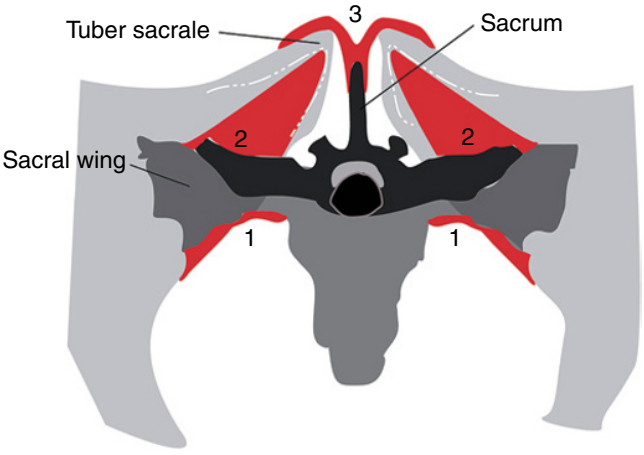


Figure 6.23. Schematic drawing of the sacroiliac joint region and the adjacent ligamentous structures, cranial aspect. 1 = ventral sacral ligaments, 2 = interosseus sacroiliac ligaments, 3 = dorsal sacral ligaments. *Source*: Drawing by Maggie Hofmann.

ETIOLOGY

The muscles of the caudal spine and pelvis are major contributors to the stability of the sacroiliac region. On the dorsal aspect, the middle gluteal and superficial gluteal muscles and their fasciae support the sacroiliac region. On the caudal aspect are the semimembranosus and semitendinosus muscles, and at the ventral part of the pelvis are the psoas muscles, all of which are important contributors to stability. Under normal conditions, the muscles in this region maintain the mobility of the sacroiliac joints within their physical limits. However, when the muscles are not powerful enough to compete with the external forces that result from massive trauma due to falling, slipping, tipping over, etc., primary ligament damage and primary or secondary joint trauma can be sustained. Weakness in these muscles can be associated with sacroiliac injury. This weakness can have many causes, including fatigue from improper training techniques, overuse, and repetitive stress injuries that can occur in young horses not yet capable of the desired level of performance.[18]

The pelvic region is not a rigid structure, as was previously thought. Haussler et al.[16] discovered that there is a small amount of deformation of the pelvic bone structures during the loading cycle in motion, and all of the involved structures (the bone, ligamentous structures, and muscles) form a semiflexible complex to assist propulsion and transport this propulsion from the hindlimbs onto the spinal column.[10] These findings make it easier to understand the major impact of the dysfunction of the pelvis structures regarding locomotion and performance.

Three different types of injury occur in the sacroiliac region. In the author's experience, the most common seem to be those that cause damage to the dorsal sacral ligaments (the structure with the greatest span between tuber sacrale, sacral bone, and the sacrosciatic ligament and the most potential motion between these structures). When trauma to the ligamentous structures of the sacroiliac region is more profound, the interosseus sacral ligaments and ventral sacral ligaments also can become damaged, with (sub)luxation of the sacroiliac joint(s) as a result. The last injury to the sacroiliac region occurs when damage to the sacroiliac joint is sustained with resultant osteoarthritis of the sacroiliac joint. In the author's experience, this only occurs in 5–10% of sacroiliac disease cases.[24]

CLINICAL SIGNS

Horses with sacroiliac pathology may come with a great diversity of owner/rider complaints, including reduced stride length in one or both hindlimbs, asymmetry of the hind end with one hip lowered, and occasionally obvious atrophy of the croup muscles. Owners report changes in both the rhythm and quality of the walk, with a more lateral walk seen especially during serpentines and circles, and a reduced stride length in one or both hindlimbs. There are no clear signs at the trot, perhaps just a little stiffness with slightly reduced propulsion and engagement. Downward transitions from canter to trot or from trot to walk can be of lower quality with a disturbance of the rhythm of the new gait in the first strides after the transition. Going downhill, even when the incline is small, can be difficult, and a refusal to jump in hunting, cross-country, or eventing can be a major complaint of the rider.[2,3]

The most remarkable signs of sacroiliac problems are usually shown when cantering. The pattern of the canter causes a very unilateral loading of the hindlimb and the

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sacroiliac region during the phase when the leading hindlimb (left hindlimb in left lead canter) carries the entire body load after the suspension phase. When sacroiliac lesions are present, the horse often alters its gait to relieve this unilateral loading by cantering more as a bunny hop in which there is less separation of footfalls of the hindlimbs. Horses with sacroiliac pathology also change leads or cross canter frequently. Due to this augmentation of signs in the canter, disciplines that are judged on the quality of the canter, for example, dressage and Western pleasure, or those that require a good-quality canter for optimum performance, as in hunting, jumping, and racing, comprise a greater proportion of clients who bring their horses with sacroiliac pathology to the author's practice.

Racing horses with poor performance often have mild signs of sacroiliac disease, especially the young, immature Thoroughbred in which mild to moderate strain to the dorsal sacral ligaments is diagnosed frequently.[24] Sacroiliac disease is very common in Standardbred racehorses, again with desmopathy of the dorsal sacral ligaments as the most prominent diagnosis; however, in the author's experience, osteoarthritis of the sacroiliac joint(s) is diagnosed in this breed more often than in any other breed. Incomplete or complete rupture of the dorsal sacral ligaments and incongruence of the position of the tuber sacrales (as a sign for clinical subluxation of the sacroiliac joint) are seen, mostly in racehorses.

Behavioral issues such as kicking, rearing, striking, and bucking, as well as resisting going forward, are quite often associated with sacroiliac pathology. Riders frequently report that these behaviors occur when the horse is asked to canter, and it can be difficult to determine whether these are training/behavior issues or a reaction to pain.[2,3]

Some horses do not show evidence of sacroiliac disease until ridden under saddle. However, severe sacroiliac injury such as acute subluxation or even dislocation of one of the sacroiliac joints or rupture of the sacral ligaments can cause severe hindlimb lameness, even non-weight-bearing lameness.

DIAGNOSIS

Clinical examination of horses with suspected sacroiliac disease is the same as any other lameness examination. Emphasis is placed on observation of the gaits, in the walk, especially with serpentines, and at the canter in softer footing as well as eventually under saddle. Lateral walk (movement of the ipsilateral pair of legs at the same moment, like a camelid) in serpentines is a reliable sign of reduced mobility of the spine. When sacroiliac pain is present, especially when the dorsal sacral ligaments are involved, mobility of the tail can be reduced. Very often the tail is fixed in one position and does not move when the horse walks in a serpentine. Loss of the quality of the canter in one lead is a very strong sign for sacroiliac pain. This can be presented as less propulsion, less engagement, cross canter, no-sound 3-beat canter, or even a bunny hop canter. Asymmetry of the tuber sacrales can be observed; however, this is also found in clinically normal horses.[2,3,9,12,15]

With acute sacroiliac disease there is clear pain in the hindquarters or back. The horse can exhibit severe muscle spasm, clear asymmetry of the tuber sacrales, and favoring one hindlimb. Even non-weight-bearing lameness is sometimes found in cases of acute sacroiliac disease, as with subluxation.

The lameness is less pronounced in more chronic cases. Muscle atrophy of the affected side may be noticed with unilateral sacroiliac disease. Shorter stride length, mostly in the cranial phase, of the affected limb is a very common finding, as is lack of propulsion, less hindlimb engagement, and lack of collection (coming under).[24] The horse may appear stiff and rigid with lack of lateroflexion in the lumbosacral region. Asymmetric positioning of the tail is often observed, with a deviation of the tail in the direction of the affected ligament; this releases tension to the short part of the dorsal sacral ligament when the ligament is involved (Figure 6.20).[24]

In stance as well as in motion, the affected side often has a slight hip drop. This is different than with hindlimb lameness, in which the hip drop often is just evident in motion and less so in stance. Flexion tests of the hindlimbs may be positive; note that secondary overload of hindlimb structures may become more obvious in sacroiliac disease. In most cases with primary sacroiliac disease, the flexion tests are questionable or negative. However, standing on the affected limb when the contralateral limb is lifted and flexed can pronounce lameness of the affected side after the flexion test, resulting in more pronounced lameness on the contralateral side. During the flexion tests the horse may show reluctance to stand on the affected limb or lean over to the affected side, so the stance limb is in midposition, which reduces rotational forces to the pelvic structures.

Examination of the mobility of the spine can give specific information about reduced range of motion in the sacroiliac and lumbosacral region. Reflexes that induce dorsal and ventral flexion can be suppressed due to pain in the sacroiliac region. Passive lateral flexion is quite often reduced to the side of the location of the sacroiliac pain.

Provocation tests are useful to determine the location of the pain within the sacroiliac region. Manipulation of the tuber ischium forward with one hand and moving the tuber coxae of the same side upward and downward introduces ventroflexion and rotation in the sacroiliac joints. Pressure on the tuber sacrale identifies pain at the insertion of the dorsal sacral ligament.

Recently, a retrospective study has provided some clinical guidance to help determine if sacroiliac dysfunction is likely to be present based on the observation of six clinical parameters during the standard lameness examination.[22] These parameters are tracking narrow behind, lateral walk on a serpentine, haunches in or out in a straight line and on circles, an asymmetric tail position, a bunny hop canter, and a reduced mobility of the lumbosacral region. Ultrasound examination provides more detailed information about the condition of the dorsal sacral ligaments.[4,17,19] Dorsal percutaneous ultrasonography using a linear probe with a higher frequency (10–15 MHz) is very useful for evaluating the dorsal sacral ligaments and their attachment to the tuber sacrale. Transverse views are used to identify the ligaments, check their size, and compare the left and right

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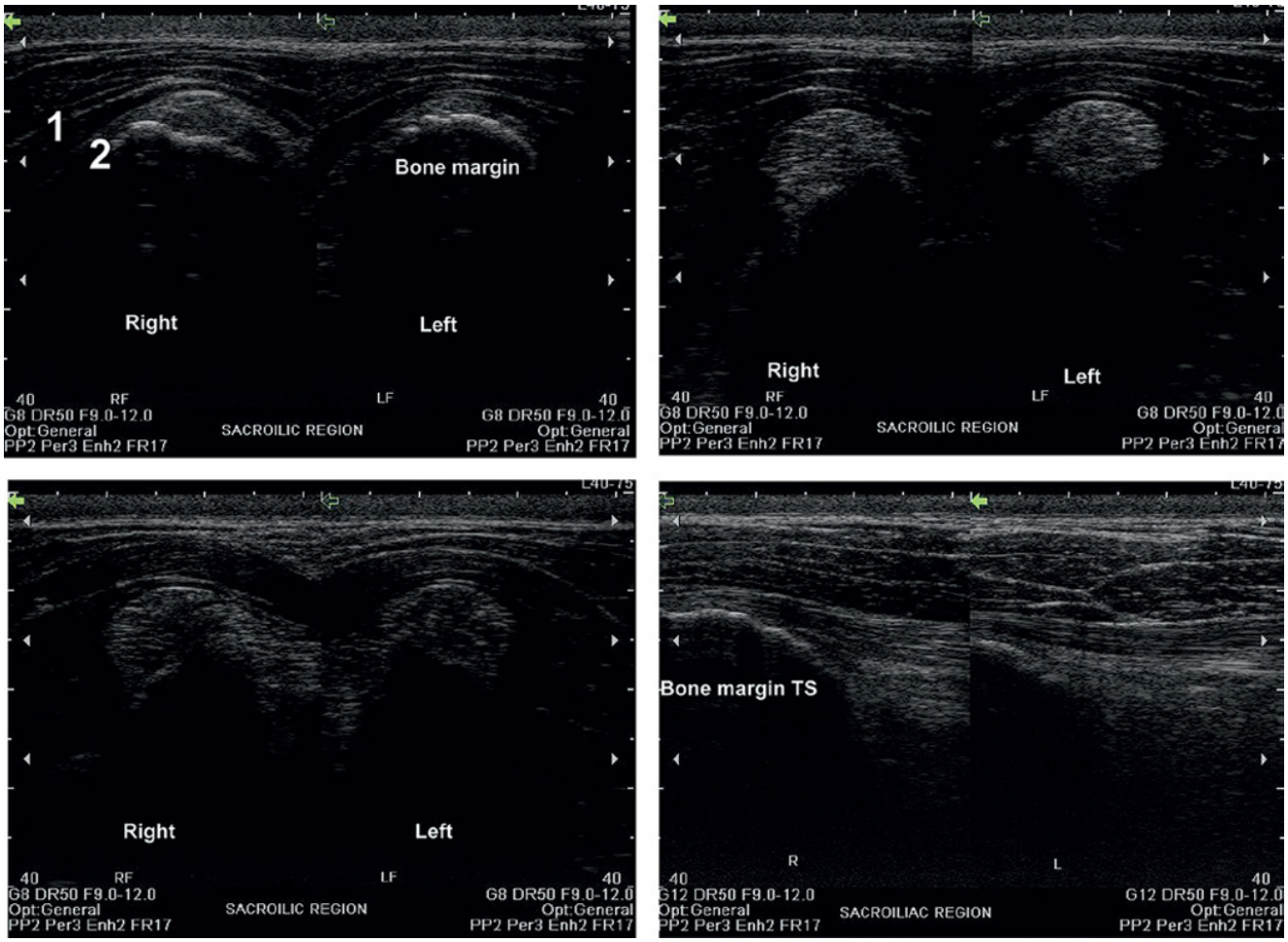


Figure 6.24. Ultrasonographic images. Right top view: transverse view. 1 = short portion of the dorsal sacral ligament (DSL), 2 = long portion. Note the enlargement of the right DSL and the loss of echogenicity in the medial aspect when compared with the left. The bone margin on the right, however, is more smooth and normal than at the left, where the tuber sacrale is more irregular.
Left top view: Transverse view, more caudal than in right top view; the bone margin is no longer visible. Only ligament is visible, where it crosses the space between the tuber sacrale and the dorsal processes of the sacral bone. The right DSL is larger when compared with the left.
Right bottom view: Transverse view, more caudal than in left top view; the DSL starts to attach to the dorsal process of the sacral bone in the midline. Note again the increase in size of the right ligament. However, there is some loss of echogenicity in the body of both ligaments.
Left bottom view: Longitudinal view. In the right DSL there is loss of echogenicity and fiber pattern alignment, indicating desmitis of the DSL.

sides of these structures (Figure 6.24). Evaluation of the structures and the adjacent muscle tissue with Doppler ultrasonography can give valuable information about vascular activity in this area. Longitudinal views are better for evaluation of the alignment of the tissue and the contour of the tuber sacrale bone.

Ultrasonographic transrectal examination of the sacroiliac joints has been described to evaluate the bony edges of the joints[6] for bone proliferation (Figure 6.25). However, in the author's opinion, there is little clinical relevancy for this technique, because necropsy findings show bone proliferation in this region in many horses without clinical symptoms. Radiographic techniques have been described for evaluation of the sacroiliac joint,[11] but they require general anesthesia and may not show a high correlation between radiological findings and clinical symptoms. Nuclear scintigraphy is the gold standard for showing clinically relevant involvement of the sacroiliac joint.[3,21,24]

Desmitis and enthesopathy (inflammation where ligaments attach to the bone) are the most commonly diagnosed entities at the author's practice. Desmitis is seen as an enlargement of the dorsal sacral ligaments, usually combined with reduced echogenicity of the ligament in comparison with the contralateral one. The acute phase, often caused by a partial rupture of the ligament, is observed as an area with very low echogenicity. The vascular activity around the ligaments is clearly increased when compared with the healthy contralateral ligament. Quite often small avulsion fragments can be observed within the ligament.

More chronic desmitis and enthesopathy can be recognized as a higher echogenicity of the ligament, nearly comparable with the contralateral one, but enlarged and

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with an irregular and less clearly defined bone margin. This is more a repetitive stress injury than an acute trauma.

In cases with massive damage to the ligamentous structures, a more complete displacement of the sacral bone is visible with the ilial wings displaced dorsally and the sacral bone displaced ventrally. In this case, one or both of the tuber sacrales are more pronounced (Figure 6.26). The latter condition is often called "hunter's bump."

In the case of significant displacement of the sacral bone, the incongruence in the articular surfaces of the sacroiliac joints leads to osteoarthritis. Osteoarthritis also can be a consequence of sacroiliac instability due to chronic desmitis of the dorsal sacral ligaments.

Scintigraphy facilitates evaluation of the sacroiliac joints as well as identification of enthesopathy of the tuber sacrale and the dorsal processes of the sacral bone.[3,21] Lateral (Figure 6.27), dorsoventral, and oblique views of the sacroiliac region pinpoint increased bone

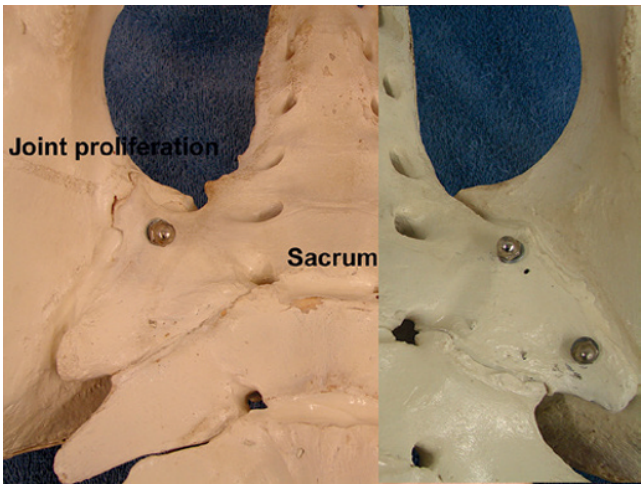


Figure 6.25. Anatomic specimen of the sacroiliac joint region. Note the visible bone proliferation at the ventral edge of the joints at the left view when compared with the right smooth joint margins.

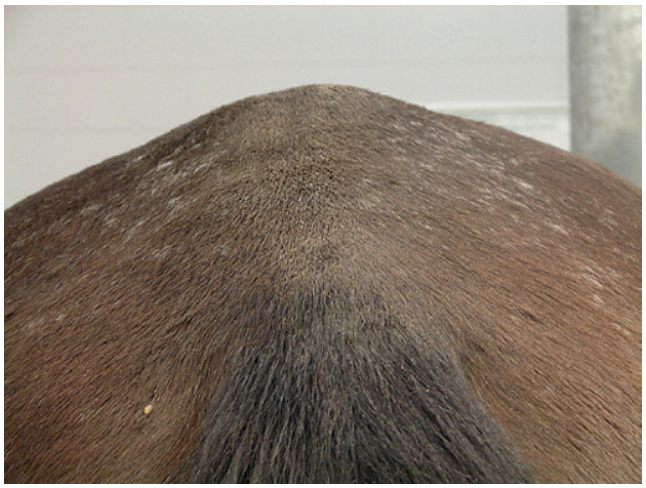


Figure 6.26. Caudal view of a horse with asymmetric tuber sacrales. Due to ligament damage, the right tuber sacrale is much higher than the left.

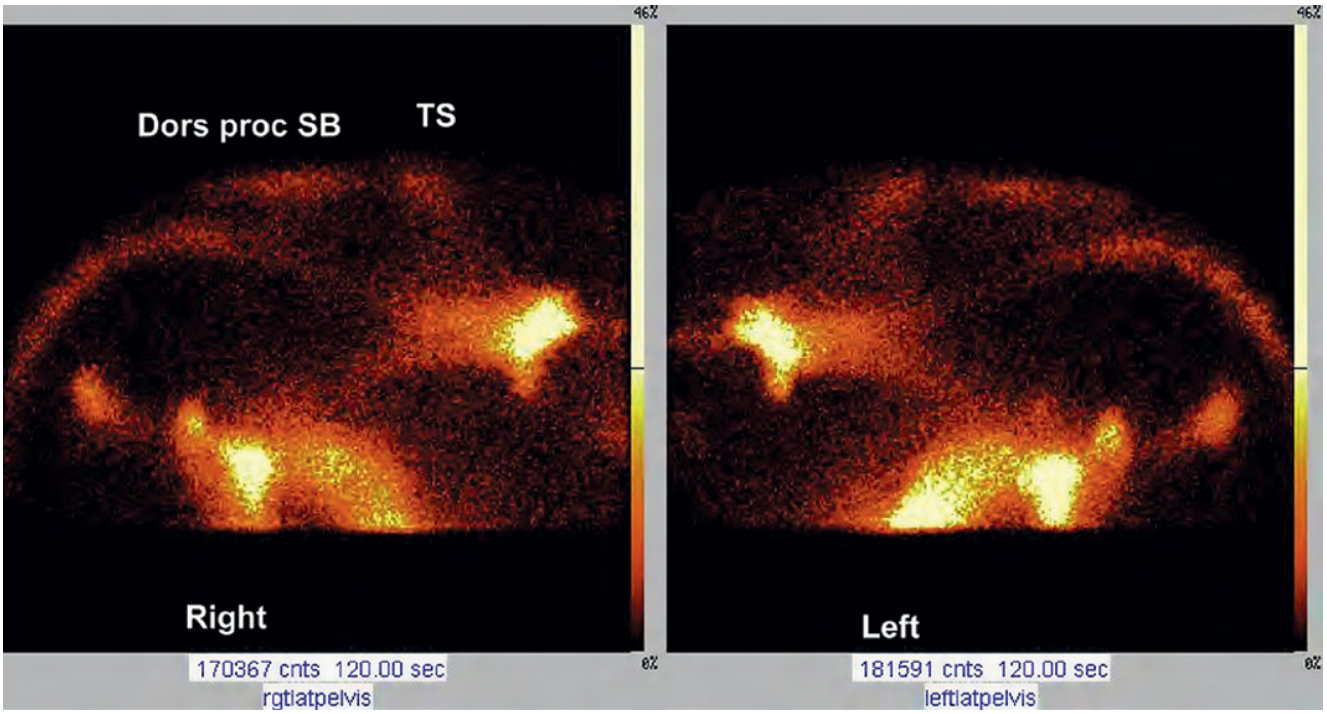
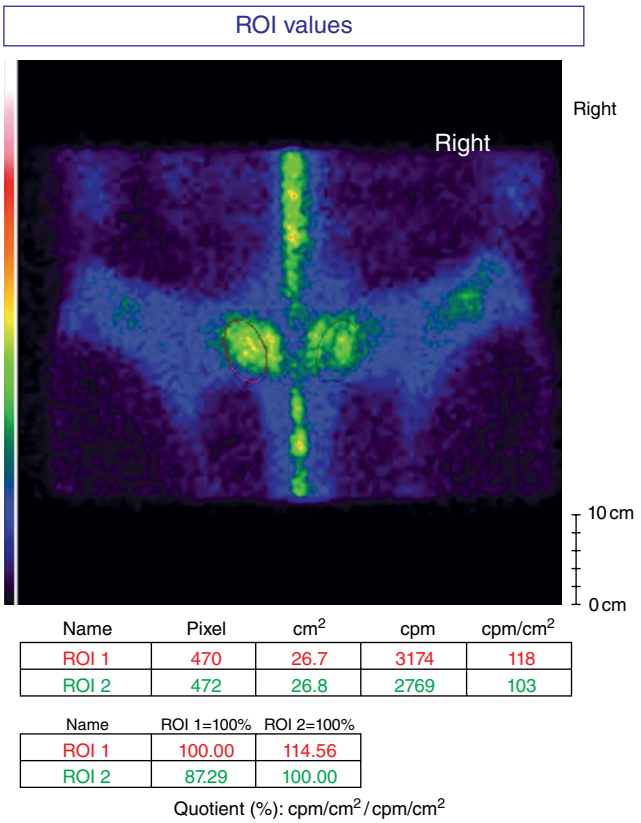


Figure 6.27. Scintigraphic images of the pelvic region in lateral view from left and right. Note the slightly higher uptake in the left tuber sacrale when compared with the right. There is also slightly increased uptake visible in the dorsal processes of the sacral bone. This can be a sign of enthesopathy of this structure or an indication of lower musculature size in this region.

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Name	Pixel	cm²	cpm	cpm/cm²
ROI 1	470	26.7	3174	118
ROI 2	472	26.8	2769	103

Name	ROI 1=100%	ROI 2=100%
ROI 1	100.00	114.56
ROI 2	87.29	100.00

Quotient (%): cpm/cm² / cpm/cm²

Figure 6.28. Scintigraphic image of the pelvic region with region of interest counts. The count on the left sacroiliac joint region is significantly higher (14% higher) than at the right joint region.

metabolism of the tuber sacrale in the case of enthesopathy and a more condensed higher bone metabolism in the ilial wing in the case of sacroiliac joint pathology (Figure 6.28). Lateral views taken in the first stage of uptake of radiopharmaceutical (soft tissue phase) can show increased uptake in the dorsal sacral ligaments as well as in the epaxial muscles.

TREATMENT

In all cases of sacroiliac disease, a complete and intensive rehabilitation program is of paramount importance for the complete recovery of the horse.[2,3,13,23,24] When enthesopathy and desmitis are the most obvious diagnoses, an initial 2-month period of rest to immobilize the region is the first step of treatment. Uncontrolled motion in the pelvic region must be prevented, and complete stall rest with no turnout for the first 2 months of treatment is the only effective way to achieve immobilization of the pelvic region. During this period of stall rest, the horse is hand-walked on a straight, flat surface 2–3 times a day for 10–15 minutes. During this time oral medication with NSAIDs is needed to assist in the complete reduction of inflammation in the ligaments and bones. In the author's practice, phenylbutazone (1 g q 12 hours for a 500- to 600-kg horse) is the drug of choice.

After the initial 2 months of stall rest, 1 month of walking under saddle can begin. During this time, the horse is walked in serpentines and circles to increase lateroflexion and rotation in the lumbosacral and sacroiliac region. The rider should make the horse bend on the circles and gradually reduce the circle to a smaller diameter, thus increasing lateroflexion.

In the fourth month of rehabilitation, the same exercises are done at the walk and also at the trot to increase speed and thus loading of the lumbosacral and sacroiliac region.

In the last 2 months of rehabilitation, the horse is ridden for longer periods, until achieving 1–1.5 hours of work daily. The canter is introduced with emphasis on brief episodes with many downward transitions to trot, creating more dorsal and ventral flexion in the lumbosacral and sacroiliac region. Short intervals of canter are extremely important to prevent fatigue of the muscles that support flexion and extension of the joints and their supporting ligaments to avoid mobility beyond their physical limits, which may be diminished by the primary injury.

This rehabilitation program gradually introduces more mobility and develops better muscle support of the sacroiliac region. The exercises develop more muscle power in the epaxial muscles as well as in the muscles of the croup, which are of eminent importance in limiting the range of motion of the sacroiliac joints and their supportive ligamentous tissue.

When the sacroiliac joints are involved (in the author's practice this is only 10–15% of all cases and is almost always unilateral), injection of the joint with a corticosteroid may be needed. The author prefers triamcinolone to prednisolone, and it causes less tissue irritation (AP Pease, personal communication).

A single injection of the affected joint with triamcinolone (20–30 mg) coupled with concurrent phenylbutazone and the rehabilitation program described above gives results that are quite similar to those described for treatment of enthesopathy and desmitis. In the author's experience, when scintigraphy findings lead to the diagnosis of sacroiliac joint disease, results of oral medication and a prolonged rehabilitation program without an injection are disappointing.

Injection directly into the sacroiliac joint is very difficult because the ilial wing makes access into the joint nearly impossible. However, depositing a corticosteroid very close to the joint can be effective in reducing inflammation within the joint. There are several different techniques for depositing a corticosteroid close the joint.[1,4,5,7,8] At Michigan State University, a novel technique for injection of the sacroiliac joint (region) has been tested and is preferable to the author.[25] This approach is with a straight needle (15-gauge, 20–25 cm) with an insertion site 3–4 cm cranial and 1–2 cm paramedian to the contralateral tuber sacrale, through the interspinous ligament of L5 and L6, and aimed toward the ipsilateral tuber ischium (Figure 6.29). The medication is deposited when the tip of the needle makes contact with the ventral aspect of the ilial wing as ventral as possible. Landmarks for the location of the insertion site of the needle are the dorsal process of L5 and the tuber coxae. The insertion site of the needle is located on the contralateral side of the joint to be injected. The length between the cranial palpable tips of the dorsal processes of L5 and L6 is measured. Half of this length is the distance that the insertion site is lateral of the cranial tip of L5 on the contralateral side. This technique facilitates insertion of the needle at the correct location in horses of different sizes.[25]

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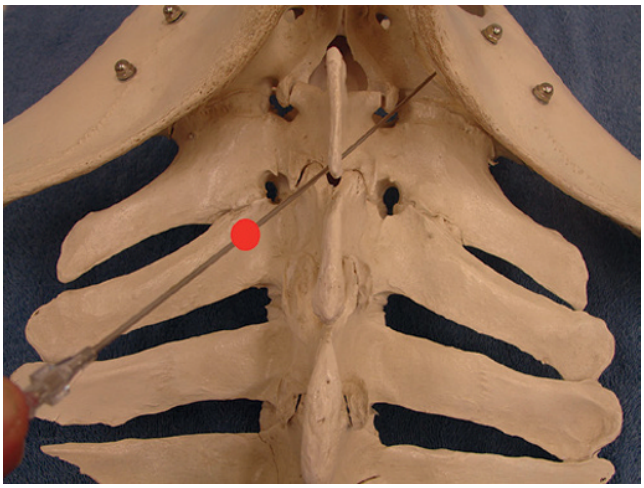


Figure 6.29. Injection of the sacroiliac joint: needle insertion at the left side 2 cm out of the median, through the intervertebral ligament between the dorsal processes of L5 and L6 into the right sacroiliac joint. The red dot indicates the location of needle insertion on the skin.

In cases with severe damage to the dorsal sacral ligaments (disruption of the fiber pattern) as with partial or complete rupture of these ligaments, injection of platelet-rich plasma (PRP) into the damaged tissue has shown impressive results at the author's hospital. In comparison with cases that were treated conservatively, the architectural structure of the ligament improved much faster and was more completely restored after treatment with PRP. However, no change of overall rehabilitation time (4–6 months) has been observed.[26]

PROGNOSIS

In nearly all of the past reports, prognosis for sacroiliac diseases has been rather guarded.[3,14,20] In the author's experience with more than 200 cases in the last 4 years involving desmopathy of the dorsal sacral ligaments and unilateral OA of the sacroiliac joint, more than 90% of the horses returned to work at their original level or higher. All of these cases were on a 6-month rehabilitation program with rechecks at 2-month intervals. The horses were evaluated clinically and ultrasonographically at every recheck, and adjustments to the rehabilitation program were made based on the outcome of this evaluation.

For cases with partial or complete rupture and subluxation of the sacroiliac joint, prognosis seems to be favorable; however, at Michigan State University the author has seen fewer than 10 cases with this type of injury over the last 4 years. With enough time for initial healing of the ligamentous structures (2-plus months of stall rest with some hand walking) and 4–6 months for rehabilitation exercises, these horses were able to return to their original level of performance.

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THORACOLUMBAR SPINE/BACK

Rob Van Wessum

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OVERRIDING/IMPINGEMENT OF DORSAL SPINOUS PROCESSES

Etiology

The impingement of the summits of the dorsal vertebral processes, also known as "kissing spines," is a relatively common diagnosis in horses with back pain.[7,8,12] The most common location for these lesions is in the thoracic spine (between T5 and T18), but impingement of the lumbar dorsal processes has also been reported.[8,17] Pathology may be caused by the repetitive, traumatic contact between the dorsal processes. Sclerosis of the cortical margins of the dorsal processes, narrowing or loss of the interspinous space, and osteolysis of the spinous processes are affirmative signs of bony pathology. When the supraspinous ligament or the interspinous ligaments are involved, enthesiophytes and avulsion fragments may be present at the summit (supraspinous ligament) or at the caudal or ventral aspect of the dorsal spinous process (interspinous ligament).[11]

When adjacent structures are damaged, they can lead to impingement of the spinous processes. A primary injury to the ventral and ventrolateral supportive structures of the annulus fibrosis of the intervertebral disc can cause asymmetrical narrowing or collapse of the intervertebral joint, which may tilt the dorsal vertebral processes toward each other.[30] In that case, the impingement may be a secondary finding at the initial radiological examination, underestimating the extent of the trauma. Spondylosis at the ventral aspect of the spine and narrowing or the loss of definition of the intervertebral disc space can be an additional radiological finding, pointing to the involvement of more spinal structures than just the spinous processes.

Clinical Signs

Impingement most often reduces the ventrodorsal mobility of the spine, but when pain is present, the lateral mobility may be limited due to reflectory muscle spasm, thus affecting performance.[12] Horses with impingement of spinous processes have a variety of symptoms, often related to the discipline in which they perform. Although show jumpers and hunters seem to be more affected, many horses that perform without clinical signs of back pain show radiological evidence of impingement. When impingement is present in the cranial part of the thoracic spine (T5 to T12 or T13), the horse may have a painful or violent response when putting the saddle on or when the rider mounts. Differentiating an injury from saddle fit issues can be a challenge.

Diagnosis

Palpation along the back may reveal irregularities in the size of the summits of the spinous processes of the thoracic or lumbar vertebrae. Deep digital palpation is necessary in most cases to reveal these changes. Examination should begin at the withers and work toward the sacrum. It is important to differentiate the resentment shown by some horses to this examination from distress resulting from other back conditions.[12] Clinical examination can show pain with localized digital pressure of the specific dorsal process or the supraspinous ligament.[7] Limitation of dorsoventral mobility as well as possible lateral reduction of motion is limited to the specific part of the spine.

Radiological examination can clearly identify bony changes as sclerosis and osteolysis.[26,37] Ultrasonographic examination is very useful to evaluate the contact and remodeling between two adjacent spinous processes, transverse thickening of the processes, and abnormal alignment. Concomitant lesions in the supraspinous ligament and enthesopathy on the summits of the spinous processes also can be imaged with ultrasonography.

Scintigraphy may help to identify evidence of active bone metabolism and remodeling of the spinous processes (Figure 6.30), as well as adjacent structures of the spine that can be involved (intervertebral disc, facet joint, vertebral body).[33] Localized increased activity may be observed or absent in completely sound and clinically asymptomatic horses, as well as in horses with clear symptoms of back pain caused by impingement of spinous processes.[15]

Infiltration with a local anesthetic of a site that is suspect for pain caused by impingement can give more information about the likelihood of the observed impingement causing the symptoms.[10,13] The most comprehensive and complete examination of the thoracolumbar spinous processes should incorporate radiography, scintigraphy, and diagnostic analgesia.[39]

Treatment

Conservative therapies include rest, nonsteroidal anti-inflammatories, local injections of anti-inflammatory agents, acupuncture, and physiotherapy. Horses that are rested often improve during the rest period but become sore again shortly after reinstating exercise. Systemic therapy with NSAIDs helps horses with very minor lesions, but does not cure more severe lesions. Injections of corticosteroids between affected spinous processes, combined with nonsteroidal systemic therapy, can be quite helpful in reducing or removing pain.

Several surgical techniques have been described to remove an affected part of a dorsal process.[27] Newer techniques are less invasive, and endoscopic resection of the spinous process and the interspinous ligament has been performed.[14]

Prognosis

The prognosis for impingement of spinous processes is guarded when the signs are primarily attributable to this disease. In a study by Erichsen,[15] scintigraphic and

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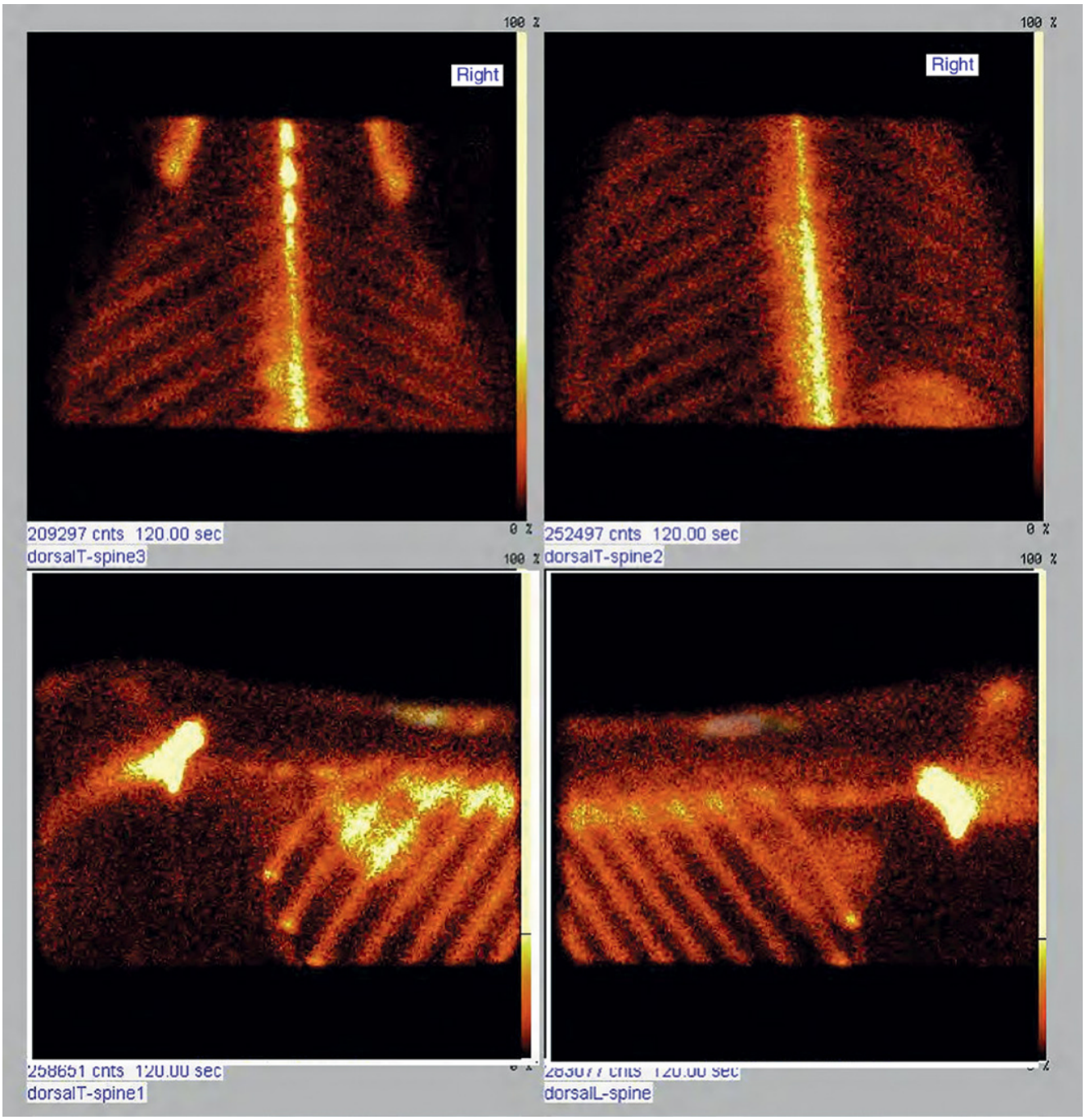


Figure 6.30. Scintigraphic image showing IRU in the dorsal processes of the caudal thoracic spine, visible on lateral views and a dorsal view. This horse was a 13-year-old show jumper with an international Grand Prix career that had no major clinical problems.

radiological signs of impingement were found in completely sound horses. The most important factor in determining the prognosis for impingement of the spinous processes is the complete diagnosis. When ligamentous structures are involved (e.g., supraspinous ligament, intraspinous ligament, ventral longitudinal ligament), the prognosis for a quick recovery to the original workload is less favorable than when just osseous changes are seen on radiographs. When there are adjacent pathologic conditions in the intervertebral disc or the facet joints, prognosis is also less favorable. For more severe cases, prognosis for a return to the original work level after months of rehabilitation seems to be better when resection is performed than when surgery is not performed.[14,27]

SUPRASPINOUS LIGAMENT INJURIES

Injuries of the supraspinous ligament are most common between T15 and L3.[7] They can be associated with localized thickening and pain.

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Clinical Signs

Signs of desmopathy of the supraspinous ligament are similar to those of impingement of dorsal spinous processes. When only ligament pathology is present, signs can be mild and very difficult to detect. Occasionally owners may recognize the increased size of the ligament (focal thickening), and this can be the primary complaint. In cases with more swelling in and around the ligament, the appearance of the shape of the spine can resemble kyphosis. In that case, radiography shows a normal configuration of the spine, but ultrasonography can show the soft tissue involvement and edema.

Diagnosis

Ultrasonography is the tool of choice to image these injuries.[22] Diagnosis of the pathology of the supraspinous ligament can be made with ultrasonographic examination. Radiography and scintigraphy can give additional information about the involvement of the spinous processes. Radiographic examination of desmopathy of the supraspinous ligament may show irregular bone margins of the summit of the dorsal spinal processes, avulsion fragments, and sclerosis. Scintigraphy may show increased uptake in the summits of the spinous processes (Figure 6.31). These findings are similar to those of impingement of the dorsal spinous processes, and quite often both pathologic conditions are found in the same horse.

A study of Henson et al. found ultrasonographic changes in the supraspinous ligament in clinically normal horses as well as in horses with signs of back pain.[22]They concluded that when ultrasonography alone does not confirm the diagnosis of desmitis of the supraspinous ligament, then local (infiltration) anesthesia should be performed as well.

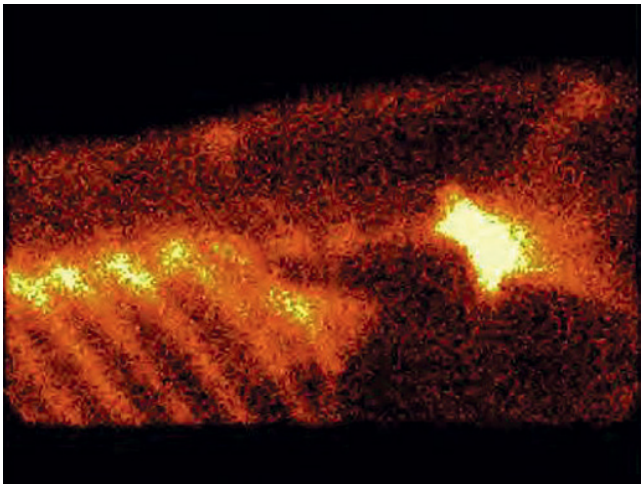


Figure 6.31. Scintigraphic image showing mild focal IRU at the summit of T18, indicative of desmitis of the supraspinous ligament. Radiography did not show any change, and ultrasonography showed mild desmitis with roughened bone margin of the summit of the spinous process of T18.

Treatment

As with any ligamentous structure, treatment is focused on rest, medication, and rehabilitation. Controlled stretching of the ligament with a lower position of the head (i.e., in pasture, with hay on the ground, when ridden with a lower head and a longer neck) and a gradual increase in the workload is recommended.

Prognosis

When the ligament is the only affected structure and adequate time for a complete recovery before resuming the original work is allowed (4–6 months), the prognosis is usually favorable. When more structures are involved, as with impingement of dorsal spinous processes, facet joints, or intervertebral discs, the prognosis tends to be less favorable.

FRACTURES OF THE SPINOUS PROCESSES

Etiology

Fractures of the dorsal spinous processes occur mostly in the cranial thoracic spine (the withers region) when horses flip over, fall backwards, or run into objects with their withers (such as when the door is lower than the horse).[3,24] Sporadic fractures of the dorsal spinous processes in the lumbar spine can be caused by excessive trauma such as falling and turning at high speed, for instance, in cross-country riding, jumping, barrel racing, hunting, or traffic accidents. This places rotational force on the spine, which can cause fractures of the lumbar lateral spinous processes.[19]

Clinical Signs

Fractures of the withers have very clear symptoms, with swelling in this region and deformation of the normal contour of the withers. A significant dent can be observed in the normal curvature of the withers region, and the distal fragment will dislocate in most cases and end up lateral to the remaining spinous process, giving the withers a wider appearance. Fractures of the spinous processes of the more caudal thoracic spine and the lumbar spine are less pronounced in appearance. When the tips of the spinous processes are viewed from above, a clear change in the alignment may be visible, with the tip of the fractured spinous process out of line to the lateral side, possibly with some dislocation to the ventral side. A fracture of the lateral spinous process in the lumbar spine may be difficult to see when observing the horse due to the fact that they may not be dislocated.

In the acute stage, pain is present with generalized back stiffness, localized swelling, and muscle spasms in the epaxial muscles. In older cases, the change in the alignment of the withers or the caudal spine is the clear remains of the fracture, with few other clinical signs. When the mobility of the spine is evaluated, there may be slight alterations at the location of an older fracture, due to remodeling of the fragments and attached muscles.[20,28]

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Diagnosis

Radiographic examination is the first diagnostic technique to image the spinous processes because the fragments, which are often dislocated, show clearly on the radiographs.[37] The summits of the thoracic spinous processes, especially in the withers region (T3 to T12), can vary in shape, show a variety in the pattern of accessory centers of ossification, and easily be misinterpreted as a fracture. Dislocation of the fragment is a very common finding in fractures of the spinous processes in the withers region, which helps to distinguish them from irregularities and delayed ossification. In the more caudal thoracic spine and lumbar spine, radiographic examination may be less useful because the muscle mass in these regions can make radiographic imaging incapable of identifying a nondisplaced fracture.

Ultrasonographic examination can give good additional information, especially to identify fractures of the lateral spinous processes in the lumbar spine.[9]

Scintigraphy can show increased radiopharmaceutical uptake after a fracture, but is not specific enough to identify the fractures. For lateral spinous process fractures, scintigraphy can be useful to identify the location of a possible fracture (Figure 6.32), but adjacent ultrasonography is needed to verify this.

Treatment

Treatment for fractured spinous processes is often not required, other than medication to deal with the associated pain of an acute fracture. Surgery is usually not required. Rest and controlled exercise for 1 to several months to keep the horse in good muscular condition usually are sufficient. The saddle should not be used when the withers region is involved.

Prognosis

Prognosis for most spinous process fractures is favorable. Sometimes after a fracture in the withers region, it is difficult to make the saddle fit properly due to the changed shape of the withers. When the horse is judged on shape and conformation in its discipline (i.e., hand showing, breeding championships), the abnormal shape of the withers can influence the judging. Providing the client with a clear veterinary statement that the abnormality is caused by a traumatic event and not genetic and that the horse is capable of competing facilitates a successful show career.

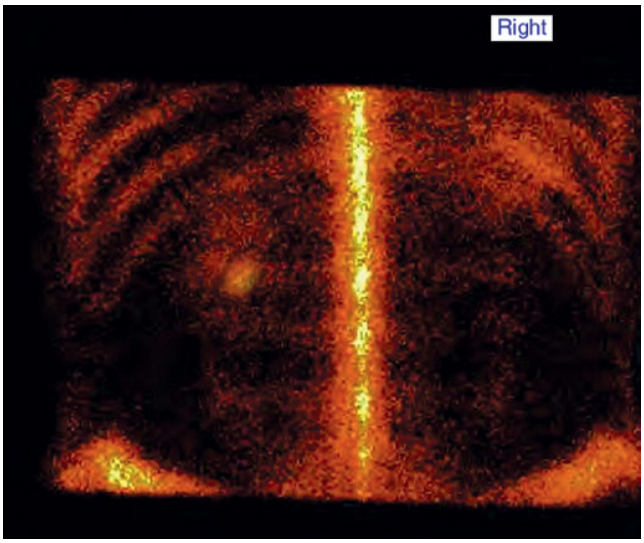


Figure 6.32. Scintigraphic image of the lumbar spine, dorsal view. Note the IRU in the left transverse spinous process of L2. This is indicative of a fracture of the transverse process.

VERTEBRAL FRACTURES

Etiology

Most vertebral fractures are traumatic in origin; however, pathological fractures can occur with processes affecting the vertebrae such as neoplasia, metastasis, and osteomyelitis.[34]

Stress fractures of the vertebra most likely occur more frequently than previously believed. In one study, 18 of 36 specimens had incomplete fractures and focal periosteal proliferation of the lamina stemming from vertebral stress fractures.[19] The fractures occurred most often near the junction of the cranial articular process and the spinous process.

Clinical Signs

Acute vertebral fractures are accompanied by severe pain. The horse is reluctant to move the affected part of the spine and shows increased muscle tension in the region of the fracture. Neurological symptoms are present when soft tissue swelling, hemorrhage, or fragments compress the spinal cord or root nerves. Unilateral root nerve compression can cause the horse to bend to the affected side when root nerve compression leads to irritability of the nerve and to the opposite side when the compression causes complete loss of neurotransmission.

Most stress fractures result in a lack of performance without clear-cut symptoms. Stiffness, lack of propulsion from behind, and mild back pain are common signs associated with laminar stress fractures in the young Thoroughbred racehorse. These are most often seen at the thoracolumbar junction and the lumbar vertebrae.[19,20]

Diagnosis

Radiography can image larger fragments; however, with the initial radiographic examination, these fractures can be easily overlooked when there is little dislocation present. In addition, fractures with a longitudinal fracture line are nearly invisible on lateral views. Scintigraphy is a sensitive technique for vertebral fractures and is nearly the only way to determine active stress fractures.[33]

Treatment

Treatment is conservative in most cases of vertebral fracture without neurological symptoms. Stall rest with very controlled exercise and medication with NSAIDs and often muscle relaxants to reduce the pain and muscle spasms are sufficient. When neurological symptoms are present, treatment focuses more on reducing swelling

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around the spinal structures; medication with systemic corticosteroids and DMSO may be indicated.

Prognosis

The prognosis for nondisplaced vertebral fractures without neurological symptoms is favorable, as is the prognosis for most stress fractures. When neurological symptoms are present, the amount of time needed to reduce these symptoms with medication is a good indicator for future expectations. The longer it takes for the recovery of neurological functions, in general, the poorer the prognosis.

FACET JOINT ARTHRITIS AND VERTEBRAL FACET JOINT SYNDROME

Etiology

When facet joint osteoarthritis is present in the thoracolumbar spine, similar to this process in humans,[31,32] a complex of processes in and around the vertebral facet joints can lead to a painful condition known as facet joint syndrome. In the cervical spine, however, osteoarthritis of the facet joints seems to be less frequently related to facet joint syndrome than in the thoracolumbar spine, and the accompanying muscle spasms in the segmental muscles are absent. This gives the impression of an isolated joint disease with a reduced range of motion and pain in just the affected joint. However, compression of nerve roots due to proliferation of the bone around the facet joints is a more common observation in the cervical spine than in the thoracolumbar spine.

The initiating incident in facet joint syndrome is damage to the facet joint, primarily to the joint capsule and the adjacent connective tissue, or else to the cartilage and/or subchondral bone. In the horse, this primary incident quite often is trauma, caused when the horse slips, falls, flips over, gets cast, or injures itself in any other way. Due to the rich innervation of the facet joint tissues, the inflammation reaction caused by the primary trauma starts a cascade of reactions that is more obvious than in the joints of the limbs.

One of the first reactions of the horse is stabilization of the spine by muscular contraction and spasm of the epaxial and subaxial muscles. This can occur in just the segment that is related to the injured vertebral facet joint, but a larger portion of the vertebral column may be involved. This muscle contraction can be short, last hours to days, or last weeks or even months. It is very likely that the prolonged contraction or spasm is very painful and partially immobilizes the spine, as is the situation in humans.[31,32]

As a consequence of this immobilization, the facet joint is more or less fixed in one position, which makes regaining later mobility a painful experience.[18] Due to the muscle spasm, no normal sequence of contraction–relaxation occurs in the muscle, and the normal supportive function is less effective.[21] Consequently, when excessive force is applied to the spine again, repetitive injury to that facet joint can be the result.

Clinical Signs

Due to the contribution of the spine to the normal gait pattern in the horse, as described earlier in this chapter, horses that suffer from vertebral facet joint syndrome are often presented for lameness. In acute cases, the pain in and around the facet joints can be so severe that the horse does resist all movement and stand with the hindfeet parked out and the back lowered. This position closes the facet joints in the thoracic and lumbar spine, causing less tension in the joint capsules. The muscles of the back and hind end are contracted. This attitude can be difficult to differentiate from tying-up syndrome; however, with vertebral facet joint syndrome, the serum levels of creatine kinase (CK) and aspartate aminotransferase (AST) are within normal limits or only slightly elevated.

With severe pain in the thoracic or lumbar spine, signs can be similar to those of colic, with restlessness, pawing, looking back, or standing with the hindlegs parked far out, giving the impression of wanting to urinate. In those cases, the heart rate can be as high as 60–80 beats per minute, but an examination for colic reveals that the horse is within normal limits.

In more chronic cases, the locomotion of the horse can be altered and a stiff back noticed. At the trot, the propulsion of the hindlimbs can be reduced unilaterally or even bilaterally.[21,25] Due to the attachment of the front limbs with ligaments, tendons, and muscles to the thoracic vertebrae, motion of the front limbs also affects the position of the thoracic facet joints. At the trot, due to the diagonal gait, the thorax moves between the scapulae, with a maximum extent of motion in the dorsal processes of the withers of 1–3 cm to left and right. Pain in the facet joints of the thorax thus can lead to less mobility in the thoracic spine, and an altered gait pattern in the trot is best described as stiff or shortened.[21]

During the canter the thorax moves less, except in downward transitions (changing from canter to trot or trot to walk) when there is more loading of the facet joints, as well as in landing after a jump or going downhill. Therefore, when thoracic facet joints are involved, signs mentioned by the owner or rider can include stiffness, reluctance to go downhill or jump, and refusal or difficulty in extended trotting. Especially in jumping, a trainer or rider may note a preference of the horse to go left or right after a jump, and this is quite often thought to be related to an injury to the distal limb but in fact is caused by facet joint problems.

Because lateral bending is a prominent mobility feature of the thoracic vertebrae, lateral bending of the horse may be reduced when thoracic vertebrae are involved with facet joint syndrome. This can be observed during circles and serpentines and in lateral exercises such as shoulder in, travers, and half pass of dressage, as well as in turns in barrel racing, polo, and eventing, or in between fences when jumping. Tightening the girth or putting the saddle on also can reveal an adverse reaction of the horse, because these actions can load the thoracic facet joints.

When lumbar facet joints are involved in facet joint syndrome, the most affected gait is the canter, because it is then that the dorsoventral flexion of the lumbar spine is most prominent. The lateral exercises—shoulder in,

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travers, and half pass—can show alterations when lumbar facet joints are involved because the rotation in the lumbosacral region makes an important contribution to these movements. In racing, dorsoventral flexion of the lumbar spine is a prominent contribution to the propulsion phase of the hindlimbs, so loss of performance may be caused by lumbar facet joint syndrome.

Diagnosis

The thoracic vertebrae can be viewed with radiography with the lungs facilitating radiographic imaging in the cranial part of the thorax. In most horses, the vertebral bodies of T1 through T15–T16 can be imaged adequately to evaluate the intervertebral disc spaces and vertebral body as well as the ventral aspect of the vertebrae.[32] In the more cranial part of the thorax, it is difficult to get a clear image of the intervertebral disc space and the facet joints because of the superimposing structures of the scapulae. Depending on the conformation of the horse, the width of its thorax, musculature, and body condition, oblique lateral views can isolate the unilateral facet joints from T5–T7 to T18 (Figure 6.33). When pathology of the facet joints is present, there are signs of sclerosis of the bone just around the facet joint, narrowing of the joint space, irregular shape of the joint space, and spur formation at the edges of the joint space or complete ankylosis visible on the radiographs. Lumbar vertebrae are the most difficult to visualize with radiography. Especially well-muscled horses of the bigger breeds often give disappointing images, and it is difficult to differentiate their facet joints from the massive vertebral bodies of the lumbar spine. Oblique lateral views can sometimes facilitate imaging of the unilateral facet joints of the lumbar spine. Even under general anesthesia, the facet joints of the sacrum and the adjacent pelvic bones cannot be viewed accurately with radiography. However, the coccygeal vertebrae are easy to see with radiography.

Ultrasonography is a very useful tool for examining the facet joints of the vertebral column.[9] It can give information about ligamentous structures and muscle conditions (Figure 6.34), high muscle tension can be shown as increased muscle echogenicity, and fasciculations can be visualized in real-time ultrasonography. The supraspinous ligament, interspinous ligaments, and dorsal sacral ligaments can be examined with this as well. Paramedian longitudinal views of the facet joints can be used to determine the edges of these joints, which should normally be smooth and bilaterally symmetrical. Possible effusion of these joints, bony proliferations at the joint margins, and fractures and avulsion fragments and ankylosis of the facet joints (Figures 6.34–6.36) can be seen with ultrasound.

Doppler ultrasonography can provide valuable information about vascular activity around the facet joints (Figure 6.37). In cases of synovitis or osteoarthritis of a facet joint, increased vascular activity can be visualized around the facet joint, especially when the adjacent facet joints and the contralateral facet joint are compared.

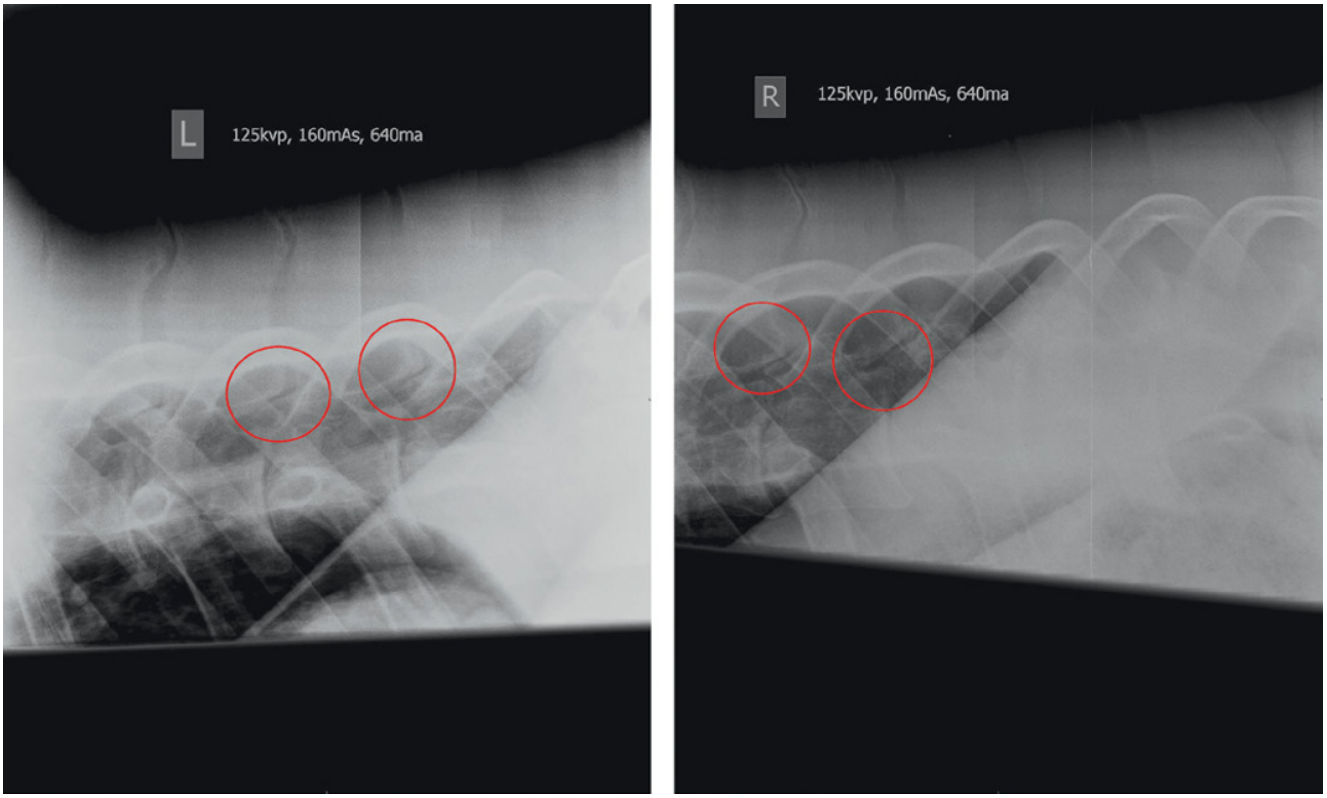


Figure 6.33. Radiographic image of the thoracic spine, (oblique lateral view) to expose the left facet joints (circles) at the left, and the right facet joints at the right. Note the more irregular joint space and some sclerosis in the right facet joints. This is indicative of osteoarthritis of the right thoracic facet joints.

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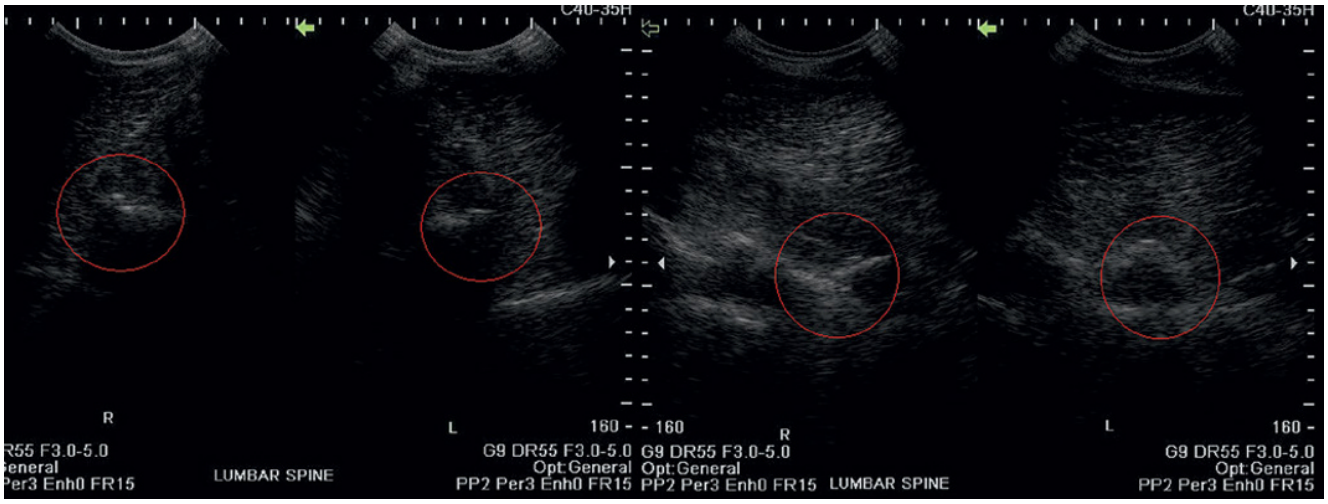


Figure 6.34. Ultrasonographic images of the lumbar spine. Transverse views (left images) of the left side and right side; the facet joint surfaces and the joint spaces are seen in the circles. Right side, two longitudinal paramedian views. The respective facet joints are seen in the circles; the joint space and both facets are in the left circle, and in the right circle the joint space is not visible.

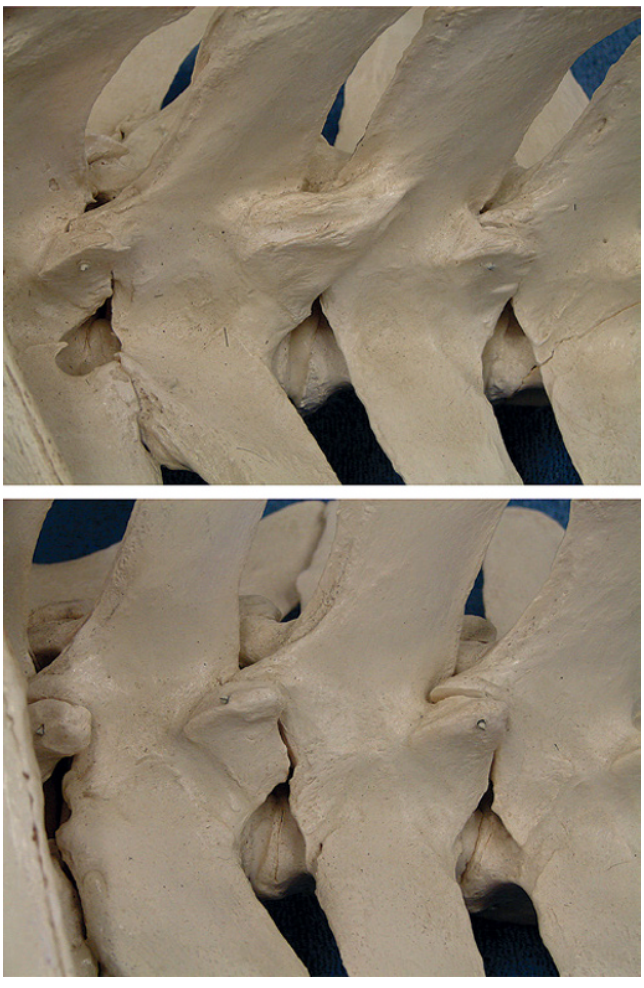


Figure 6.35. Anatomical specimen of the lumbar spine with the facet joints visible. In the lower image, the facet joints have smooth edges and appear to be normal. In the upper image the facet joints have more irregular edges when compared with the facet joints in the lower image. Complete ankylosis is present in one of the facet joints.

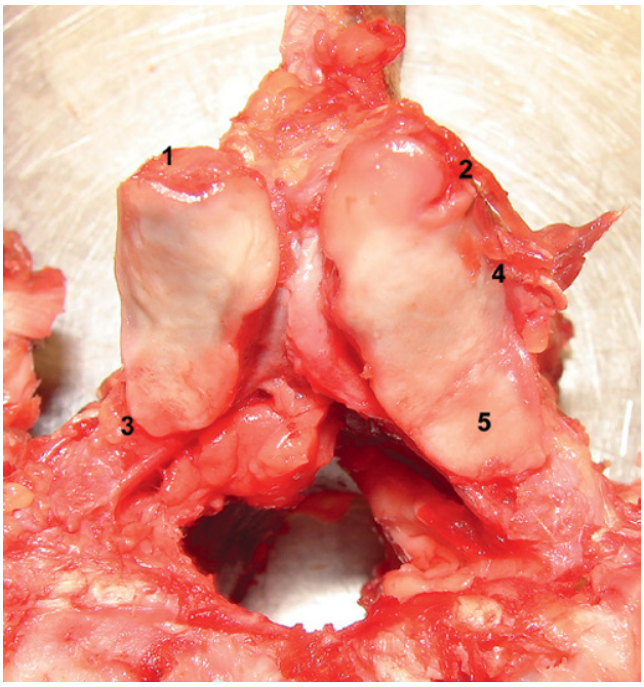


Figure 6.36. Necropsy specimen of the thoracic facet joints of T7. Note the asymmetry and difference in size from the left and right facet joint. 1 = erosion of cartilage and hemorrhage from the subchondral bone, 2 = fraying of the cartilage, 3 = compression damage of the cartilage, 4 and 5 = some ulceration of cartilage.

When muscles are evaluated with Doppler ultrasonography, vascular patterns within the muscles can provide information about myositis and muscle tension, in which cases increased and decreased vascular activity can be present, respectively.

Scintigraphy is a perfect modality for imaging the vertebral column. When the resolution of the gamma camera is adequate, it is possible to differentiate increased uptake of radiopharmaceutical in the vertebral body from that

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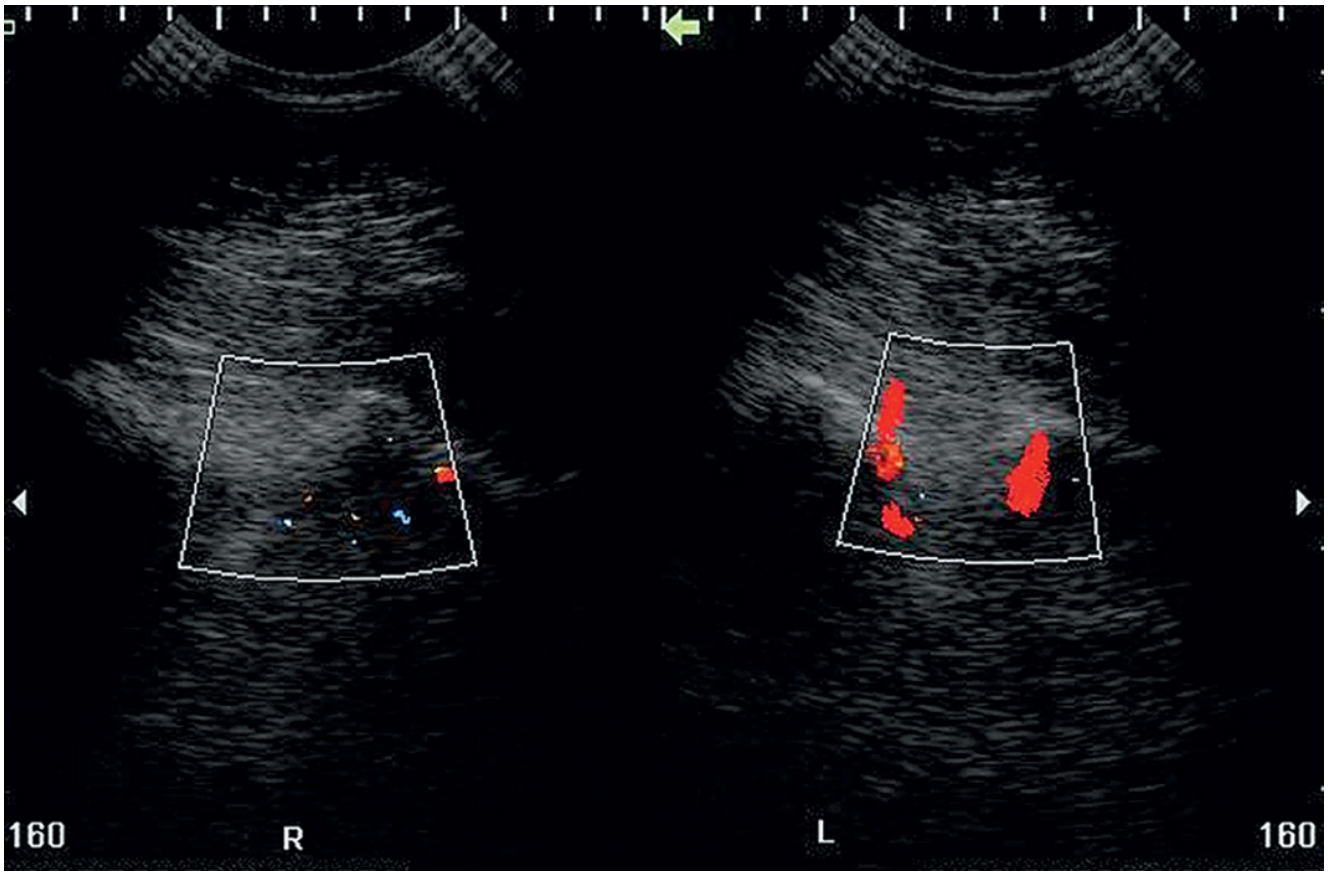


Figure 6.37. Ultrasonographic image of the lumbar facet joints. Doppler is used to identify higher blood flow around the facet joint at the left facet joint (right in the image).

just dorsal in the facet joint. A recent study showed 61.5% of horses with back pain complaints had an increased uptake of radiopharmaceutical in one or more facet joints.[16] Comparison of images obtained from the left, right, and dorsal aspects can assist in determining the exact location of higher bone metabolism in a facet joint.

The final diagnosis of equine vertebral facet joint syndrome is often a summation of the results of two or three imaging techniques. When the results of Doppler ultrasonography and scintigraphy are combined, it may be possible to differentiate between synovitis with its increased vascular activity around the facet joint and osteoarthritis, which can have both increased uptake of radiopharmaceutical in the region of the facet joint and increased vascular activity.

The study by Gillen concluded a high predictive value for negative scintigraphy findings to exclude osteoarthritis of facet joints as the cause of back pain in horses.[16] The authors found a high predictive value for scintigraphy in the detection of radiographic lesions and back pain. Thus, scintigraphy is a valuable tool in the evaluation of thoracolumbar pain.[16]

Local anesthesia is not a reliable tool for confirming facet joint pain. Anesthetic solution is deposited in muscle tissue rather than in the facet joint or its capsule and diffuses rapidly within the muscle, affecting nerve sensation in the rami of the spinal nerves. Furthermore, the reflectory spasm that is caused by the facet joint arthritis takes several hours to relax, making it very difficult to adequately interpret the results of the block. In humans, anesthesia of a suspect facet joint is performed on a more regular basis, but people can communicate and tell the examiner when the main joint pain they were experiencing is diminished, even when a muscle spasm is still present.[31]

Treatment

Rehabilitation is a significant factor influencing the final outcome of facet joint arthritis and vertebral facet joint syndrome.[29] The primary goal is to break the vicious cycle of inflammation that triggers the nerves, which leads to muscle spasm, immobility of the spine, and repeated injury. Treatment can be aimed at multiple spots in this cycle, but the best outcome can be expected with complex treatment plans that deal with as many aspects as possible.

Anti-inflammatory therapy is essential. NSAIDs, especially phenylbutazone (1 g/500 kg BID), flunixin meglumine (0.5 mg/kg SID), naproxen (10 mg/kg SID), and meclofenamic acid (2 mg/kg SID), must be administered for a prolonged time, often up to 1–2 months. In that case, additional medication to prevent gastrointestinal side effects may be needed. The newer NSAID firocoxib may be a good choice for horses with gastrointestinal side effects that are on other NSAIDs, but it must be limited to a 3-week period of medication, according to the manufacturer. Some positive experience

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has been obtained in back pain cases with bisphosphonates, especially when osteolysis is present.[6]

More localized anti-inflammatory therapy is obtained by direct injections of a corticosteroid into or close to the facet joint. The author's drug of choice is triamcinolone (10–30 mg intra-articularly). The triggering facet joint must be established by scintigraphy that shows increased uptake of radiopharmaceutical in the area of the joint, possible radiographic changes, and/or ultrasonography that reveals increased vascular activity around the facet joint. With ultrasound guidance, a 20-gauge spinal needle is inserted into the dorsal aspect of the facet joint, and 10–30 mg of the corticosteroid is injected into or close to the joint capsule. Because the medication is deposited in the joint capsule or in the adjacent multifidus muscle with an important proprioceptive function in intervertebral motion (the multifidus muscle stabilizes the spine and facilitates limb motion without deformation of the axial skeleton), the use of sarapin to reduce pain sensation is contraindicated.

Anti-inflammatory treatment should diminish muscle spasms, and quite often, muscle relaxation is observed within 2–3 days of facet joint injection. When only systemic medication is used, beneficial effects occur more slowly, and muscle relaxation can take up to 2–3 weeks. Use of a muscle relaxant such as methocarbamol (15–25 mg/kg slow IV infusion) can be effective in reducing the muscle spasm, but the supportive function of the spinal muscles may be diminished, so care must be taken to reduce the possibilities for injury to the spinal structures. No turnout and a reduced workload are important in a horse that is on muscle relaxants; however, this may compromise an effective rehabilitation program.[29]

After reduction of inflammation and the adjacent muscle spasm, the next step in treatment is the design of a rehabilitation program aimed at gaining more mobility in the affected part of the spine as well as the development of better muscle support for that region. In the author's practice, rest is prescribed for 4–6 weeks. Pasture is ideal, but if the horse is turned out with others, it must only be with pasture mates that will not force the patient to run. The more the horse walks, trots, and canters at its own pace, the better the mobility achieved. Grazing with the head down opens the facet joints in the thorax and lumbar region, thus counterbalancing the spasms in the dorsal epaxial muscles. In humans, one of the modern rehabilitation techniques with tendon and ligament injury is eccentric exercise. The stretching effect of the grazing position on the dorsal epaxial muscles as well as the tendons and ligaments around the spine may be a very important side effect of being out in pasture. When a patient cannot go out in pasture, mimicking this position by feeding small portions of feed and hay on the ground at multiple times during the day is a good alternative.

After the initial period of rest, a period of gradually increased work must be incorporated in the rehabilitation program. At first, just circling exercises at the walk and later at the trot facilitate more lateroflexion, thus providing stretch to the outside part of the spine and some compression and rotation to the inside part of the spine. By modulating the size of the circle and the direction of the circle in a 1- to 2-minute pattern, gradual mobilization of the entire spine can be achieved. In a later stage, some cantering can be introduced in circles as well as at the track or over terrain, together with more strenuous exercises to stimulate more powerful muscle contraction. In the author's clinic, a complete rehabilitation program can easily take 4–6 months, after which a large number of cases are fully serviceable at the original level of performance or even higher.

Prognosis

The prognosis for horses with thoracic or lumbar facet joint arthritis depends on a number of factors. The discipline in which the horse performs is very important. In the racing horse, thoracic facet joint arthritis has a better prognosis than facet joint arthritis of the lumbar facet joints, whereas the prognosis for thoracic facet joint arthritis for a barrel racer may be more performance limiting than lumbar facet joint arthritis. Three-day event horses and jumpers often have a more guarded prognosis than dressage horses. In the dressage discipline, rehabilitation of the core muscles of the trunk can assist in stabilizing the spine and prevent recurrent injury. Racing trotters seem to have the worst prognosis for full return to a racing career when diagnosed with facet joint arthritis or thoracolumbar pathology in general.

DISCOSPONDYLITIS

Etiology

Discospondylitis is an inflammatory condition involving the vertebral bodies adjacent to the symphysis between two vertebrae, and includes the intervertebral disc.[30] The most common cause is a septic process, in which the infection is caused by hematogenous spread. The primary site of infection should be identified. Blood flow through vertebral endplate venous channels is relatively slow and tortuous, which may promote localization of the infection in the vertebra and spread to adjacent discs.[36] Bacteria isolated from cases of equine adult discospondylitis include *Brucella abortus*, alpha-hemolytic *Streptococcus*,[1] coagulase negative *Staphylococcus*,[5] and *Staphylococcus aureus*.[35] *Rhodococcus equi* has been implicated in a foal.[4]

Traumatic discospondylitis has been documented in clinical cases and at postmortem examination.[30] Intervertebral disc lesions, including fissuration, calcification, and herniation, have been described ultrasonographically and at postmortem examination at the lumbosacral junction and the cervical spine.[11]

Clinical Signs

Signs of weight loss, back or neck pain, fever, stiffness, and ataxia may all be notable in discospondylitis. Atrophy of the epaxial muscle in the thoracolumbar region may be evident in some horses suffering from chronic discospondylitis in this region.[2] Some horses are so affected that they are reluctant to eat off the ground. Lateral neck flexion can be markedly obtunded as well, and the abdomen may be held rigid as though intra-abdominal pain were present. When compression of the spinal cord or root nerves is present, neurological

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symptoms can be observed.[10] Blood analysis may reveal a leukocytosis and elevated fibrinogen.

Diagnosis

Diagnosis of discospondylitis is based upon appropriate clinical signs and radiographical findings.[1] Radiographic features of discospondylitis include lysis and/or proliferation of the vertebral bodies adjacent to the affected disc. As the disease progresses, the intervertebral disc space may narrow and collapse, and a smooth bony bridge may unite the affected vertebrae.[36] Scintigraphy,[23] computed tomography,[34] and ultrasonography[35] have been used to facilitate the diagnosis of equine discospondylitis and vertebral osteomyelitis. Scintigraphy may be useful to determine whether multiple sites of bone involvement are present.[37]

Treatment and Prognosis

Successful treatment of discospondylitis usually involves long-term antimicrobial administration.[2,23,36] In specific cases surgical curettage of the lesion is an option when access to the lesion is possible.[35] If the causative organism is not isolated, as is usually the case, broad-spectrum antimicrobials should be administered. Intravenous antimicrobials often reach higher tissue concentrations than oral medication and are recommended at the onset of therapy. Antimicrobials allowing distribution and penetration into the bone should be administered; therapeutic options may include fluoroquinolones, macrolides, cephalosporins, and potentiated sulphonamides.[2] The total duration of antimicrobial treatment can be 4–6 months, which can make the treatment very costly.

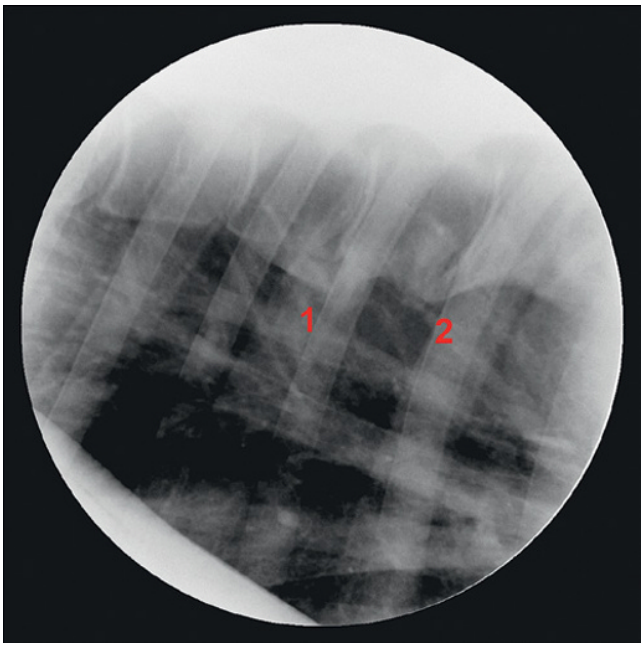


Figure 6.38. Radiographic image of the thoracic spine of a horse with severe spondylosis. There is proliferation of bone at the ventral aspect of the vertebral bodies. 1 = no complete contact between the proliferations, 2 = seemingly complete bridging of the intervertebral disc space.

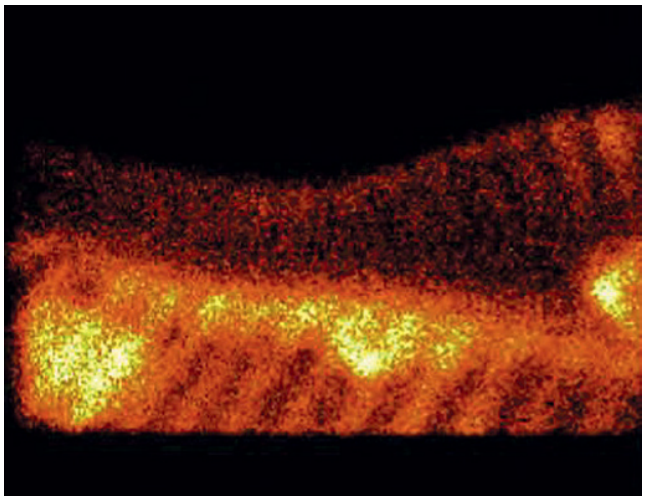


Figure 6.39. Scintigraphic image of a horse with IRU in the midthoracic spine, suggestive of spondylosis.

Although the prognosis for horses with discospondylitis has generally been considered guarded, favorable outcomes have been associated with early detection, nonseptic lesions, the absence of spinal cord compression, administration of long-term antimicrobial therapy, and surgical curettage.[1,23,35]

SPONDYLOSIS

Etiology

Spondylosis (deformans) is more common than discospondylitis. It involves the vertebral body only, without including the intervertebral disc.[30] It is a degenerative condition affecting the vertebral body on the ventral aspect in the thoracic spine and merely the lateral aspect in the lumbar spine. Spondylosis results in osteophytes, often seen in several adjacent vertebrae. The most common location for these osteophytes is the vertebral segment between T10 and T14.[10,13] Probably because of regional biomechanical influences, the osteophytes are located at the ventrolateral aspect of the vertebral bodies in the thoracic region. Pathogenesis of osteophyte formation involve mechanical stress at the attachment of the most peripheral fibers of the intervertebral disc and the ventral longitudinal ligament (enthesiophytes).

Postmortem results of acute cases showed hemorrhage and fraying of collagen fibers of the intervertebral disc, no deeper than the annulus fibrosis. The facet joints in these cases had been jammed together, and there was evidence of fraying and erosion of cartilage and hemarthrosis.[30] The osteophytes resulted in reduced mobility of the spine at the site of the osteophytes (with the end stage of complete ankylosis), putting more of the motion load on the adjacent vertebrae that induces more active bone remodeling, often visible on scintigraphic examination.

Clinical Signs

In the more acute stage, when active bone remodeling is in progress, the horse can show severe back

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Figure 6.40. Seven-year-old Thoroughbred with marked kyphosis in the thoracolumbar spine. No clinical symptoms of back pain were present.

pain with generalized stiffness and reluctance to work. These cases seem to relate to the lay term of cold back, in which saddling, mounting, or starting to ride initiates a violent reaction of the horse, including bucking, running away, and laying down. In older cases the condition is less painful but more limiting in the motion of the spine, giving the horse a more stiff appearance. In chronic cases, there is often loss of cartilage in the facet joints, and the dorsal spinous processes can come close or make contact, giving the horse the appearance of a hollow back or acquired lordosis. This condition seems to be more common in event horses, show jumpers, and hunters than in Thoroughbred and Standardbred racehorses. Working draft horses endure enormous forces on their spine when pulling heavy loads and seem to be more predisposed for spondylosis.[30]

Diagnosis

Radiography and scintigraphy (Figures 6.38 and 6.39) are the main diagnostic tools to confirm spondylosis. Scintigraphy can show the amount of bone activity as an indicator for acuteness and activity.

Treatment

Medication with NSAIDs is the only option. No surgical techniques have been described in the horse to stabilize the spine.

Prognosis

Prognosis for spondylosis to return to athletic career is guarded.

SCOLIOSIS, KYPHOSIS, AND LORDOSIS

These conditions can be congenital defects. The basic pathological defect of both scoliosis and lordosis is hypoplasia or aplasia of the articular facets of the intervertebral articulations, primarily in the thoracic spine.[38] Acquired lordosis can be seen in the older horse with osteoarthritis and ankylosis of the facet joints.[30]

Kyphosis in the lumbar spine seems to occur in Thoroughbreds (Figure 6.40). No data is available on the performance of horses with this condition.[6] With congenital deformities, it is likely that these conditions are not painful, but that they can limit athletic careers due to limitations in motion and propulsion. In the acquired cases, the initiating condition (osteoarthritis) can cause pain and clinical symptoms, as can localized pathology of the supraspinous ligament caused by focal kyphosis.

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THE NECK AND POLL

Rob Van Wessum

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This section emphasizes the cervical disorders that directly cause or contribute to lameness. The developmental cervical disorders, most often causing neurological symptoms, are covered in Chapter 12.

As described in the first part of this chapter, the cervical spine has an important function in locomotion.[6] Any dysfunction of the cervical spine can cause an altered gait pattern, often described as lameness. Sometimes the symptoms of the dysfunction of the cervical column are observed as altered behavior (head shaking, not connecting with the rider's hand, pulling, bucking, rearing, etc.) but are presented to the veterinarian as lameness. Other cases are presented as lower limb lameness or shoulder lameness.[15] The following sections cover the common causes of cervical dysfunction and their symptoms, diagnosis, and treatment.

NUCHAL LIGAMENT

The nuchal ligament connects the caudal aspect of the skull, dorsal processes of the cervical vertebrae, and withers region (the dorsal processes of the first thoracic vertebrae) and has an important function in supporting the entire neck. Elastic fibers in this structure store kinetic energy during locomotion and release the energy at specific phases of the locomotion when certain muscle groups move or activate parts of the cervical spine and the attached front limbs, thus contributing to an efficient gait. The head and neck function as a sling during propulsion and have a coordinating function in the specific pattern of the gait. For instance, the periodic sinusoid motion instigates activation patterns in the muscles of the neck and shoulder.[10]

Desmopathy at the origin of the nuchal ligament located at the base of the skull and the insertion to the dorsal processes of the cervical and cranial thoracic vertebrae is associated with pain and can alter this sinusoid pattern, thus altering the gait.[4,12,13] Tension in the nuchal ligament and its attachments is greater, and symptoms of desmopathy can be more pronounced in specific positions, such as when the neck is more flexed, the head is more parallel to the vertical or even behind the vertical, or the head is in a higher or lower position.

Etiology

Etiology of desmopathy of the nuchal ligament is very often associated with trauma. Falling down or pulling backward when tied, being caught in fences, and trailering accidents can put extreme force on the ligament and its attachments, causing trauma to the nuchal ligament and other structures in the neck and poll. Training methods such as tying the horse's head to the side or between the front limbs, as is sometimes practiced in preparation for Western pleasure, or hyperflexion or "Rollkur," as is practiced by some dressage trainers, can predispose horses in these disciplines for nuchal ligament pathology.

Clinical Signs

Symptoms of nuchal ligament pathology are diverse and very often more visible when the horse is worked. The horse may appear stiff, the neck does not move up and down as much as usual at the walk, and when worked on circles the head is out with no flexion of the neck according to the diameter of the circle.

When ridden or driven, the contact with the bit through the reins or lines can feel different to the rider/driver, with one side feeling more rigid or as if the horse is pulling on one side. Dental issues can have the same symptoms, so excluding dental problems helps to confirm or at least suspect neck problems. Particular head positions can cause pain, making the horse behave defensively or reluctant to perform certain exercises.

The neck is an important balancing tool for the horse during work, so in disciplines in which balance is of major importance for performance, such as jumping, dressage, and eventing, general lack of performance can be one of the more indistinctive signs.

Diagnosis

Desmopathy can be found at the attachment of the ligament to the bone, which may cause enthesophytes that can be seen with radiographs. It also may be located in the ligament itself, in which case the altered fiber pattern is visible on ultrasonographic examination. Nuclear scintigraphy can show the increased bone turnover at the attachment sites (Figure 6.41). MRI and CT can give detailed information about the condition of the



Figure 6.41. Radiographic image of the cervical spine with C5, C6, and C7 in view. There is mild enlargement of the facet joints between C5 and C6 and C6 and C7, with irregular joint spaces. This is indicative of mild to moderate osteoarthritis of the caudal cervical facet joints and is a common finding in horses without clinical signs.

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more cranial cervical vertebrae and the ligament. Newer MRI equipment (with smaller bore and larger aperture) facilitates imaging of the more caudal vertebrae, but in most cases imaging is limited to the level of the fourth or fifth cervical vertebra.

Treatment

Therapy for desmopathy of the nuchal ligament is similar to that of any desmopathy: rest and anti-inflammatory medication, as well as possible extracorporeal shock wave treatment. Biological treatments with stem cells, A-cell, and PRP are in the stage of experimental use, and studies to be published in the near future on these therapy modalities will show their effectiveness in treatment of desmopathies in general and in the nuchal ligament in particular.

A specific structure in the poll, the nuchal bursa, which is present at the dorsal aspect at C1 in nearly all horses and less frequent at C2, can be involved in pathology of the nuchal ligament. Bursitis is considered to be a rare condition. However, many veterinarians in the field of sports medicine diagnose bursitis of the nuchal ligament quite frequently. It is difficult to completely confirm this diagnosis on ultrasonographic examination alone. CT or MRI imaging, performed under general anesthesia, can give a more detailed image of the bursa(e) and its adjacent structures along with a more confirmed diagnosis. A common treatment modality for bursitis of the nuchal bursa is the ultrasound-guided injection of corticosteroids into the bursa. Calcifications in the bursa region are observed quite frequently in the author's clinic on routine radiographs of this region; however, in these cases the owner of the horse may not always remember any treatment or problem in this region.

Prognosis

When given enough time for complete recovery and rehabilitation, prognosis for nuchal ligament desmopathy is favorable.[4] The only exception may be the high-level dressage horses when the initial injury to the ligament has not been recognized and has developed into a more chronic phase or is considered to be a repetitive stress injury. In these cases, when the horse's head is in a very high and upright position, as needed for the highest grades of collection, strain to the ligament is the highest, and the horse can show pain or defensive behavior such as rearing or shaking the head.

When calcifications are present due to mineralization of a hematoma or corticosteroid deposition, signs of altered behavior or performance are common, and prognosis is guarded for complete recovery to the initial level of performance.

CERVICAL FACET JOINTS

Osteoarthritis of the cervical facet joints is very common in many horses, even without clinical signs.[4,11] Enlargement of the more caudal facet joints with irregularly shaped or narrowed joint spaces is common on cervical radiographs in horses of any breed at a slightly older age (older than 4–5 years) (Figure 6.41).

Etiology

Osteoarthritis of the cervical facet joints can be the result of osteochondrosis of the articular surfaces, as in any other joint affected by osteochondrosis. Trauma to the neck is often a cause for facet joint arthritis. The trauma may be the result of trailering accidents, falling or hanging in cross ties, running into objects with the neck, falling during exercise (racing, eventing, jumping), flipping backwards, etc. Instability of the neck due to malformation of vertebrae or joint surfaces predisposes the horse to facet joint arthritis, and it is often found in conjunction with several congenital or developmental cervical malformation syndromes.

Clinical Signs

Osteoarthritis of the cervical facet joints can cause a great diversity of symptoms. At first, pain can cause behavior similar to that described for nuchal ligament pathology.

When severe bone proliferation is present at or around the facet joints, mobility can be limited, thus limiting athletic performance. In dressage, the horse may be reluctant to bend the neck in a lateral direction, bring the head vertical in the collection position, or stretch down. A jumper can be difficult when asked for short turns or lack balance during the jump due to neck stiffness, while the barrel racer can be difficult to turn around the barrels.

Due in which several muscles originate at locations in the neck and have their insertion to the scapula (trapezius muscle, rhomboideus muscle) or humerus (brachiocephalicus muscle), contraction of these muscles can cause pain in the arthritic facet joints and thus alter the gait (e.g., shorter anterior stride, reduced protraction of the scapula) and mimic shoulder or even lower limb lameness.[15]

Neurological symptoms can occur when bone proliferation of the arthritic joint structures protrudes into the spinal canal, with lower hindquarter activity as the most common sign. When the bony proliferation invades the intravertebral foramen, root nerves can be compressed.[11] The cervical nerves between C5 and C6, C6 and C7, and C7 and T1 (the sixth, seventh, and eighth cervical nerves) form the brachial plexus, so compression of these root nerves can be sensed as pain in the limb, and limb lameness may be observed.[11,14] When root nerve compression is present, a patchy spot of sweating may be noticed on one or both sides of the neck and/or thorax.

Diagnosis

At the clinical examination, cervical facet joint arthritis can be suspected when the range of motion in the neck is limited. With complete flexion to the side, the horse should be able to flex the neck laterally, touching the thoracic wall with its nose without rotation in C2 in both directions. A 20–30% limitation in the total range of motion in the lateral plane is found in many older horses without clinical symptoms and can be considered normal. Cervical facet joint arthritis is highly suspected with an asymmetric limitation of the range of motion.

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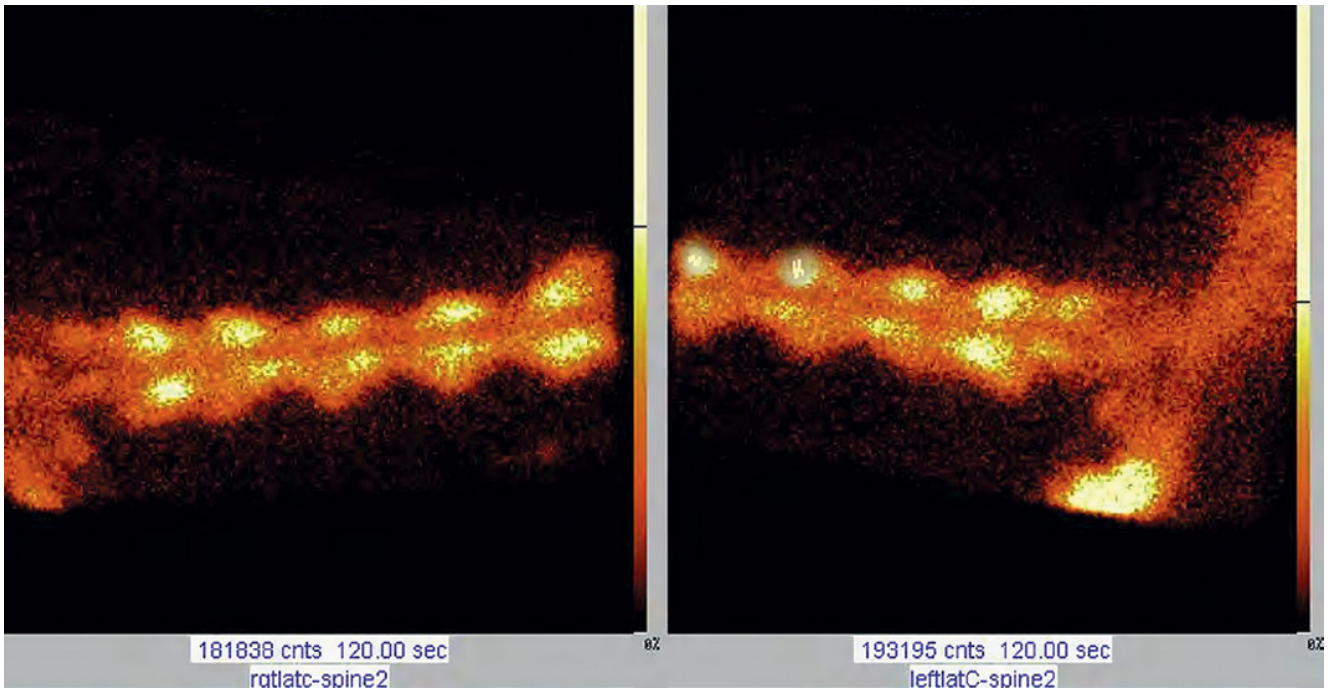


Figure 6.42. Scintigraphic images of the cervical spine. In the left image, IRU in C7 is visible at the right side of the neck in the arch and the body of C7; this is normal. In the right image, there is IRU visible in the arches of C4 and C5 on the left side of the neck, indicative for active facet joint osteoarthritis.

Range of motion in the dorsoventral plane can be tested by manually stretching the neck upward, while a carrot stretch between the front limbs can show the range of motion in the ventral direction. The dorsoventral range of motion tests are not as easy to interpret as the lateral plane, because they cannot be checked for symmetry, so there is no clear maximum range of motion to be defined. Palpation of the facet joints, left and right, can provide information about their size and shape, as well as pain when palpated.

When a particular joint is identified as highly suspected for causing the symptoms, ultrasound-guided intra-articular block of the facet joint can be used to verify the cause. This can best be seen when the horse is performing the work in which the symptoms are most obvious, for instance, so under saddle, at work, etc.

Radiography is the first imaging technique of choice.[1,7–9] Lateral views with a small degree of angle from the horizontal plane can be used to project the left or right facet joint without superimposing on top of the ipsilateral joint.

Ultrasonography can be used to image the facet joints as well, providing visual information about joint space size and shape and bone proliferation at the edges of the facet joint. Ultrasonography is also very helpful in treating cervical facet joint arthritis, because ultrasound-guided injection of the facet joint is not difficult.[2]

Nuclear scintigraphy (Figure 6.42) can be used to identify facet joints with an increased uptake of radiopharmaceutical as a sign of active inflammation to distinguish the older, consolidated proliferations, which are visible on radiographs, from signs of active arthritis/synovitis. This is useful in making decisions about the injection of corticosteroids.

MRI and CT imaging give much more detailed information about the size and shape of the facet joints and their potential compression effects on root nerves and the spinal cord but are limited to the more cranial facet joints. These techniques also involve general anesthesia and carry higher costs.

Myelography is considered the gold standard for identifying possible involvement of root nerves and/or spinal cord compression; however, this technique is only 50% accurate at identifying the specific location of compression of the spinal cord when compared with necropsy findings (Pease AJ, personal communications). Electromyography can be used to identify changes in muscle signals in the segmental muscles in the neck and thorax to show decreased innervation of these muscles as a sign of root nerve compression.[16]

Treatment and Prognosis

As with any osteoarthritis, treatment for cervical facet joint osteoarthritis can include medication with NSAIDs. Intra-articular medication with corticosteroids, deposited in or close to the joint under ultrasound guidance, has been described as a useful treatment. Triamcinolone is the corticosteroid of choice because it does not cause as much irritation in the adjacent structures such as the ligamentous tissue and muscle as methylprednisolone acetate when not deposited in the joint itself.

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RADICULOPATHY

When one or more segmental root nerves in the cervical spine are compressed, damaged, or irritated due to trauma or enlarged facet joints, lameness symptoms may occur.[5] In these cases, blocking of the distal limb nerves will give a negative outcome. Physical exam might show a reduced mobility in the neck, but sometimes the musculoskeletal exam is uneventful. The neurological exam however will show clear signs of nerve impingement with loss of skin sensitivity in the affected area, while often the adjacent segments show an augmented response to skin sensitivity testing. Ultrasonography can be a useful tool in identifying enlargement of the soft tissues of the cervical facet joints with enlargement of the synovial membrane and joint effusion when radiographs are not showing clear indication of affected cervical facet joints. Imaging using Doppler can identify the most affected region.

Osteoarthritis of facet joints, especially in the more caudal aspect of the cervical spine or cranial thoracic spine, may result in an associated enlargement of the joint capsule and subsequent pressure to the spinal cord or root nerves. Oral dexamethasone may be used to reduce this swelling and the signs of nerve compression, or infiltration of the epidural space with corticosteroid can be performed.[11]

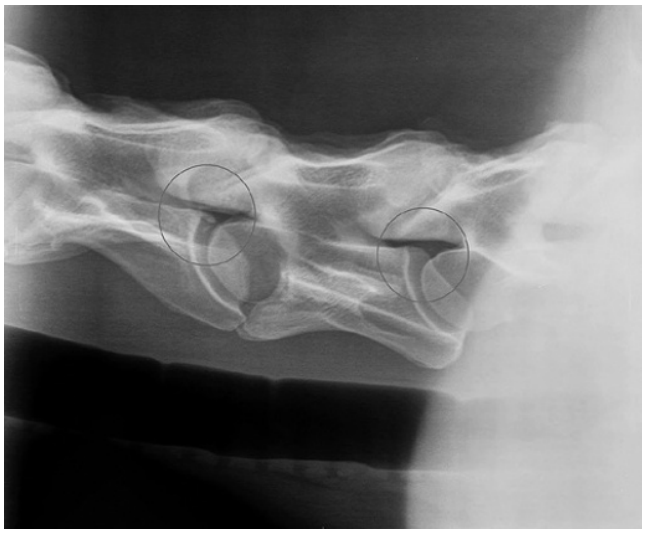


Figure 6.43. Radiographic image of the cervical spine. Note the change at the caudal aspect of the end plate of C5 and the small fragment (left circle) when compared with the clean appearance at C6 (right circle). This not only is most likely caused by trauma to the intervertebral ligament at the ventral base of the spinal canal but also can be caused by local mineralization within the intervertebral disc. In the acute stage, swelling can cause neurologic symptoms related to compression of root nerves or the spinal cord.

DISCOSPONDYLITIS

As described in the section on the thoracolumbar spine, discospondylitis can occur in the cervical spine, most likely in the caudal cervical vertebrae. Signs of neck pain and front limb stiffness or lameness may be present. Diagnosis can be made with radiography and scintigraphy, and the prognosis is guarded.[3,4,14]

CERVICAL FRACTURES

As described earlier in this chapter, fractures of cervical vertebrae can occur, and in nearly all cases this is due to severe trauma. Fractures of facet joint surfaces are rather common, but laminar fractures and fractures of the body or the arch of the cervical vertebrae also have been described. Depending on the location and the extent of the fracture, hemorrhage and swelling can extend to the spinal cord and cause ataxia. Horses with an acute fracture show pain and are unable to move the cervical spine in the affected region. Radiography is the diagnostic tool of choice, and in most cases lateral views are sufficient to show the fracture. Ventrodorsal views can assist in determining the extent and location. In specific cases scintigraphy can give adjacent information from 7 to 10 days after the injury, when displacement of the fracture and recognition of the fracture line are difficult on the initial radiological examination. Ultrasonographic examination of the cervical spine is an option but primarily shows fractured facet joints. It is difficult to make an interpretation of larger fractures through the body or arch of the vertebra.

When there is no damage to the spinal cord or the adjacent root nerves, and there is no displacement of the fragments, the prognosis can be moderate. Treatment includes stall rest or confinement in a small pen or paddock to reduce motion. However, callus formation can cause compression of the root nerves or the spinal cord in a later stage, and fractures of facet joint surfaces often result in facet joint arthritis. Trauma to ligamentous structures and the intervertebral disc may be not visible on initial radiographs (Figure 6.43) and can easily be overlooked or not recognized, but may result in discospondylitis, spondylitis, or osteoarthritis in a later stage.

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CHAPTER

Principles of Musculoskeletal Disease

JOINT INJURIES AND DISEASE AND OSTEOARTHRITIS

C. Wayne McIlwraith

Arthritis may be defined simply as inflammation of a joint. It is a nonspecific term and does little to describe the nature of the various specific entities that affect equine joints. The role of inflammation also varies considerably among the different conditions. Such general terms are no longer appropriate in the management of equine joint conditions. Specific diagnoses must be made to effectively treat the horse and make accurate prognoses.

Updated knowledge of etiology, pathogenesis, diagnosis, and treatment of each of the different equine joint conditions has been recently published in a separate textbook on equine joint disease.[68] To understand joint disease in the horse, a basic knowledge of the anatomy and physiology of joints, as well as their pathobiologic responses, is necessary.

ANATOMY AND PHYSIOLOGY OF JOINTS

Classification

Joints are most often classified according to their normal range of motion. Three groups are recognized: synarthroses (immovable joints), amphiarthroses (slightly movable joints), and diarthroses (movable joints), also known as synovial joints. Synovial joints have two major functions: (1) to enable movement and (2) to transfer load.[107] The entire structure is invested by a fibrous capsule. Diarthroses include most of the joints of the extremities. Because these are the joints that we are essentially concerned with in equine lameness, their anatomy and physiology are described in detail.

General Anatomy

The synovial or diarthrodial joint consists of the articulating surfaces of bone that are covered by articular cartilage and secured by a joint capsule and ligaments and a cavity within these structures containing synovial fluid (Figure 7.1). The joint capsule is composed of two parts: the fibrous layer, which is located externally and is continuous with the periosteum or perichondrium (and, ultimately, bone), and the synovial membrane, which lines the synovial cavity where articular cartilage is not present.

The fibrous portion of the joint capsule is composed of dense fibrous connective tissue that provides some mechanical stability to the joint. The collateral ligaments are associated with the joint capsule. Intra-articular ligaments normally have a synovial membrane cover. Type I collagen predominates in the fibrous connective tissues of the joint.[108] It is vascular and has afferent pain fibers. Using immunohistochemistry with substance P as a more sensitive way of identifying the distribution of nociceptor fibers, it has been shown that sensory nerve fibers are present in the synovial membrane and subintimal layers as well as collateral ligaments, suspensory ligament, and distal sesamoid ligament attachments.[10]

Adams and Stashak's Lameness in Horses, Seventh Edition. Edited by Gary M. Baxter.
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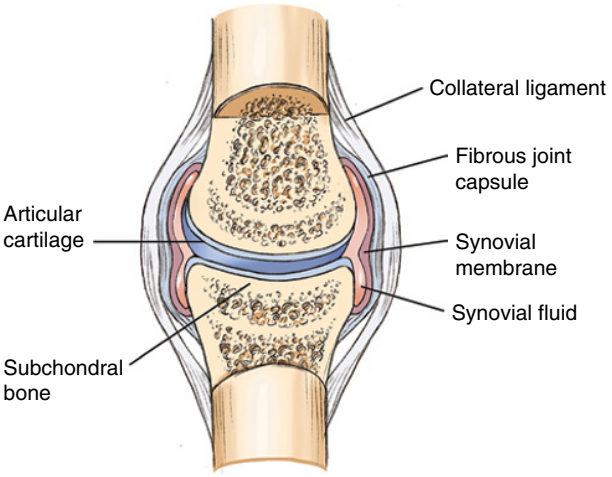


Figure 7.1. Diagram of a typical synovial joint.

The insertions of the fibrous capsule and articular ligaments into the adjacent bones demonstrate a zonal organization with a gradual transition of joint capsule and ligaments to mineralized fibrocartilage and then to bone. This enhances the ability of the insertions to distribute forces evenly and decrease the likelihood of pull-out failure.[55]

Stability of the joint is provided by the bony configuration of the joint, the ligamentous and capsular support systems, and the musculotendinous units controlling the joint. In addition, there is a negative hydrostatic pressure within the synovial cavity of normal joints, and this is considered to impart a "suction" and stabilizing effect.[85]

It has also been demonstrated that only the periarticular soft tissues (capsule, ligaments, and synovial tissue) and bone have significant force-attenuating properties (these studies were done with longitudinally applied, external impulsive force on adult bovine interphalangeal joints).[83] Articular cartilage and synovial fluid have little effect on force attenuation. This emphasizes the importance of both soft tissues of the joint and bone in providing shock absorption.

Synovial Membrane Structure and Function

Equine synovial membrane (also called synovium) is smooth and glistening in some regions of the joint and is formed into numerous villi in other regions. These villi have specific locations and a diverse morphology. Although villi proliferate in association with trauma and other insults, they are present in the fetus and, therefore, at birth.

Histologically, the synovial membrane is a modified mesenchymal tissue and consists of two layers: the intima, an incomplete cellular lining layer that lies next to the joint cavity and overlies a deeper layer of connective tissue (fibrous, areolar, or adipose) termed the subsynovial layer, or the subintima. Generally, the synovial membrane is fibrous and flat in regions where it is subjected to increased pressure; it is areolar and may be folded where it must move freely and independently of the joint capsule. The areolar subintima consists of loose



Figure 7.2. Diagram of a portion of synovial membrane demonstrating synoviocytes (types A and B, a and b) within the intercellular stroma of the intima, capillary (c), and collagenous matrix of intima (d). Tissue deep to this is considered subintimal.

fibrous connective tissue that continues into the central core of the villi. Numerous blood vessels are present.

The synovial cells of the intima (synoviocytes) form an incomplete layer that is 1–4 cells thick, and no basement membrane can be detected (Figure 7.2).[63] The synoviocytes have been classified at the ultrastructural level into two principal types designated as types A and B. The type A cells resemble macrophages and type B cells resemble fibroblasts. Intermediate cells are also observed. A concept has evolved in which type A and type B cells are not distinct, but rather merely cells whose differences in morphology reflect the functions they currently perform.[63]

Numerous blood vessels are present in the subintima and extend to within 5–10 μm from the intimal surface.[63] It appears that each joint has a dual nerve supply consisting of specific articular nerves that reach the joint capsule as independent articular branches of adjacent peripheral nerves and by articular branches that are nonspecific and arise from related muscle nerves.[63]

The three principal functions of the synovial membrane are phagocytosis, regulation of protein and hyaluronan (HA) content of the synovial fluid, and regeneration.[63] Excessive phagocytic activity or disruption of lysosomal or cellular membranes releases enzymes into the environment (a typical feature of

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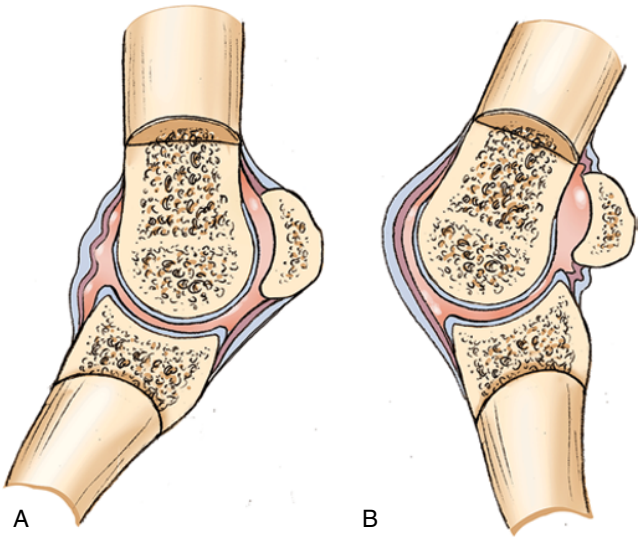


Figure 7.3. Diagram of a metacarpophalangeal joint demonstrating how redundant synovial membrane gathers at the dorsal aspect on extension (A) and at the palmar aspect on flexion (B).

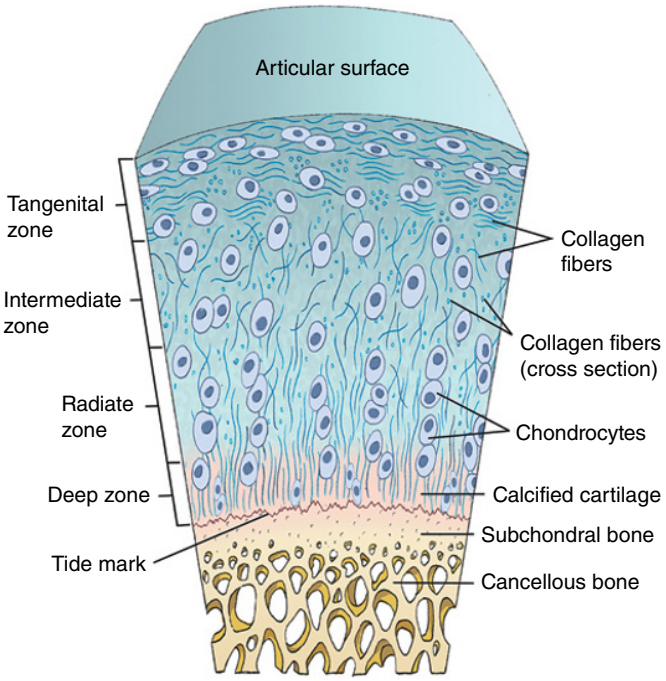


Figure 7.4. Diagram of adult articular cartilage showing the four layers and the arrangement of the chondrocytes and collagenous fibers.

synovitis). The synovial membrane acts as an important permeability barrier that, in turn, controls synovial fluid composition. Most small molecules cross the synovial membrane by a process of free diffusion that is limited by the intercellular spaces in the synovial membrane rather than by blood vessel fenestrations.[101] In traumatic effusions, changes in protein content and composition have been associated with both increased vascular permeability and increased protein synthesis by the synoviocytes.

The ability of the synovial membrane to reform following synovectomy has been described in the horse[40,107] and reveals that 120 days after subtotal synovectomy, there is evidence of restoration and an intimal layer was present.[107] However, the synovium was devoid of villi and there was subintimal fibrosis.

Another important property of the joint capsule is its ability to allow complete range of motion. The example of a metacarpophalangeal (fetlock) joint, illustrated in Figure 7.3, shows that synovial membrane gathers at the dorsal aspect of the joint in extension and at the palmar aspect in flexion. This property of gathering is called "redundancy."[101] Inflammation and fibrosis impede this property and result in joint stiffness. Elasticity of the joint capsule is also important because of its role in shock absorption.[85]

Articular Cartilage Structure and Function

Grossly, normal articular cartilage appears milky and opaque in the thicker regions and translucent with a slight bluish tinge in the thinner regions. The surface is not smooth. Studies using the scanning electron microscope have demonstrated undulations and irregular depressions.[28] The articular cartilage of equine joints is generally of the hyaline type, but fibrocartilage is also present at the junction of articular cartilage, synovial membrane, and periosteum (called the transition zone) and in menisci.[63]

Histologically, adult articular cartilage has been divided into four layers, and the chondrocytes have different appearances within these layers (Figure 7.4):

1. The tangential or superficial layer, containing flattened or ovoid chondrocytes and tangentially oriented collagenous fibrils
2. The intermediate or middle layer, containing larger chondrocytes that may be single or paired and randomly oriented collagenous fibrils
3. The radiate or deeper layer, containing chondrocytes arranged in vertical columns separated by collagenous fibrils that have an overall radial arrangement
4. The calcified cartilage layer, composed of mineralized cartilage and chondrocytes in various stages of degeneration

A basophilic-staining, undulating line of division between the radiate layer and the layer of calcified cartilage is termed the "tide mark" or "tide line." It delineates the elastic, nonmineralized layers of the articular cartilage from the layers of calcified cartilage that has little resilience. The extracellular matrix of the articular cartilage is a complex of collagen fibrils, proteoglycans, glycoproteins, and water (Figures 7.5 and 7.6).[63]

Collagens

Type II collagen comprises 90%–95% of the collagen in articular cartilage and forms fibrils and fibers intertwined throughout the matrix. Equine type II collagen has been characterized biochemically by cyanogen bromide-cleaved peptide profiles[111] and is secreted as a procollagen.

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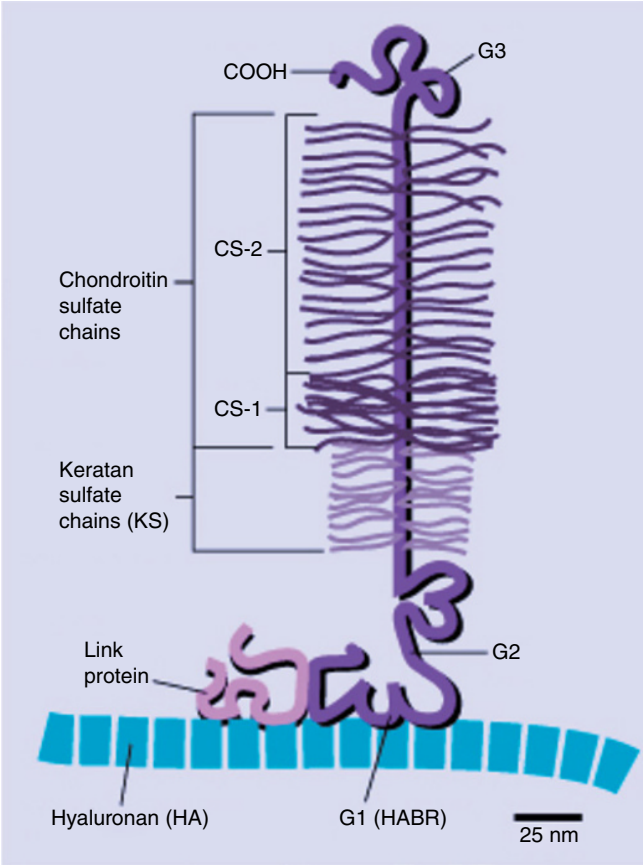


Figure 7.5. Diagram of a proteoglycan monomer formed by a protein core and glycosaminoglycan (chondroitin sulfate and keratin sulfate) side chains. Each proteoglycan core of the proteoglycan molecule is attached through a G-1 domain to an HA backbone through link protein to form aggregating proteoglycan (aggrecan). There are two other globular domains (G2 and G3).

The complete sequence of equine type II procollagen messenger RNA has been reported[88] and these authors also showed that chondrocytes harvested from juvenile horses exhibited more synthetic activity in culture with high steady-state levels of messenger RNA for type II procollagen. These authors also showed that IL-1β and tumor necrosis factor-α (TNFα) produced a dose-dependent decrease in the steady-state mRNA levels for type II collagen. Type II procollagen is expressed at very low levels in adult horses compared with younger ones. This may be relevant to naturally occurring changes of cartilage in the joints of older horses.

There are small amounts of type VI, IX, XI, XII, and XIV collagen, which help form and give stability to the type II fibril network.[76] The collagenous fibrils provide tensile strength to the articular cartilage in adult articular cartilage; this property chiefly resides in the superficial layers where the collagenous fibers are oriented parallel to the surface of the cartilage. In the middle layer of the cartilage, the collagenous fibrils are randomly arranged, and they become radially arranged in the deep layer. Tensile strength is not as critical in these deeper layers in normal cartilage, but if superficial erosion occurs, the collagen of these deeper layers is vulnerable to disruption. It has been shown that in immature cartilage the deeper layers also possess considerable tensile strength, but this is lost with maturation.[95] The tensile properties automatically change with enzymatic degradation of hydroxy-pyridinoline cross-links, which emphasizes the importance of these cross-links in providing cartilage stiffness, strength, and tension.[95]

Collagen fibers are also arranged concentrically around chondrocytes to form a capsule, called a chondron. Each chondron contains one or more chondrocytes, is invested by collagenous pericellular capsule, and is surrounded by a proteoglycan-rich territorial matrix.[82] Collagen type VI, fibronectin, and thrombospondin are present in this chondron capsule and help anchor the chondrocyte within the chondron and attach the chondron within the extracellular matrix.[77]

Proteoglycans

The proteoglycans (previously called mucopolysaccharides) are the other major solid component of the articular cartilage matrix. They occupy the spaces between the collagen fibrils. The basic proteoglycan molecule is a monomer formed by a protein core and glycosaminoglycan (GAG) side chains (Figure 7.5). Most of the proteoglycans (85%) form large aggregates by noncovalent attachment of the core protein of the proteoglycan to HA under the stabilization of a link protein (Figure 7.6). This aggregate is called aggregating proteoglycan or aggrecan.

The major GAG in adult articular cartilage are chondroitin-6-sulfate and keratan sulfate. Chondroitin-4-sulfate is an important constituent of immature articular cartilage, but its level decreases to a low percentage with cartilage maturity. The GAG consist of repeating units of disaccharides, which are important because of their polyanionic nature (carboxyl and sulfate radicals in chondroitin sulfate and sulfate radicals in keratan sulfate). The charges of the polyanionic GAG side chains both repel each other and attract a hydration shell. These properties, in turn, provide the articular cartilage with a physicochemical stiffness and affect cartilage permeability.[56] The aggrecan molecules are contained by the collagen network, and hence the proteoglycans impart compressive stiffness.[46] It should be noted that enmeshment of the proteoglycans by the collagenous framework and specific interactions between the two components are necessary for the proteoglycans to function.[7]

The average proteoglycan unit in articular cartilage has a molecular weight of 3 million Daltons and contains 100 chondroitin sulfate side chains and 100 keratan sulfate side chains.[76] More than 100 of these proteoglycan monomers attach to a HA backbone to form the proteoglycan aggregate aggrecan, which has a molecular weight of more than 200 million Daltons (Figure 7.6). The proteoglycan monomer has been the subject of many studies. There are two globular domains (G1 and G2) at the HA-associated end followed by an extended glycosaminoglycan-containing region (E_2), with a third globular domain (G3) at the other end. The N-terminal region of the aggrecan is attached to HA, and the region that contains the majority of the GAG is an extended region lying between G2 and G3.

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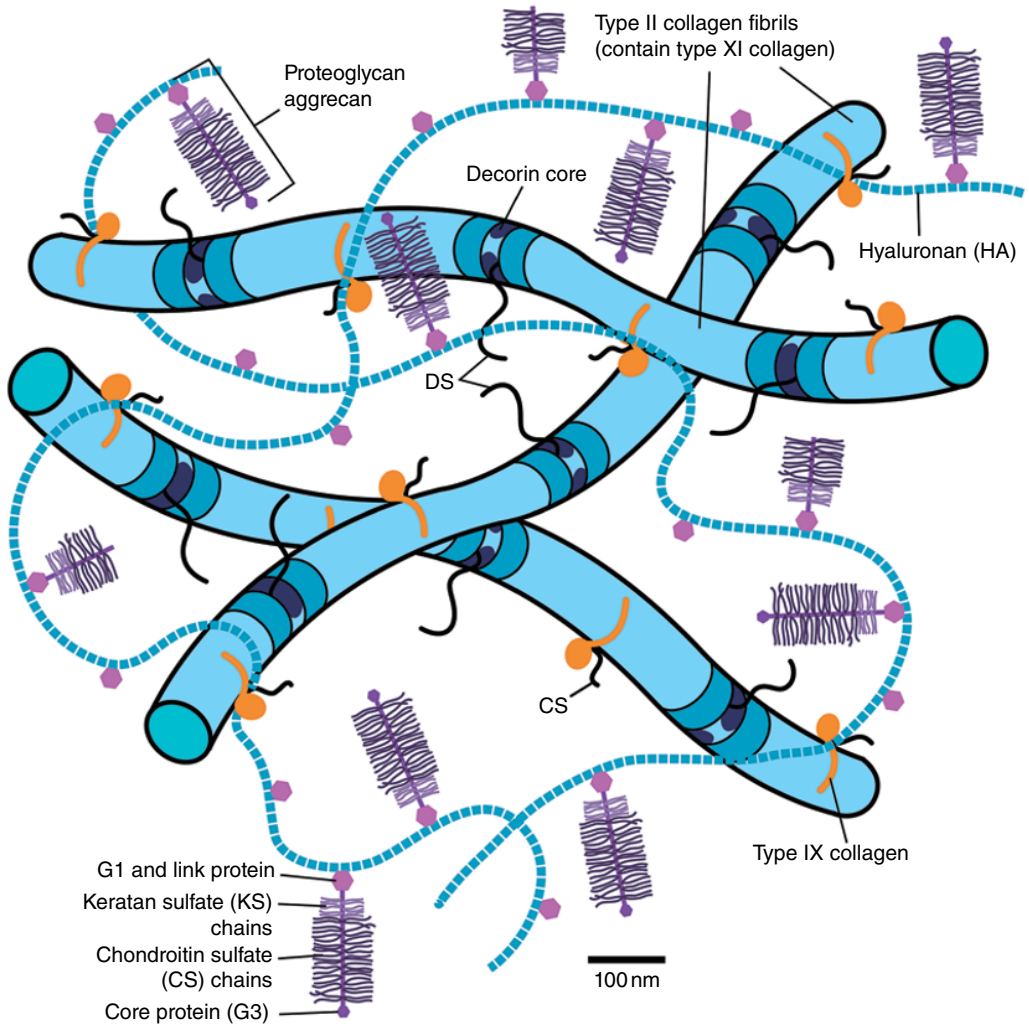


Figure 7.6. Diagram representing a portion of extracellular matrix showing organization of type II collagen fibers and multiple proteoglycan molecules that bind HA to form aggrecan entrapped within the collagen fibers. *Source*: Adapted from Poole.[81] Reproduced with permission of JAMA.

Other nonaggregating proteoglycans in articular cartilage include biglycan, decorin, and fibromodulin. Equine decorin has been characterized biochemically in the horse,[80] as have the gene sequences of biglycan and decorin.[81]

Glycoproteins

Noncollagenous, nonproteoglycan glycoproteins constitute a small but significant portion of articular cartilage.[108] There are three fractions of equine link protein: LP1, LP2, and LP3.[23,79] Other glycoproteins of cartilage include chondronectin (thought to adhere chondrocytes to type II collagen surfaces), fibronectin (adheres cells to molecules and surfaces), cartilage oligomeric matrix protein (COMP), thrombospondin, and anchorin C-II, as well as cartilage-derived growth factor.

Cartilage Nutrition and Innervation

The articular cartilage is avascular, lacking both blood and lymph vessels. The deep layers of immature cartilage are penetrated extensively by vascular buds from the ossified portion of the epiphysis, which appear to play an important role in nutrition of the cartilage from the subchondral region.[63] Immature articular cartilage is an articular–epiphyseal complex with the deeper layers constituting a growth zone. In adults, the articular cartilage is separated from the subchondral vascular spaces by an end plate of bone (the subchondral plate), and nutrition of the articular cartilage occurs by diffusion from the synovial fluid. Chondrocytes function by anaerobic glycolysis. Under physiologic loads, cartilage can be compressed to 40% of its original height. The depth to which the diffusing nutritional gradient can extend is limited and was calculated to be a maximal cartilage thickness of 6 mm.[56] Because of this limit, necrosis secondarily occurs when retained thickened cartilage, as develops in some cases of OCD, exceeds diffusion limits.

There are no nerves in articular cartilage, and the bearing surface of the joint depends on nerve endings in the joint capsule, ligaments, muscle, and subchondral bone for appreciation of pain and proprioception.

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Biomechanics

As indicated previously, articular cartilage is a tissue consisting of aggrecan that is stiff in compression, collagen that is stiff and strong in tension, and somewhat freely moving fluid carrying mobile ions (interstitial fluid). These components interact to provide the following mechanical and physical characteristics when young and healthy: (1) a permeable matrix that is stiff in compression, (2) a fibrous network capable of withstanding high tensile stresses, (3) a fluid that flows under load or deformation and aids in dissipating high stresses in the tissue, and (4) a high swelling pressure that results in a matrix swollen with water.[73] It has also been noted that an important function of aggrecan in cartilage is to retard the rate of stretch and alignment when a tensile load is suddenly applied and that this mechanism may be useful in protecting the cartilage collagen network during physiologic situations.[97]

In an unloaded joint, the opposing articular surfaces are not completely congruent.[28] However, under physiologic loading, because the cartilage is soft, deformation causes an increase of contact area (reducing tissue stress levels) and increases joint conformity (which provides additional stability). The adaptation of the shape of loaded cartilage may also help to form and retain boundary lubrication (see below). As articular cartilage directly under load is compressed, the surrounding areas are subjected to transverse tensile strains. These forces tend to redistribute fluid away from the compressed area and into the stretched regions. As mentioned previously, the major contribution to the osmotic pressure provided by the proteoglycans is derived from negatively charged sulfate and carboxyl groups of the GAG and the associated cations (ionic or Gibbs–Donnan effect).[108] Water is attracted by the high charge density, and this osmotic pressure may contribute up to 50% of the compressive stiffness of the articular cartilage. The swelling pressure is balanced by the tensile stress of the collagen framework.

Mow and colleagues have described the extracellular matrix as a cohesive porous composite.[73] Their biphasic model for articular cartilage considers the tissue as an interacting mixture of two continuums: a porous permeable elastic solid and interstitial fluid. Because of swelling pressure, the collagen network of the articular cartilage is under a tensile prestress even when unloaded. During compression, the concentration of the organic material and the charge density increase because the interstitial fluid is forced to flow from the matrix. A new equilibrium is reached when the charge density, collagen tension, and applied load are in balance (Figure 7.7). The deformation of this cartilage in association with fluid exudation is called creep. The compression time curve consists of the creep phase, controlled by fluid exudation, and the second phase, which is related to the collagen proteoglycan matrix component. During prolonged periods of stationary loading, fluid is slowly exuded and redistributed within the cartilage until an equilibrium position is reached. At this stage, the increased concentration of the fixed charge density is counterbalanced by the increased osmotic swelling pressure of the proteoglycan. When fluid motion ceases, the entire external load is borne by the solid extracellular matrix.

Intermittent pressures created by the interaction of the opposing articular surfaces pump fluid through the cartilage for nutrition and the removal of metabolic byproducts.[63] Expressed fluid from the articular cartilage resembles synovial fluid except for its low protein content and viscosity.[49] It has been shown that the concentration of fixed charges from GAG is the prime determinant of cartilage permeability.[56] Simple diffusion seems sufficient for nutrition of all but the deepest layers of articular cartilage, but joint movement probably facilitates the process.

Studies have shown that proteoglycans restrict solute movement within the tissue matrix. The proteoglycans restrict the diffusion of large, uncharged solutes but do not affect the diffusion of small, uncharged solutes. The molecular size and conformation of the solute is also an important factor. Removal of proteoglycans increases the influx of large molecules into the matrix, and this suggests that proteoglycan removal may also increase the efflux of large molecules out of the tissue matrix.[108] It has been suggested that the marked loss of aggrecan and newly synthesized proteoglycan monomer from the matrix in osteoarthritis (OA) is probably a direct result of the increased mobility of these macromolecules as the tissue matrix components continue to degrade.

It is also well accepted that mechanical forces modulate the metabolic activity of chondrocytes; clarification of the specific mechanisms of mechanical signal transduction in articular cartilage is emerging.[71] One proposed pathway is that chondrocytes may perceive changes in their mechanical environment through cellular deformation.[33] More recently it is demonstrated that the chondrocytes possess a primary cilium that is involved in mechanotransduction events related to cartilage extracellular matrix synthesis.[114] This study confirmed that primary cilia were essential organelles for cartilage mechanotransduction, as well as identified a novel role for primary cilia not previously reported in any other cell type, namely, cilia-mediated control of ATP reception. It is well established that the GAG content of habitually loaded areas of cartilage is greater than that of habitually unloaded areas. Work in sheep stifle joints has shown that different areas of articular cartilage subjected to differing mechanical stresses contain a phenotypically distinct chondrocyte population.[50] Chondrocyte phenotypes were identified by the relative biosynthesis of aggrecan, biglycan, and decorin.

Articular Cartilage Metabolism and Matrix Turnover

The chondrocytes synthesize all of the components of the cartilage matrix. At each stage of growth, development, and maturation, the relative rates of matrix synthesis and degradation are adjusted to achieve net growth, remodeling, or equilibrium. A unique interaction exists between chondrocytes and the surrounding matrix. This may be facilitated by a cilium from each chondrocyte that extends into the matrix and acts as a probe, sensing changes in the matrix composition such as a loss of proteoglycan or collagen or an increase or decrease in HA concentration. This information is relayed to the cell. Interaction between the pericellular and territorial matrix and the chondrocyte cell membrane also may include transmission of mechanical

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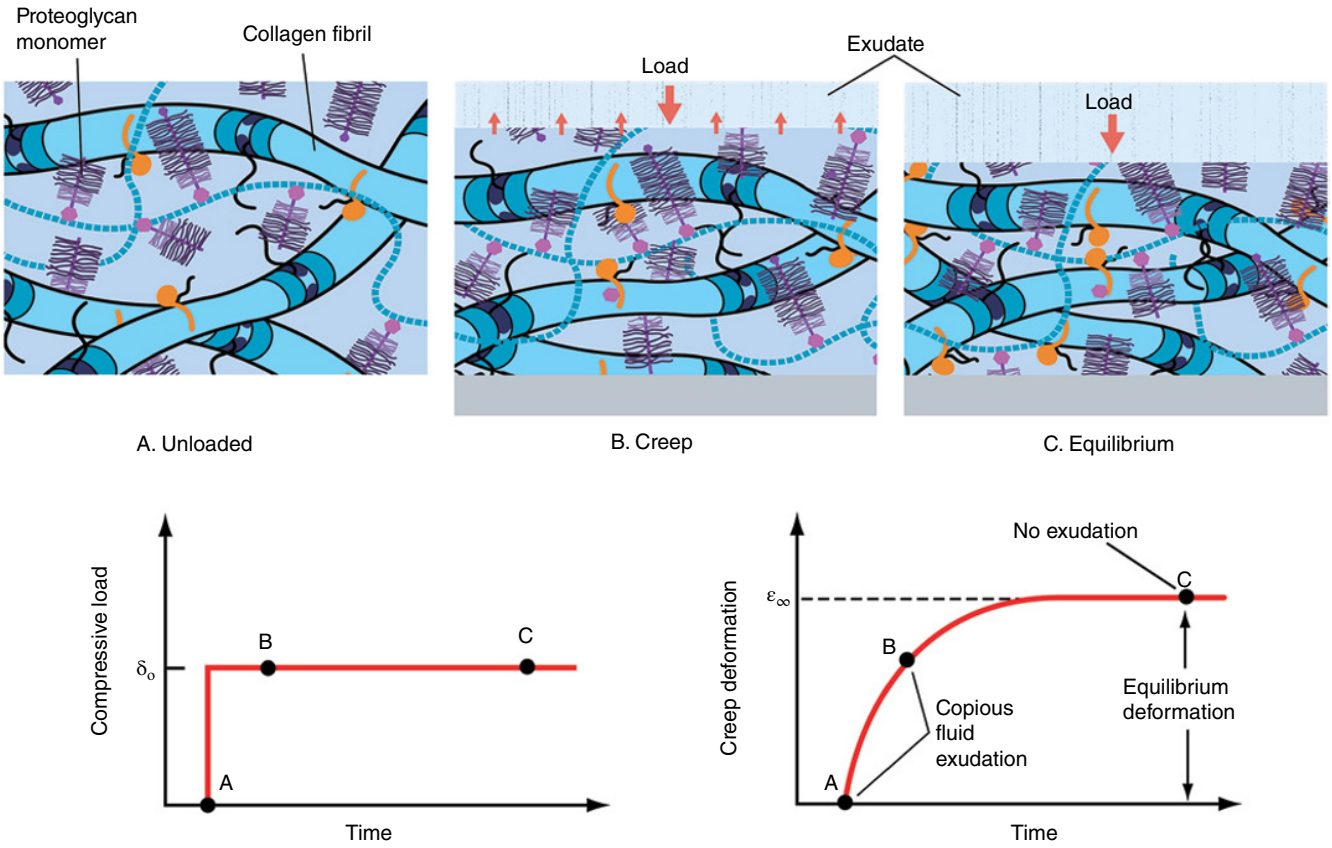


Figure 7.7. Deformation changes occurring over time during confined compression testing of articular cartilage. When a load is applied instantaneously to a confined piece of cartilage and held constant, compressive deformation increases with time (i.e. creep occurs) until an equilibrium position is obtained. The drawings at the top of the figure illustrate consolidation of the matrix under a constant load. The first phase component corresponds to the elastic spring of the biphasic model of cartilage behavior. The fluid is the second phase of the model and is exuded under load. The behavior of a nonviscoelastic material is shown by the graph on the lower left. The behavior of a viscoelastic material (articular cartilage) is shown by the graph on the lower right. The transient portion of the curve results from compression from a viscoelastic material as controlled by fluid exudation and equilibrium position by the elastic spring. *Source*: Adapted from Myers and Mow.[74] Reproduced with permission of Elsevier.

signals by changes in matrix tension or compression. Other investigators have provided support for the idea that forces perceived by chondrocytes dictate their shape and then stimulate alterations in cellular biochemistry and matrix metabolism.[36]

Collagen turnover times within normal articular cartilage have been estimated to be 120 years in the dog and 350 years in adult human articular cartilage.[108]

However, collagen breakdown is an early event in OA and can be detected with biomarkers before it is apparent morphologically (see below).[65] On the other hand, the protein core of aggrecan is commonly cleaved, particularly the portion between the G1 and G2 domains. Cleavage here leaves a large C-terminal proteoglycan fragment, which diffuses out of the tissue. The overall proteoglycan turnover time in adult rabbit and dog articular cartilage is about 300 days.

Dynamic load and the action of cytokines are thought to be involved in matrix turnover. The cytokines of principal interest at the moment are the interleukins and TNFα. These factors act on chondrocyte receptors and influence the production and activation of metalloproteinases (MMPs), PGE_2, and aggrecanase. The activity of MMPs, in turn, is inhibited by tissue inhibitors of MMPs (TIMPs 1 and 2), and it has been shown that there is a slight excess of TIMP over metalloproteinase concentration in normal articular cartilage.[18] See the section on pathobiology for further information on the action of cytokines.

Not all cytokines cause degradation. There are a number of growth factors such as insulin-like growth factor 1 (IGF-1) and various members of the transforming growth factor (TGF) superfamily, which includes bone morphogenic proteins (BMPs), that are positively involved in articular cartilage synthesis.

Lubrication of Joints

The synovial joint contains two systems that require lubrication: a soft tissue system, involving the sliding of synovial membrane on itself or other tissues, and a cartilage-on-cartilage system. Lubrication of the synovial membrane is by boundary lubrication, and HA is the important component in the synovial fluid that performs this function.[63] The HA molecules stick to the surface of the synovial membrane and allow sliding of synovial

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membrane over the opposing surface. This function is important because a major part of the frictional resistance in joint movement is in the synovial membrane and fibrous joint capsule.[55]

Considerable investigations have clarified the lubrication of articular structures in synovial joints, and the modes have been classified as fluid filled or boundary. The operative lubrication modes depend on the normal and tangential forces on the articulating tissues, the relative rate of tangential motion between these surfaces, and the time and history of both loading and motion.[73] One type of fluid-mediated lubrication is hydrostatic. At the onset of loading and typically for a prolonged duration, the interstitial fluid within cartilage becomes pressurized and rises to the surface by a weeping mechanism. In boundary lubrication, the load is supported by surface-to-surface contact, and the associated frictional properties are determined by lubricant surface molecules. This mode is thought to be important because the opposing cartilage surfaces make contact over approximately 10% of the total area, and this may be where most of the friction occurs.[70]

With increasing loading time and dissipation of hydrostatic pressure, lubricant-coated surfaces bear an increasingly high proportion of the load relative to pressurized film, and consequently the friction coefficient is dominated by this mode of lubrication. Boundary lubrication is therefore important in reducing articulation-induced shear and wear. Boundary lubrication is mediated by synovial fluid.[99] Components of the synovial fluid involved in boundary lubrication include HA,[78] proteoglycan 4 (PRG4),[101] and surface active phospholipids (SAPL).[99] Maintenance of normal lubricating functions is considered critical to the health of articular cartilage.[95]

Subchondral Bone

The subchondral plate and epiphyseal bone beneath it form an integral part of the joint structure, providing structural support to the overlying articular cartilage. The subchondral plate consists of cortical bone that varies in thickness depending on the joint. With exercise, remodeling occurs, and the amount of dense cortical bone can increase, at least in the carpus and fetlock, but there is marked variation between horses.[44]

The relationship between the mechanical properties and morphometry of the bones of horses has been explored.[119] In the third carpal bone, maximal subchondral bone stiffness occurs 10 mm palmar to the dorsal margin. There is also a significant negative correlation between subchondral stiffness and the porosity of the radial facet of the third carpal bone. Acquisition of such basic data is important in studying traumatic joint disease in the horse. Kawcak et al. have shown microdamage in the subchondral bone (including microcracks and osteocyte death) as relatively early events in exercising horses.[44]

Cytokines and growth factors similar to those in articular cartilage are also present in bone. These include IGF-1, the TGFα family (including the BMPs 2–7), platelet-derived growth factor, and fibroblast growth factors. These peptides are produced by bone cells and are present in bone matrix. IL-1 and TNFα are also present in bone. Presumably, disease in subchondral bone would expose the articular cartilage to these cytokines. More details on the relationship between articular cartilage and subchondral bone are presented in the section on pathobiology.

The innervation of the subchondral bone plate has been a subject of controversy. However, recent immunohistochemical techniques identifying substance P (a neuropeptide specifically located in sensory nerves and CNS elements) have helped study the innervation of the subchondral plate further.[76] Substance P immunoreactive neurofilaments have been identified in the subchondral bone of normal equine metacarpophalangeal joints. The nerves existed in the small cancellous spaces and diversion canals. Previous immunohistochemical studies of the subchondral plate of diseased human patellae revealed substance P fibers in the periosteum and subchondral plate of patellae affected with degenerative disease.[115] Another interesting finding in the human study was the observation of substance P fibers in erosion channels running through the subchondral plate to the deep surface of the articular cartilage, where nerve terminals were seen. Because the erosion channels were not seen in normal patellae, the presence of the substance P fibers in the erosion channels may represent a morphologic change explaining why degenerative patellae become painful.

PATHOBIOLOGY OF JOINTS AND THEIR REACTION TO INSULT AND INJURY

Traumatic Arthritis: Multiple Manifestations

In its broader sense, "traumatic arthritis" includes a diverse collection of pathologic and clinical states that develop after single or repetitive episodes of trauma and may include one or all of the following:

1. Synovitis (inflammation of the synovial membrane)
2. Capsulitis (inflammation of the fibrous joint capsule)
3. Sprain (injury of specific ligaments associated with the joint)
4. Intra-articular fractures
5. Meniscal tears (femorotibial joints)
6. OA (when degradative changes have occurred in the articular cartilage)

Any of 1 through 5 above can potentially progress to OA. To facilitate the discussion of pathobiology, it is convenient to divide articular trauma into three entities:

Type I: Traumatic synovitis and capsulitis without obvious disturbance of the cartilage or destruction of major supporting structures. This includes acute synovitis and most sprains.

Type II: Disruptive trauma with damage to the articular cartilage or complete rupture of a major supporting structure. This includes severe sprains, meniscal tears, and intra-articular fractures.

Type III: Posttraumatic OA. Equine OA may be considered as a group of disorders characterized by a common end stage in which progressive deterioration of the articular cartilage is accompanied by changes in

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the bone and soft tissues of the joint. It is inevitably the result of severe trauma to the joint or ineffective treatment of any of the predisposing conditions.

The joint is an organ and all of its tissues can be injured. Cyclic trauma to the synovial membrane and fibrous joint capsule results in synovitis and capsulitis and is a common entity in the equine athlete. Direct trauma can occur to the articular cartilage and subchondral bone, whereas inflammatory mediators resulting from synovitis can cause biochemical damage. Intra-articular and extra-articular ligaments of the joint can be injured, as can the menisci in the stifle.

The reaction in the various joint-associated tissues should not be considered in isolation, as evidenced by the example of the carpus of a racehorse. Considerable damage may be inflicted directly to the articular cartilage and bone, resulting in cartilage ulceration and intra-articular fractures of the carpus that cause varying degrees of articular cartilage loss. However, cyclic fatigue damage to the collagen network could be an important step in the pathogenesis of a more insidious osteoarthritic entity, exposing chondrocytes to deleterious physical forces and inducing injury and metabolic changes.

Primary damage to the subchondral bone other than fracture also may occur (subchondral bone disease) and lead to secondary damage to the articular cartilage, either from loss of support or secondarily from release of cytokines. Subchondral sclerosis may also lead to further physical damage to the articular cartilage because of decreased shock absorption. Acute synovitis and capsulitis is a common problem in these same joints and may also contribute to the degenerative process by the release of deleterious mediators.[63]

Physiologic Trauma Leading to Osteoarthritis

OA is a disease that results from an interaction of a number of complex mechanical and biological processes and predominantly, but not solely, affects the diarthrodial joint. As noted above, the end result is progressive deterioration of the articular cartilage. While a definitive characteristic of OA is articular cartilage destruction, we have come to recognize the accompanying changes of synovitis and shown that it can be a primary disease process leading to OA. We also know that many of the mediators involved in early deterioration of the articular cartilage are present in increased amounts (or there are decreased amounts of their inhibitors), which represents an exaggerated normal metabolic process.

Athletic Activity Potentially Leading to a Pathologic Process

It is well accepted that equine athletes carry an increased risk for development of OA. It should be noted that intact articular cartilage possesses optimal load-bearing characteristics that adjust to the level of activity. Increasing weight-bearing activity in athletes and adolescents has been shown to improve the volume and thickness of articular cartilage[41] and increase cartilage GAG content in the human knee.[94] In the healthy human athlete, a positive linear dose–response relationship apparently exists for repetitive loading activities and articular cartilage function.[60] However, studies also indicate that the dose–response curve reaches a threshold and activity beyond this threshold can result in maladjustment and injury of articular cartilage.[47]

High-impact joint loading above this threshold has been shown to decrease cartilage proteoglycan content, increase levels of degradative enzymes, and cause chondrocyte apoptosis.[1,52] If the integrity of the functional weight-bearing unit is lost, either through acute injury or chronic trauma in the high-impact athlete, a chondropenic response is initiated that can include loss of articular cartilage volume and stiffness, elevation of contact pressures, and development or progression of articular cartilage defects.[61,62] It has been proposed that concomitant histologic factors such as ligamentous instability, malalignment, meniscal injury, or deficiency can further support progression of the chondropenic cascade and without intervention, chondropenia leads to progressive deterioration of articular cartilage function and may ultimately progress to OA.

Evidence has accumulated with regard to various inflammatory mediators having an impact on matrix homeostasis of articular chondrocytes by altering their metabolism.[57] The authors of a recent paper on inflammatory factors involved in human OA cited evidence that points to the proinflammatory cytokine interleukin-1β (IL-1β) as the most important factor responsible for the catabolism in OA.[57] However, the authors pointed out that new members of the IL-1 superfamily have recently been identified (ILF-5, ILF-10), some of which have been suggested to be of interest for arthritic disease, and that other proinflammatory cytokines such as tumor necrosis factor (TNF)-α-6 and other interleukins can be contributing factors. They also noted that the exact role and importance of each within the OA process are not yet clearly identified. In addition to cytokines, other inflammatory mediators that may play a major role in the OA process include nitric oxide (NO), eicosanoids (prostaglandins and leukotrienes), and a newly identified cell membrane receptor family, the protease-activated receptors (PARs).

Discussion of mediators for equine traumatic joint disease and OA in this chapter focus on mediators that have been shown to be increased in equine joint disease and/or their inhibition can ameliorate progression of OA.

When considering a traumatically injured joint, two basic pathobiologic processes should be considered: inflammation of the synovial membrane and fibrous joint capsule (synovitis and capsulitis) and physical or biochemical damage to the articular cartilage and bone. Acute synovitis and capsulitis can cause significant clinical compromise and may also contribute to the degenerative process by releasing enzymes, inflammatory mediators, and cytokines.[64] These processes are outlined in Figures 7.8 and 7.9.

Synovitis and Capsulitis

Treatment of synovitis and capsulitis, particularly the acute form, is indicated to (1) alleviate the immediate compromising effects of inflammation, including pain and reduced function; (2) prevent the development of

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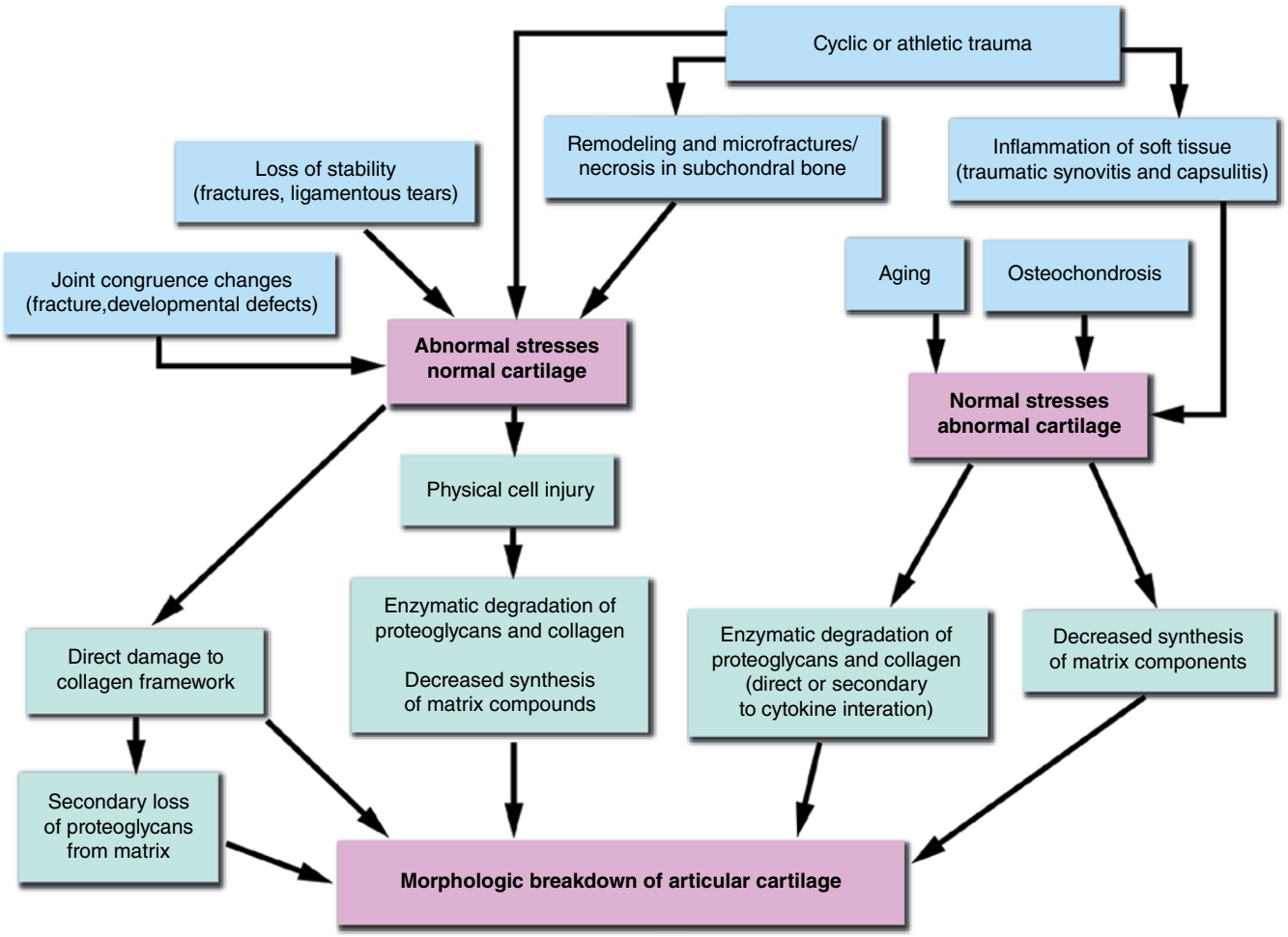


Figure 7.8. Possible pathways for degradation of articular cartilage secondary to joint trauma. *Source*: Redrawn from figure 4, McIlwraith.[64] Reproduced with permission of American Association of Equine Practitioners.

permanent fibrosis in the joint capsule, which in turn causes decreased motion and compromised shock absorption capabilities in that joint; and (3) prevent or minimize the development of OA.

Synovitis and capsulitis as primary entities in athletic horses are presumed to be associated with repeated trauma.[61] Severe injury to the fibrous joint capsule also can cause instability. The synovial membrane itself is mechanically weak and has no known biomechanical role, but it is recognized that synovial injury may have pathophysiologic consequences in the joint.[24] Some injuries may affect diffusion across the synovial membrane, and others have a primary effect on the metabolism of the chondrocyte.[24] Mechanically damaged synoviocytes may release degradative enzymes and cytokines, which alter the intra-articular environment and possibly affect articular cartilage. It has also been suggested that high intra-articular pressures in injured joints associated with effusion could be sufficient to impair the flow of blood through the synovial capillaries and potentially lead to reperfusion injury.[48] Flexion of a joint with sufficient synovial effusion could raise the intra-articular pressure to levels of impaired blood flow through the synovial capillaries.

In addition to direct injury that may occur to the synovial membrane, the reaction of the synovial membrane to articular cartilage damage or other mechanical destruction of intra-articular tissues is well recognized. The presence of cartilaginous wear particles increases the cellular production of prostaglandin E_2, cytokines, and the neutral metalloproteinases (collagenase, stromelysin, and gelatinase).[24] It has also been shown that the proteoglycans released into synovial fluid cause synovitis.[6]

The ability of synovial membrane inflammation alone in the absence of trauma and/or instability to damage articular cartilage has previously been demonstrated in the horse.[66]

The Importance of Synovitis

Synovitis (and capsulitis) is important to the horse because it produces pain, the increased synovial effusion is uncomfortable and it eliminates the normal small negative pressure within the joint (therefore promoting micro-instability), and it produces products that are deleterious to joint health as a whole and articular cartilage in particular. The mediators currently considered to be

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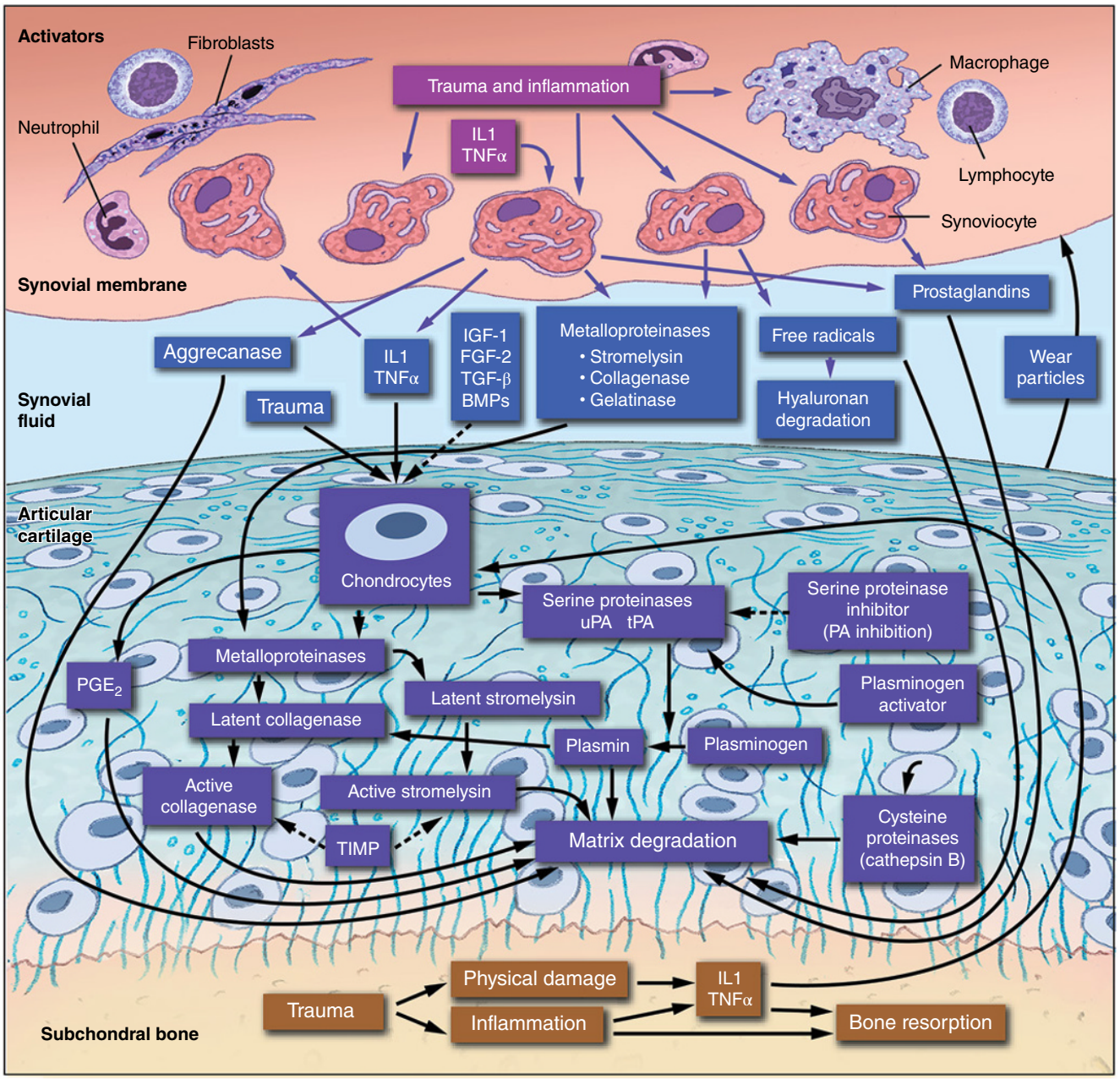


Figure 7.9. Factors involved in degradation of the articular matrix: interleukin-1, tumor necrosis factor-α (TNFα), fibroblast growth factor-2 (FGF-2), PLA2, urokinase plasminogen activator (uPA), tissue plasminogen activator (tPA), plasminogen activator (PA), insulin-like growth factor-1 (IGF-1), transforming growth factor-β (TGF-β), and bone morphogenic protein (BMP). *Source*: Redrawn from figure 5, McIlwraith.[64] Reproduced with permission of American Association of Equine Practitioners.

of significance in equine joint disease include metalloproteinases and aggrecanases, prostaglandins, free radicals, cytokines, interleukin-1 (IL-1), and possibly TNFα (Figure 7.9).

Matrix Metalloproteinases and Aggrecanases

Matrix metalloproteinases (MMPs) may be subdivided into collagenases, gelatinases, and stromelysins. Three collagenases have been identified, and they are interstitial or tissue collagenase (also called MMP-1), PMN collagenase (or MMP-8), and collagenase 3 (also known as MMP-13). Based on evidence in both human and equine studies, it appears that the primary collagenase involved in the degradation of type II collagen of articular cartilage is collagenase 3.[10,70] Caron et al. found that MMP-13 is produced by equine chondrocytes and that MMP-13 expression was significantly stimulated by rhIL-1.[10]

Consideration of the breakdown of aggrecan initially focused on stromelysin (also called proteoglycanase or MMP-3), but now recognizes an initially undefined enzyme called "aggrecanase" (now known as aggrecanase-1 and aggrecanase-2 or ADAMTS-4 and ADAMTS-11). Stromelysin has a wide variety of substrates, including

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proteoglycans (aggrecan, decorin, fibromodulin, link protein) and type IV, V, VII, IX, and XI collagen.[25] It also cleaves type II collagen in nonhelical sites.[118] *In vitro* study of IL-1-induced cartilage degeneration has revealed evidence of collagen degradation that could be attributed to stromelysin in addition to that attributed to collagenase.[21] Molecular cloning and cartilage gene expression of equine stromelysin 1 (MMP-3) has been described by Balkman and Nixon.[2] Stromelysin cleaves the protein backbone of cartilage proteoglycans at the asparagine 341–phenylalanine 342 bond.[57] Cleavage due to aggrecanase, on the other hand, occurs at the GLU373-ALA373 site.[68] Based on preliminary work in the horse using markers that differentiate these two cleavage sites, it appears that aggrecanase is the principal enzyme degrading proteoglycan in equine joint disease.[51]

Equine MMPs 2 and 9 are two gelatinases that have been characterized in the horse.[11,12] It is known that the one-fourth and three-fourth fragments generated by cleavage by fibrin or collagens by collagenases can unwind and are then susceptible to further cleavage by MMP-2 and MMP-9. MMPs 2 and 9 are produced by a variety of equine cell types, and these enzymes have been demonstrated in elevated levels in synovial fluids from horses with joint disease.[11,12]

The metalloproteinases are inhibited by two tissue inhibitors of metalloproteinase and these are known commonly as TIMP (TIMP-1 and TIMP-2).[75,102] TIMP is found in many connective tissues and may be the most important inhibitor found in articular cartilage. This current belief is that the balance between MMPs and TIMP is important to prevent progression of articular cartilage degradation.

In summary, MMPs are considered to play a major role in articular cartilage degradation. They are secreted as latent proenzymes and activated extracellularly by serine proteinases. Plasmin may activate stromelysin, which in turn is an important activator of collagenase. Upregulation of these enzymes in synovial membrane and articular cartilage samples from traumatic equine joint disease has been demonstrated.[110] It is also known that the production of MMPs is upregulated by IL-1.

Prostaglandins

Prostaglandins (primarily E group) are produced in inflamed joints and can cause a decrease in the proteoglycan content of the cartilage matrix.[104] The presence of prostaglandin E_2 in synovial fluid from inflamed joints has been demonstrated in the horse.[105] In the author's laboratory, PGE_2 measurements are used as an objective index of the level of synovitis.[26,43] Actions of PGE_2 in joints include vasodilation, enhancement of pain perception, proteoglycan depletion from cartilage (by both degradation and inhibition of synthesis), bone demineralization, and promotion of plasminogen activator secretion. PGE_2 is released from chondrocytes on stimulation of these cells by IL-1 and TNFα.

Oxygen-Derived Free Radicals

Oxygen-derived free radicals, including superoxide anion, hydroxyl radicals, and hydrogen peroxide, may be released from injured joint tissues. Studies have demonstrated cleavage of HA by free radicals.[32,116] There is also evidence that superoxide can degrade the alpha chains of collagen based on the finding that superoxide treatment inhibits gelatin.[31] Proteoglycans may be cleaved by free radicals.[31] Increased free radicals in the synovial fluid of cases of equine joint disease have been demonstrated more recently.[20]

Nitric oxide has been recognized as an important physiologic mediator. It combines avidly with superoxide anion, and although this was originally thought to provide a protective function, it has been suggested that this reaction can generate further destructive species, including peroxynitrite anion and hydroxyl radicals.[81] A recent review has noted that the specific effect of a radical on a given cell function very much depends on experimental context. Specifically, the simultaneous presence of superoxide, which leads to the formation of peroxynitrite (due to interaction of NO and supraoxide), a stronger and highly reactive radical, is likely to lead to cell and tissue damage.[53] However, NO has certain protective effects in cartilage and other tissues, and inhibition of NO *in vitro* or *in vivo* can potentially lead to undesired exacerbation of inflammation and tissue destruction under certain conditions. Selective scavengers of peroxynitrite must be more promising candidates for the treatment of OA than specific inhibition of NO. The role of nitric oxide in joint disease needs, and is receiving, further attention.

Cytokines and Articular Cartilage Degradation

Cytokines are soluble peptides produced by one cell affecting the activity of other cell types. Studies of cytokines in joint tissues suggest that IL-1 and TNFα modulate the synthesis of metalloproteinases by both chondrocytes and synovial cells[16,17,117] and are therefore important mediators in joint disease. Because IL-1 and TNFα may be produced by synovial cells,[17] they may therefore be of importance in the deleterious effects of synovitis on articular cartilage. It is thought that the normal turnover of the extracellular matrix of the articular cartilage is regulated by the chondrocytes under the control and influence of cytokines and mechanical stimuli. Articular cartilage degradation in association with disease represents an exacerbation of these normal processes. It is widely accepted that the cytokine IL-1 induces proteoglycan depletion in articular cartilage by either increasing the rate of degradation or decreasing synthesis in association with the release of proteinases and prostaglandins from chondrocytes (Figure 7.10). IL-1 produces its effects by binding with an IL-1 receptor on the cell. The presence of IL-1 in equine osteoarthritic joints was first reported by Morris et al.[72] An equine IL-1 containing extract was produced by May in 1990.[59]

The significant role of equine IL-1 has been further consolidated, starting with the cDNA sequences for IL-1α and IL-1β being constructed.[34,35] After generation of these DNA sequences, the IL-1α and IL-1β recombinant proteins were purified. Prior to that, only human recombinant IL-1 protein was available. Using equine articular cartilage explants, significant proteoglycan release was induced by both rEq-IL-1α and rEq-IL-1β at concentrations greater than or equal to 0.01 ng/mL with 38%–76% and 88%–90% of total GAG released by 4 and 6 days, respectively.[42,104] Significant inhibition of

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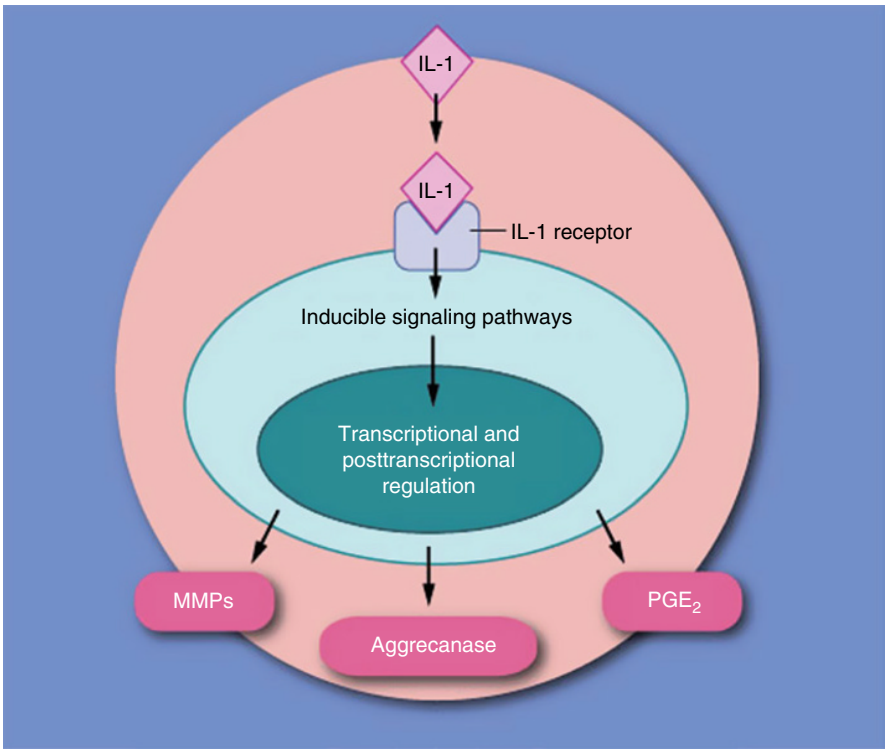


Figure 7.10. Diagram of IL-1 activation of MMPs, aggrecanase, and PGE_2 release acting through IL-1 receptors on the cell membrane. *Source*: Redrawn from figure 6, McIlwraith.[64] Reproduced with permission of American Association of Equine Practitioners.

proteoglycan synthesis (42%–64%) was observed at IL-1 concentrations greater than or equal to 0.01 ng/mL at 2 and 4 days. Increased PGE_2 concentrations were observed at IL-1 concentrations greater than or equal to 1.0 ng/mL at 2 and 4 days. This work showed that the much lower concentrations of equine IL-1 could cause these effects compared with previously reported studies using human recombinant IL-1.[104]

The IL-1 system consists of the two agonist members IL-1α and IL-1β, and these evoke signal production in response to binding IL-1R transmembrane receptors to induce downstream effects (Figure 7.10). The significant role of IL-1 in the pathogenesis of cartilage degradation in the horse was best demonstrated by the work of Frisbie et al.[27] with interleukin-1 receptor antagonist (IL-1ra) using gene therapy. This study demonstrated that if IL-1 can be inhibited, articular cartilage degradation in experimental OA can essentially be stopped.

The role of TNFα in equine OA is less certain. Billinghurst et al. first demonstrated induction of intra-articular TNF during acute inflammatory responses in equine arthritis.[5] It appears that IL-1 is the principal cytokine responsible for articular cartilage degradation and TNFα contributes more to clinical morbidity and pain. IL-1 and TNFα have been demonstrated using RT-PCR in the synovial membrane of inflamed equine joints,[110] and increased serum concentrations of soluble TNF receptors (S TNF-r) have been detected in human patients with RA and OA in comparison with healthy controls.[14] A mini review of the role of cytokines as inflammatory mediators in OA concluded that it is generally accepted that IL-1 is the pivotal cytokine in early and late stages of OA, while TNFα is involved primarily in the onset of arthritis.[29] Both receptors of TNF have been identified in synovial tissue, with greater numbers seen in joints affected by RA in comparison to OA.[19]

The Pathways to Morphologic Breakdown of Articular Cartilage: The Critical Manifestation of OA

Trauma can cause an immediate physical defect or initiate a degradative process by direct damage to chondrocytes, causing release of enzymes as well as cytokine-initiated release of metalloproteinases and PGE_2 from chondrocytes in response to IL-1. As outlined in Figure 7.8, any instability in a joint can lead to damage of normal cartilage. On the other hand, cartilage compromised by loss of GAG or collagen is vulnerable to normal forces.

As has been previously discussed, as cartilage is compressed, the collapse of the collagen network is countered by the resistance offered by the proteoglycan gel. The precise nature of the forces exerted through the network is complex and related to both mechanical entrapment and chemical bonding effects between the fibers and the proteoglycans.[7] The same author has addressed the general problem of abnormal softening in articular cartilage with simultaneous micromechanical testing, interference-like microscopy, and transmission electron microscopy. A model has been developed in which this abnormal softening in articular cartilage is related to the presence of collagen fibers strongly aligned in a

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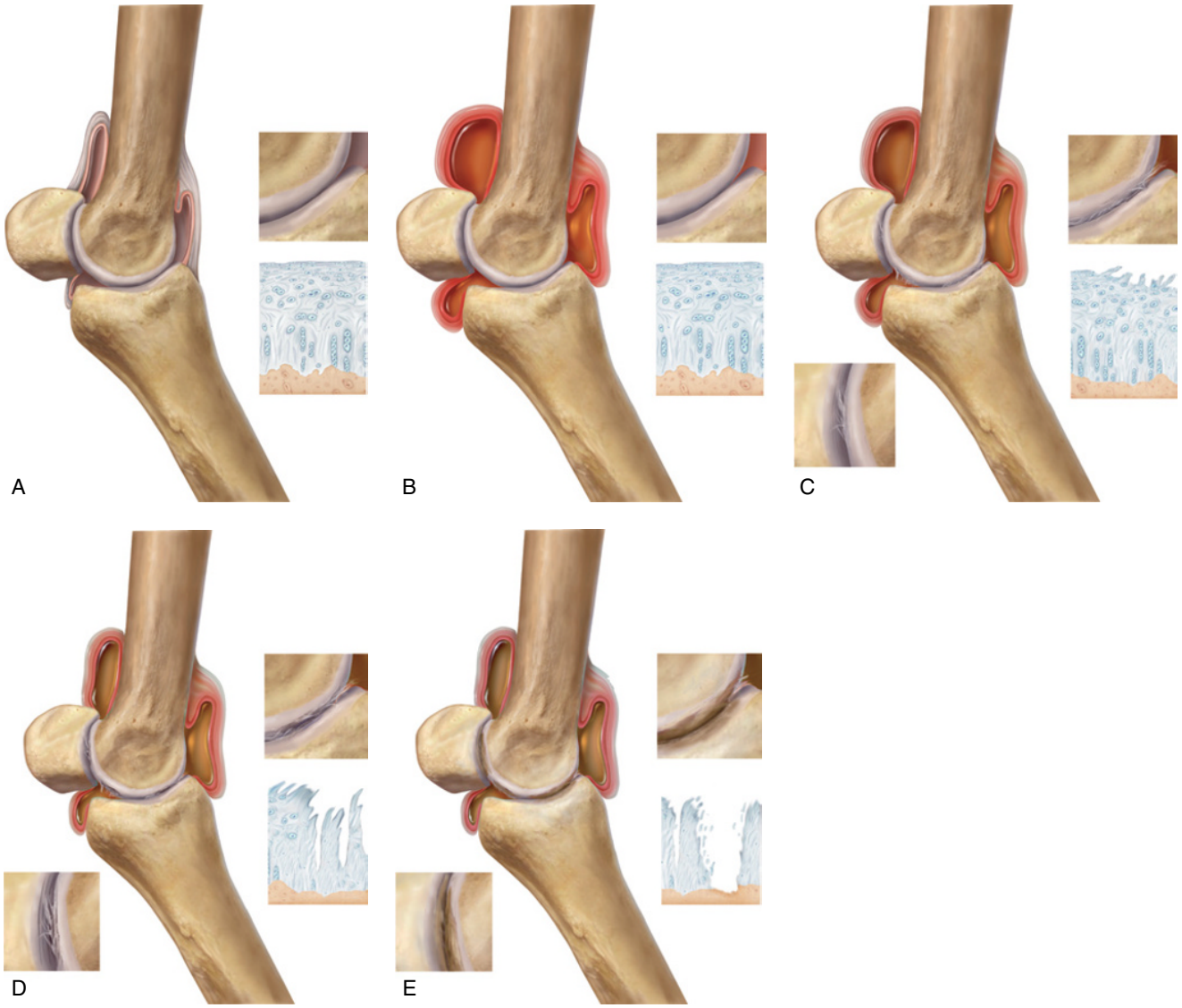


Figure 7.11. Diagrammatic series of development of osteoarthritis in a fetlock joint associated with cyclic trauma and traumatic synovitis and capsulitis. (A) Diagram of a normal joint, as well as macroscopic and microscopic views of articular cartilage. (B) Stage 1 acute synovitis without morphologic change in the articular cartilage. (C) Stage 2 with synovitis persisting and early development of superficial fibrillation in the articular cartilage. (D) Stage 3 synovitis and capsulitis becoming chronic and articular cartilage fibrillation down into the deep zone of articular cartilage. (E) Stage 4 development of full-thickness erosions in the articular cartilage that are visible macroscopically and microscopically. Joint capsule change is chronic with fibrosis, but some degree of active synovitis is still present.

radial direction and this structure, having lost its three-dimensional character, would have limited ability to contain the swelling proteoglycan aggregates remaining in the tissue and would therefore reflect a state of softening in articular cartilage.[7]

The most common reason for degradation of articular cartilage and the development of OA is generally synovitis and capsulitis. There is a progression, beginning with acute synovitis, when biochemical change can take place in the articular cartilage, but there is no morphologic change. With critical loss of matrical components, however, morphologic damage ensues, going through a cycle of chondromalacia with softening of the cartilage and swelling due to absorption of water, superficial fibrillation, and fibrillation down to the middle zone of cartilage, followed by fibrillation progressing through the deeper layer of cartilage, which then allows for loss of articular cartilage components and full-thickness erosion. Figure 7.11 illustrates four stages of OA in which the synovitis and capsulitis become more chronic. Close examination of articular cartilage shows change, and the histologic equivalent of these macroscopic lesions is reflected as well.

Articular Cartilage Repair

Healing refers to restoration of the structural integrity and function of the tissue after injury or disease, but repair usually has a more restricted meaning.[8] Repair refers to the replacement of damaged or lost cells and matrix with new cells and matrix, a process that does not necessarily restore the original structure or function

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of a tissue. Regeneration may be considered a special form of repair in which the cells replace lost or damaged tissue with a tissue identical to the original. It has been suggested that with the exception of bone fractures, most injuries and diseases of the musculoskeletal tissues do not stimulate regeneration of the original tissue.[8]

The limited potential of articular cartilage for regeneration and healing has been appreciated for more than two centuries. In 1743, Hunter stated, "From Hippocrates to the present age, it is universally allowed that ulcerated cartilage is a troublesome thing and that when once destroyed it is not repaired."[37] There are a limited response of cartilage to tissue damage and an inability of natural repair responses from adjacent tissues to produce tissue with the morphologic, biochemical, and biomechanical properties of articular cartilage.

The major limiting factor in the successful rehabilitation of any joint after injury or disease is the failure of osteochondral defects to heal.[65] Three mechanisms have been recognized as possible contributors to articular cartilage repair. Intrinsic repair (from within the cartilage) relies on the limited mitotic capability of the chondrocyte and a somewhat ineffective increase in collagen and proteoglycan production. Extrinsic repair comes from mesenchymal elements from the subchondral bone participating in the formation of new connective tissue that may undergo some metaplastic change to form cartilage elements. The third phenomenon, known as matrix flow, may contribute to equine articular cartilage repair by forming lips of cartilage from the perimeter of the lesion that migrate toward the center of the defect.[38,39]

The depth of the injury (full or partial thickness), size of defect, location and relation to weight-bearing or non-weight-bearing areas, and age of the animal influence the repair and remodeling of an injured joint surface.[9,13,62]

With a partial-thickness defect, some repair occurs with increased GAG synthesis and increased collagen synthesis.[62] However, the repair process is never completely effective. In humans, complete repair of chondromalacia of the patella has been reported to occur if matrical depletion and surface breakdown are minimal.[4] However, more recent work with arthroscopic debridement of partial-thickness defects in humans questions any actual regeneration.[98] In addition, superficial defects are not necessarily progressive and do not necessarily compromise joint function.

With full-thickness defects, the response from the adjacent articular cartilage varies little from that after superficial lesions and provides only the limited repair necessary to replace dead cells and damaged matrix at the margins of the wound. These defects heal by ingrowth of subchondral fibrous tissue that may or may not undergo metaplasia to fibrocartilage.[13,30,39,54,113]

Subchondral bone defects either heal with bone that grows up into the defect or fill in with fibrocartilaginous ingrowth.[113] Duplication of the tide mark in the calcified cartilage layer is rare, and adherence of the repair tissue to surrounding noninjured cartilage is often incomplete.[30,54]

A number of equine studies demonstrate that the size and location of articular defects significantly affect the degree of healing achieved. Convery et al. first reported that large defects were less likely to heal.[13] A more recent study distinguished between large (15 mm^2) and small (5 mm^2) full-thickness lesions in weight-bearing and non-weight-bearing areas of the antebrachiocarpal (radiocarpal), middle carpal (intercarpal), and femoropatellar joints.[39] At 1 month, small defects were filled with poorly organized fibrovascular repair tissue; by 4 months, repair was limited to an increase in the amount of organization of this fibrous tissue, and by 5 months, small radiocarpal and femoropatellar lesions were hardly detectable because of combinations of matrix flow and extrinsic repair mechanisms. Large lesions showed good initial repair, but at 5 months, perilesional and intralesional subchondral clefts developed.

The repair tissue that forms after full-thickness injury to hyaline cartilage or as a natural repair process in joints with OA is primarily composed of type I rather than type II collagen, at least at 4 months.[9,13] Identification of type II collagen is the critical biochemical factor distinguishing hyaline cartilage from repaired fibrous tissue and fibrocartilage. It is thought that the presence of an abnormal subchondral bone plate and the absence of a tide mark reforming may create a stiffness gradient and that shear stresses of the junction of the repair tissue and underlying bone develop. The propagation of such shear stresses would lead to the degradation of repair fibrocartilage and exposure of the bone. This mechanical failure has been observed experimentally and clinically in the horse.[39,67]

In a study looking at the long-term effectiveness of sternal cartilage grafting, the repair tissue in the non-grafted defects at 12 months consisted of fibrocartilaginous tissue with fibrous tissue in the surface layers, as was seen in control defects at 4 months. However, on biochemical analysis, the repair tissue of the non-grafted defects had a mean type II collagen percentage of 79%, compared with being non-detectable at 4 months.[112,113] On the other hand, the GAG content expressed as milligrams of total hexosamine per gram of dried tissue was 20.6 ± 1.85 mg/g, compared with 26.4 ± 3.1 mg/g at 4 months and 41.8 ± 4.3 mg/g DW in normal equine articular cartilage.[112,113]

The fibrocartilaginous repair seen in normal full-thickness defects is therefore biomechanically unsuitable as a replacement-bearing surface and has been shown to undergo mechanical failure with use. The lack of durability may be related to faulty biochemical composition of the old matrix and incomplete remodeling of the interface between old and repaired cartilage or to increased stress in the regenerated cartilage because of abnormal remodeling of the subchondral bone plate and calcified cartilage layer. Although recent work implies that it may be possible to reconstitute the normal collagen type in equine articular cartilage,[111] clearly there is continued deterioration of GAG content, and these are important components in the overall composition of the cartilage matrix.

The presence of a cartilage defect may not represent clinical compromise. In the equine carpus, loss of up to 30% of articular surface of an individual bone may not compromise the successful return of a horse to racing.[67] However, loss of 50% of the articular surface or severe loss of subchondral bone leads to a significantly worse prognosis.

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The inadequate healing response may not necessarily apply to immature animals or to non-weight-bearing defects. An example is the young horse after surgery for osteochondritis dissecans (OCD) that shows impressive or at least functional healing responses. This may be related to increased chondrocytic capacity for mitosis and matrix synthesis and the presence of intracartilaginous vascularity. Complete restoration of the ultrastructure and surface configuration in a hinge-like gliding joint surface such as the femoropatellar joint may be unnecessary for clinical soundness, compared with the more severe loading on an osteochondral defect located on the weight-bearing portion of the medial condyle of the femur or the midcarpal joint.

It has been suggested that increasing age may affect the response of cartilage to injury in humans because the ability of the chondrocytes to synthesize and assemble matrix micromolecules could decline with age.[8] Buckwalter cites a study of transplanted chondrocytes, suggesting that older chondrocytes produce a more poorly organized matrix than do younger chondrocytes,[8] and other studies demonstrate that the proteoglycan synthesized by the chondrocytes changes with age.[3,84] Much research is continuing to be done to develop better methods of cartilage repair, and these have been reviewed elsewhere.[64]

PRIMARY DISEASE OF SUBCHONDRAL BONE

In addition to synovial-mediated degradation of articular cartilage and direct mechanical damage, the subchondral bone can play a primary role in disease development (Figure 7.12).[78]

When considering possible pathways for mechanical destruction of articular cartilage in human OA, it has been suggested that early subchondral bone sclerosis causes a reduction in the joint's shock-absorbing ability and therefore places cartilage at risk of shear-induced tensile failure of cartilage cross-links, particularly under repetitive impulsive loading conditions.[85] Work in the author's laboratory[44] has demonstrated that when horses are subjected to athletic exercise on the treadmill, microdamage in the subchondral bone can develop early. On postmortem examination of racehorse joints (euthanized for catastrophic injuries in another limb), the range of microdamage includes not only microfractures but also primary osteocyte death. It is thought that not only is the mechanical support of the articular cartilage lost when subchondral bone microdamage progresses to macrodamage but that cytokine release from the bone also can potentially influence that state of the articular cartilage.[44,45,78] Figure 7.12 illustrates a specimen from a horse euthanized because of catastrophic injury in the other limb. An incidental finding at postmortem was the presence of subchondral bone necrosis, with a peripheral area of sclerosis and intact cartilage in the distal palmar area of the metacarpus. Figure 7.13 shows microdamage that can occur quite early in association with exercise.

Gross examination of MCP/MTP joints from racehorses revealed defects on the condylar surface that ranged from cartilage fibrillation and erosion to focal

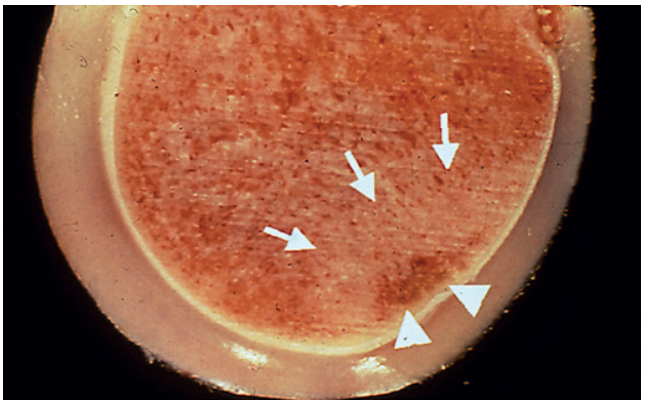


Figure 7.12. Postmortem sample of a distal metacarpus from the leg opposite to that suffering a catastrophic injury in a racehorse. Although there is intact articular cartilage, subchondral bone necrosis (arrowheads) and sclerosis (arrows) can be seen. *Source*: Norrdin et al.[78], figure 10. Reproduced with permission of Elsevier.

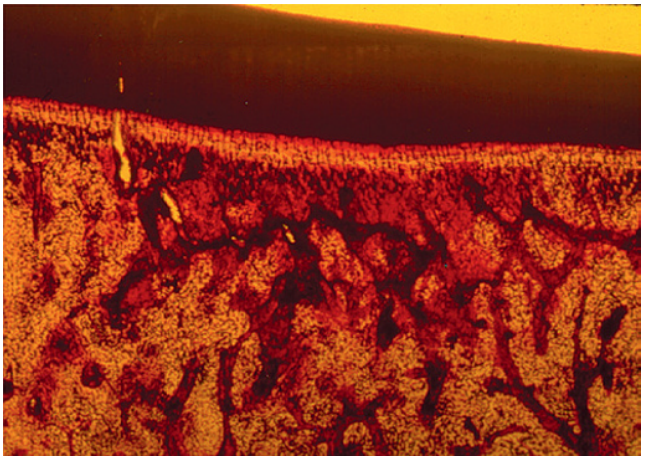


Figure 7.13. Histologic view of a section of articular cartilage and subchondral bone bulk-stained with basic fuchsin depicting microdamage with microcrack formation in the subchondral bone. *Source*: Kawcak CE. In McIlwraith.[64] Reproduced with permission of American Association of Equine Practitioners.

cartilage indentation and cavitation in subchondral bone. These lesions represented a spectrum of mechanically induced arthrosis in which microdamage is thought to play a role.[78] Lesions in the subchondral bone ranged from thickening of subchondral bone and underlying trabeculae, advancing sclerosis with increasing amounts of osteocyte necrosis, vascular channels with plugs of matrix debris, and osteoclastic remodeling. Apparent fragmentation lines in the subchondral bone suggested increased matrix fragility. Trabecular microfractures developed at a depth of a few millimeters, with increased vascularity with hemorrhage, fibrin, and fibroplasia seen in the marrow spaces at the more advanced stage. The articular cartilage in most of these instances was variously indented but remained largely viable, with degeneration and erosion limited to the superficial layers. Focally, breaks in the calcified layer appeared to lead to collagen and cartilage infolding. In metacarpal

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condyles from experimental horses run on a treadmill,[45] change in the metacarpal condyles was milder. The significance of osteochondral injury in joint disease has also been well reviewed by Riggs.[93]

In addition to its role in contributing to OA, the presence of subchondral bone injury contributing to complete failure of the subchondral bone and fractures is important.[58] "Impact fracture" is a term that has been used recently to describe a pathologic fracture that shows up as an area of lucency in the bone.[15] Pathologic change in the distal condyles of the third metacarpal and third metatarsal bones of the horse as a predisposing factor for condylar fractures was first investigated in detail by Riggs et al.[89,91,92] Recognition of this change potentially leading to fractures has led to investigations of imaging techniques to diagnose this at an early stage.[22]

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BONE INJURIES AND DISEASE

Chris Kawcak and Gary M. Baxter

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THE IMMATURE SKELETON

Bone growth occurs by both longitudinal (endochondral) and circumferential (membranous) growth. Membranous bone formation is responsible for increases in long bone width and formation of flat bones and is independent of a cartilaginous template. Longitudinal bone growth results from a series of events occurring at highly specialized regions at one or both ends of the bone. These regions are referred to as the physes, growth plates, or, more correctly, the metaphyseal growth plates.[31] The process that occurs at the growth plate, termed endochondral ossification, is characterized by rapidly differentiating and maturing cartilage cells and the replacement of cartilage by bone. There are two types of growth plates: discoid and spherical. The discoid growth plates are seen at the ends of long bones. Some bones have a physis at each end of the bone, whereas others, such as the third metacarpal/metatarsal bone and the proximal and middle phalanges, have only one distinguishable physis. A discoid physis is located between the metaphysis (the flared end of the bone that contains spongy bone) and the epiphysis. An apophysis (which is a type of discoid physis) is an epiphysis that is subject to tensile rather than compressive forces, such as at the olecranon process, calcaneal tuber, and tibial tuberosity. The growth plate of an apophysis contains greater amounts of fibrocartilage than a true discoid physis, which is an adaptation to withstand tensile forces.[85] Spherical physes are also located in the small cuboidal bones of the foal's carpus and tarsus. These growth plates develop into bones by centrifugal expansion around a central cartilage core. They begin to ossify in the center and gradually assume the contours of the bone of an adult as bone development reaches the margins of the cartilage model. (The reader is referred to Chapter 10 for further information on the morphology and physiology of the physis.)

Effect of Trauma on the Physis

When an excessive force is applied to a joint and its nearby physis, an epiphyseal or physeal injury is likely to occur. Similar to other injuries in adults, chronic repetitive trauma, such as occurs in young human athletes, can also result in damage to the physis. This is because the cartilaginous growth plate is weaker than the surrounding bone, ligamentous structures, and joint capsule. Physeal and epiphyseal injuries account for approximately 15%–18% of all fractures in children and also occur commonly in foals. Injuries that would normally produce a ruptured ligament or joint dislocation in an adult may produce traumatic separation of the physis in a young animal. For example, trauma to the fetlock region usually causes fetlock luxations in adult horses and distal metacarpal/metatarsal physeal fractures in foals. Joint luxations are rare in young horses. (The reader is referred to Chapter 10 for further information on physeal fractures in horses.) Non-fracture injuries are not uncommon in foals. Chronic angular limb deformity (ALD) and the resultant overstress of both local and distant physes in the limb can result in growth abnormalities and permanent deformity. More subtle, chronic stresses to the physes, as is common in juvenile human athletes, are not recognized in horses. However, the size and rapid growth of horses seem to make them predisposed to such problems.

Developmental Orthopedic Diseases

Developmental orthopedic disease (DOD) complex is a group of abnormalities that occurs in foals and young growing horses.[131] Other terms that are often used interchangeably with DOD include osteodystrophy or osteochondrosis although this is considered erroneous. Most of the diseases included in the DOD complex can be attributed to alterations in bone growth or development (endochondral ossification) either at the metaphyseal or epiphyseal growth plates. These include physitis, ALDs, OCD, subchondral cystic lesions (SCLs), cuboidal bone collapse or incomplete ossification, juvenile arthritis, cervical vertebral malformations (CVM), and flexural deformities.[48,131] The more specific term "juvenile osteochondral conditions" has been proposed to describe those DOD lesions specific to the immature joint and growth plate.[21] These include osteochondrosis/OCD, cuboidal bone disease, bone cysts, osteochondral collapse, insertion site avulsions, and physitis. Clinical signs of these conditions are variable but are usually seen in young horses since this is an abnormality of developing bone. Signs include variable degrees of lameness, alteration in posture or positioning of the limb, crooked legs, joint effusion, limb enlargement, ataxia (CVM only), and possible fractures. Additionally, multiple DOD conditions may be present in the same animal.

Although the exact cause for DOD is not known, the basic pathologic mechanism of disturbed endochondral ossification has been agreed upon, and there are multiple risk factors that appear to predispose young horses to the development of these diseases. Despite the lack of identification of single factors that cause DOD, a primary pathologic mechanism of vascular insult has been widely accepted.[126] Damage to the vasculature as it courses through the cartilage canals has been seen in foals, but in many cases a reparative cascade of events is often present, leading to a mix of clinical outcomes from clinical disease to healing.[87]

Despite the apparent common pathogenetic mechanism of vascular insult, the etiologic factors that incite disease are diverse. Many of the risk factors are man-made such as management and breeding practices, and others have been extrapolated from research work in other species.[27,39,86] For instance, the incidence of developmental problems in wild horses is low compared with

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domesticated breeding practices.[122] In addition, breeding for rapidly growing offspring that hopefully will do better in the show ring and feeding rations high in energy and protein appear to predispose these animals to DOD abnormalities presumably by contributing to rapid bone growth. Other risk factors include mineral imbalances such as copper deficiency or excess zinc, trauma, and genetic predilection.[99,125] Trace mineral deficiencies, copper in particular, have been incriminated in physitis and angular deformities in cattle,[105] and copper has been shown to cause clinical signs and joint pathology consistent with osteochondrosis in foals.[43,44] Copper is required for the enzyme lysyl oxidase, which itself is necessary for collagen cross-linking. Therefore, defective collagen cross-linking may impair the strength of bone collagen, producing essentially a "soft bone syndrome" particularly in the metaphyseal regions.[44] Excess zinc or alterations in the calcium/phosphorus ratios in the diet may also lead to clinical problems of DOD, but these are less well defined in horses than copper deficiency.[99]

Trauma to the metaphyseal or epiphyseal growth plates can contribute to altered growth, subchondral bone damage, and avulsion of defective bone (Figure 7.14), all of which may also predispose to DOD conditions. Abnormalities in vascular integrity of the developing osteochondral complex appear to be a factor in the pathogenesis of DOD.[88] Septic arthritis/osteomyelitis has been shown to cause cartilage canal abnormalities, leading to chondronecrosis in the articular–epiphyseal cartilage complex, causing OCD-type lesions.[132] Lecocq et al. demonstrated the histologic changes that occur in fetal and newborn foals at common sites of DOD and concluded that abnormalities in proteoglycan and collagen metabolism, cartilage canals, and vascularization of the epiphyseal growth cartilage play a role in DOD.[55] However, in most cases, the underlying cause of the DOD condition is multifactorial, usually obscure, and often never determined.[131] (The reader is referred to Chapter 10 for further information on specific DOD conditions such as physitis and ALDs.)

Incomplete Cuboidal Bone Ossification/Juvenile Spavin

Incomplete ossification of the cuboidal bones of the carpus or tarsus occurs most commonly in premature, twins, or underdeveloped newborn foals. At birth, the cuboidal bones have not ossified sufficiently to withstand the forces of normal weight-bearing, predisposing to variable degrees of carpal or tarsal bone wedging or collapse (Figure 7.15). Incomplete ossification without collapse is not readily apparent clinically and is best diagnosed with radiography to document abnormal cuboidal bone appearance. Moderate to severe incomplete ossification of tarsal bones has been seen in Thoroughbred foals born after a short gestational length, and those horses had less successful racing

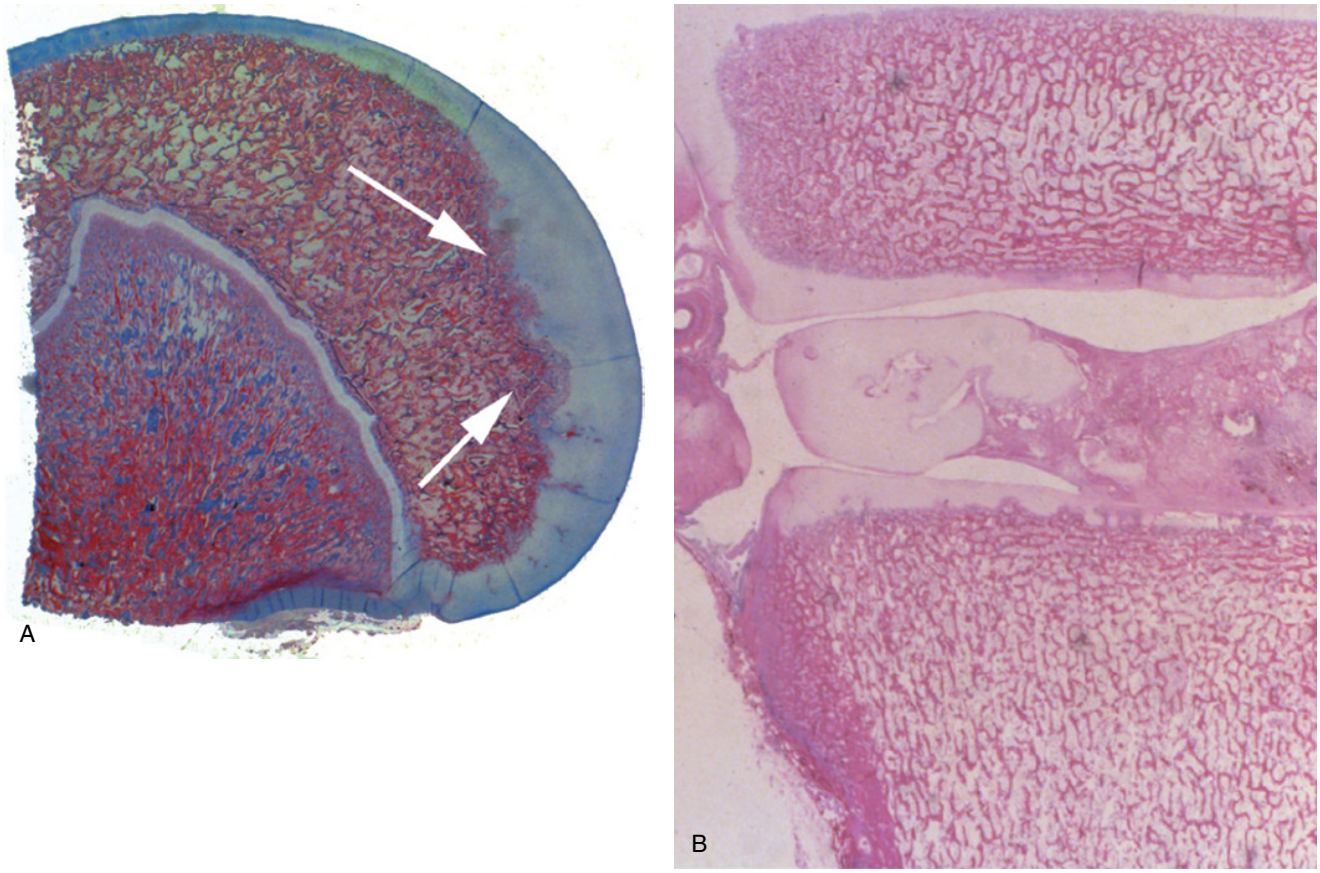


Figure 7.14. Histologic examples of abnormal bone formation typical of developmental orthopedic diseases. (A) Retained articular cartilage in the subchondral bone area (arrows) that is typical of osteochondritis dissecans. (B) Retained articular cartilage in the cuboidal bone of the tarsus leading to cuboidal bone malformation.

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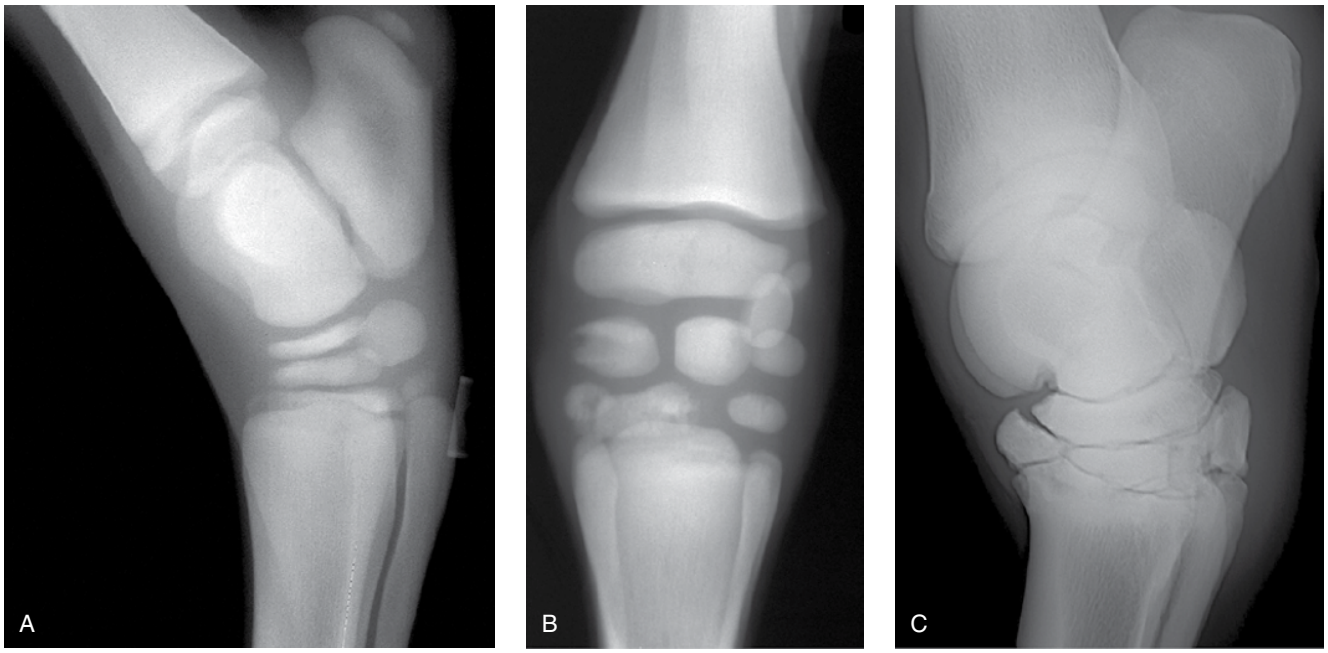


Figure 7.15. Radiographic images demonstrating incomplete ossification of the tarsal (A) and carpal (B) cuboidal bones in a premature foal. Without support, normal weight-bearing could lead to wedging and collapse of the tarsal bones (C).

careers than a group of normal foals.[38] Clinical signs associated with cuboidal bone collapse may be evident in the newborn foal as an ALD of the carpus or tarsus. Collapse of the tarsal bones is much more common than that of the carpal bones. Tarsal collapse is often associated with a sickle or cow-hocked conformation of the tarsus or the tarsus looks like it has a "curb." The degree of lameness is variable and may not become clinically apparent until later in life. Preventing cuboidal bone collapse in newborn foals with incomplete ossification involves minimizing compressive forces on the bones until they ossify. Confinement, sleeve casts, bandages, or bandages and splints may be used depending on the severity. In a previous study, foals with only minor tarsal bone collapse were able to perform as intended, whereas foals with more severe tarsal bone collapse and fragmentation could not be used for their intended purposes.[23] Additionally, incomplete ossification and mild collapse or wedging of the tarsal bones are thought to predispose to juvenile spavin in young horses. Horses with juvenile spavin have relatively severe signs of bone spavin at a young age with a history of minimal work (Figures 5.81, 10.27, and 10.58). Inherent abnormalities of the central and third tarsal bones are thought to contribute to the development of osteoarthritis (OA) in the distal tarsal joints at such an early age. In those young horses with obvious signs of juvenile spavin, surgical arthrodesis is often effective in salvaging the animal for athletic use.



Figure 7.16. Lateromedial radiographic view demonstrating a typical lateral ridge OCD lesion of the distal femur (arrow) and the fragments that have migrated within the joint (arrowhead).

Osteochondritis Dissecans (OCD)

OCD refers to cartilage or cartilage and bone (osteochondral) fragments or flaps that develop along the articular surfaces of joints in horses. These abnormalities are probably the most common manifestation of the DOD complex in horses. They usually occur along the non-weight-bearing surfaces of the joint and are especially common in the stifle, tarsus, and fetlock joints (Figure 7.16).[125] Horses with OCD lesions typically are only mildly lame but usually have joint effusion of the affected joint(s). These lesions are often bilateral and may or may not require arthroscopic surgery to remove the osteochondral fragment(s). The prognosis

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for performance of horses with OCD lesions is usually very good. More detailed discussion of OCD lesions can be found in Chapter 10.

Subchondral Cystic Lesions (SCLs)

SCLs (bone cysts or osseous cyst-like lesions) are commonly recognized pathologic entities of bones and joints in horses that may or may not cause lameness. SCLs may be nonarticular or articular. However, most lesions that contribute to lameness involve the weight-bearing area of an articular surface. Nonarticular lesions (which may or may not be classified as SCLs) usually involve the metaphysis and can go undiagnosed because they may not cause clinical signs and normal bone remodeling may resolve the defect. The most common age for diagnosis of SCLs, or at least the time when clinical signs develop, is usually 3 years or less.[6,42] However, horses demonstrate clinical signs related to SCLs over a wide age range, and the relationship between when the lesion develops and when the horse begins to show clinical signs is not known.[47] This relationship probably varies depending on the specific site of the SCL (most common location is the medial femoral condyle), the age when the lesion develops, and the occupation of the horse. Although most cases of SCL have a developmental cause, older horses with OA can develop SCL likely due to trauma. What causes or initiates the appearance of clinical signs in horses with articular SCLs remains unknown; however, the consistent morphologic characteristics of the lesions suggest a common pathologic mechanism of development.[123]

SCLs have been reported to occur at multiple locations in horses.[6,12] However, the most common sites that contribute to clinical problems involve the stifle, fetlock, pastern, coffin, and elbow joints (Figure 7.17). Controversy exists as to whether these lesions are caused by a defect in endochondral ossification, intra-articular subchondral bone trauma, or a combination of both.[6,12,47] Joint trauma can lead to the development of SCLs, and this has been shown experimentally and has been seen clinically (Figure 7.18).[6] However, many of these lesions are seen in young horses and are bilateral, suggesting a developmental defect. SCLs

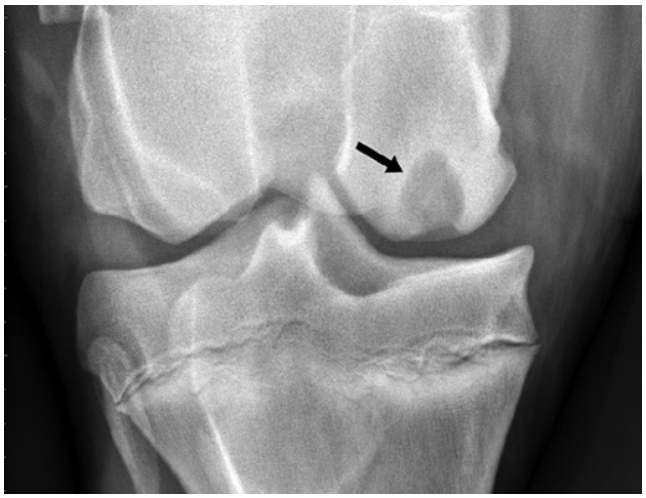
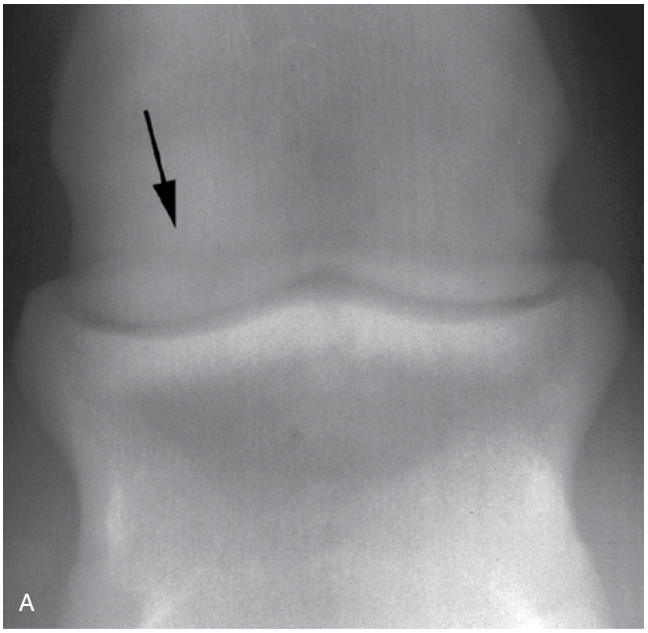


Figure 7.17. Caudocranial radiograph of the stifle reveals a subchondral cystic lesion within the medial femoral condyle (arrow) of this yearling Quarter horse filly.



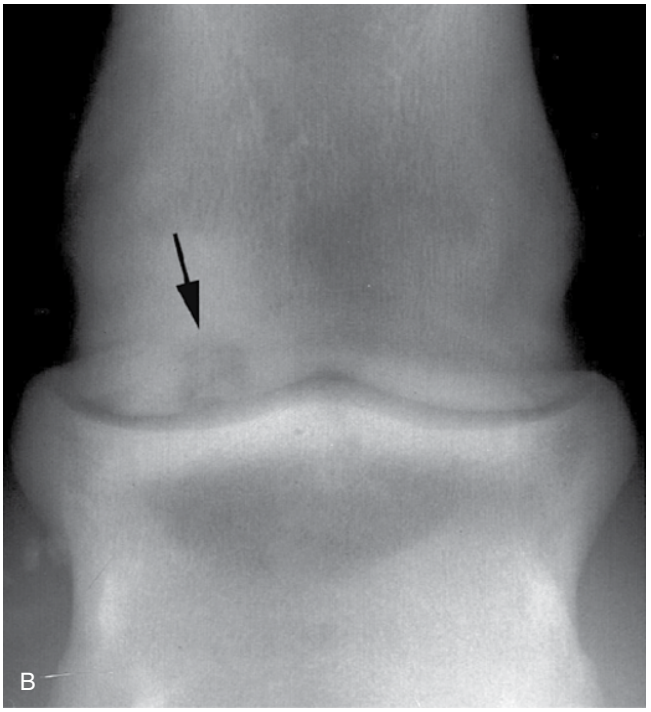


Figure 7.18. Dorsopalmar radiographs of the pastern in a 12-year-old Thoroughbred with a grade 4 out of 5 hindlimb lameness. Minimal radiographic abnormalities were visible on the initial radiograph (A, arrow). However, a subchondral cystic lesion with surrounding sclerosis was evident within the proximal phalanx on radiographs taken 6 months later (B, arrow).

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have been described as resulting from an infolding of abnormal cartilage into the underlying bony spongiosa.[6] The infolded cartilage becomes necrotic and its matrix remains nonmineralized so that osteoclasts and blood vessels do not migrate into the defect to enable repair of the defective cartilage and bone. The cystic lining is made up of fibrous tissue with active fibroplasia and capillary proliferation present in the tissue adjacent to the bone. The cyst lining has been shown experimentally to contain significant inflammatory mediators that likely contribute to the formation and progression of the cyst.[90]

Treatment of SCLs involves the use of either intra-articular medications (combined with systemic joint therapies), surgical debridement of the lesion, surgical debridement with bone graft, surgical debridement with augmentative therapies such as chondrocytes or stem cells, intralesional injection with corticosteroids, or transcystic screw placement (with or without intralesional therapy).[41,98,124] Because of the variable prognosis in horses with debridement of the lesion alone, additional treatment(s) of the bone defect is(are) being attempted by some surgeons. These include packing the entire defect with cancellous bone or packing the depth of the lesion with cancellous bone and filling the remaining bone defect with fibrin-laden chondrocytes containing growth factors. The goals of the latter treatment are to promote healing of both the bone and articular cartilage defects. Although nonsurgical treatment was usually recommended initially in most horses because of the variable success with surgical debridement of the lesions with or without the adjunctive treatments, the use of intralesional corticosteroid injections appears to give a more dependable prognosis. Use of a transcondylar screw reportedly eliminated lameness in 75% of treated horses but was reported to be less successful in horses >3 years of age.[98]

Effects of Exercise on the Immature Skeleton

Exercise can have a profound effect on the musculoskeletal system beginning at birth, and there has been considerable interest in the effects of early exercise on foals in an attempt to strengthen the system to reduce the incidence of injuries later in life. In initial studies by van de Lest et al., foals that were box stall rested were more likely to have reduced development than foals that were turned out full time and those that were turned out intermittently along with box stall rest.[58] Lepeule et al. have also shown an association between irregular exercise soon after birth and poor osteoarticular status.[57] The first author (CEK) has performed density studies in foals before and after exercise and found that prior to exercise, the subchondral bone pattern is blank, but at 6 months of age, the density pattern reflects the articulation between the joint surfaces (Figure 7.19).

In an attempt to strengthen musculoskeletal tissues later in life, a large study was performed in which foals that had additional exercise early in life were compared with those that were turned out. The goal was to provide galloping exercise in addition to normal pasture turnout to a group of foals and compare tissue responses as yearlings and as 3-year-olds. The investigators found minimal effects on tendons and ligaments, but fairly significant effects on articular cartilage. Namely, exercised foals had significantly higher viable chondrocyte content in their joints than pasture-reared foals, and they had less gross lesions in the carpi at 18 months of age.[24,54] However, there was no effect on articular cartilage changes that occurred in the palmar aspect of the parasagittal groove in the fetlock joints, a site of fracture and OA.[78] In the carpus though, Kim et al. showed that the incidence of gross lesions was significantly lower in the exercised group compared with the control group.[54] Subchondral bone formation was increased, but the overall bone content and density were no different between the groups. Overall, enforced galloping exercise did not produce negative effects and actually provided benefit to the joints.

Clinically, lack of exercise seems to have a profound effect on bone. For instance, it is not uncommon for young foals to fracture their proximal sesamoid bones during normal pasture exercise after being confined, usually in a hospital setting (Figure 7.20). The stall

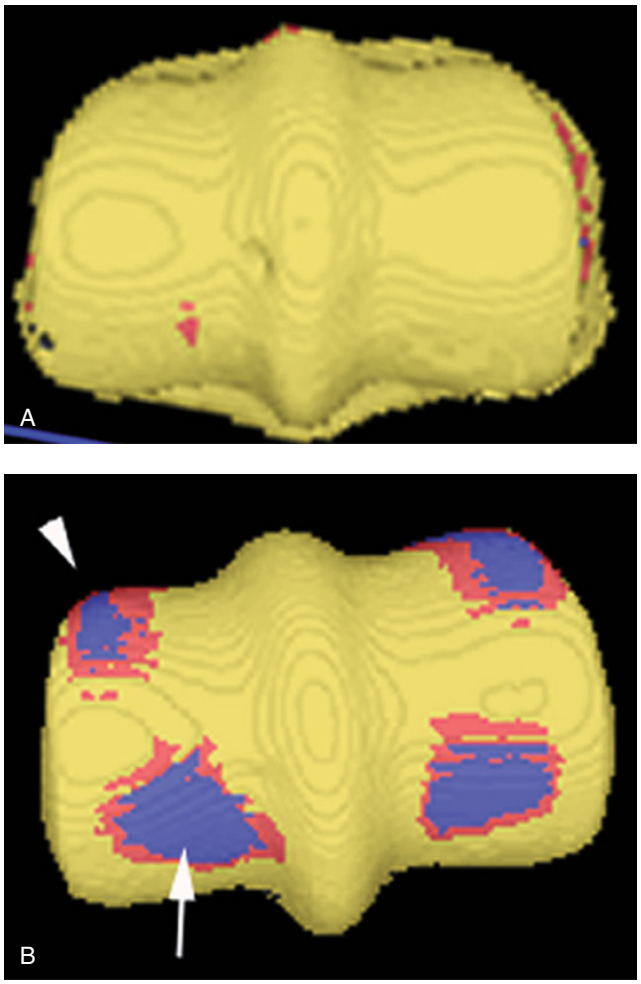


Figure 7.19. Three-dimensional computed tomographic images of the third metacarpal condyles, demonstrating the adaptation of bone to exercise in foals. In this example, subchondral bone density shortly after birth is homogeneous across the joint surface (A); however, after 6 months (B), the subchondral bone density increases in the areas that articulate with the proximal sesamoid bones (arrow) and the dorsal aspect of the first phalanx (arrowhead).

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Figure 7.20. A fracture of a proximal sesamoid bone in a foal that was initially stall confined at birth.

confinement appears to prevent normal adaptation to occur to the bones, especially the proximal sesamoid bones, and therefore create a situation in which the bones cannot withstand normal exercise. Therefore, care must be taken in introducing foals to turnout, especially if the mares sense competition with others in the pasture. The proximal sesamoid bones seem to be the most sensitive to lack of exercise. For instance, cast application to a limb will lead to radiographically apparent loss of bone density in the proximal sesamoid bones (Figure 7.21). The density of these bones will be 30%–40% lower with 6 weeks of casting.[35] This indicates that bones require a normal threshold of loading to maintain strength.

LOCAL DISEASES OF BONE

Exercise-Induced Bone Remodeling

Bone is very metabolically active under normal physiologic circumstances and in response to many types of injury. Its intricate micro- and macrostructural

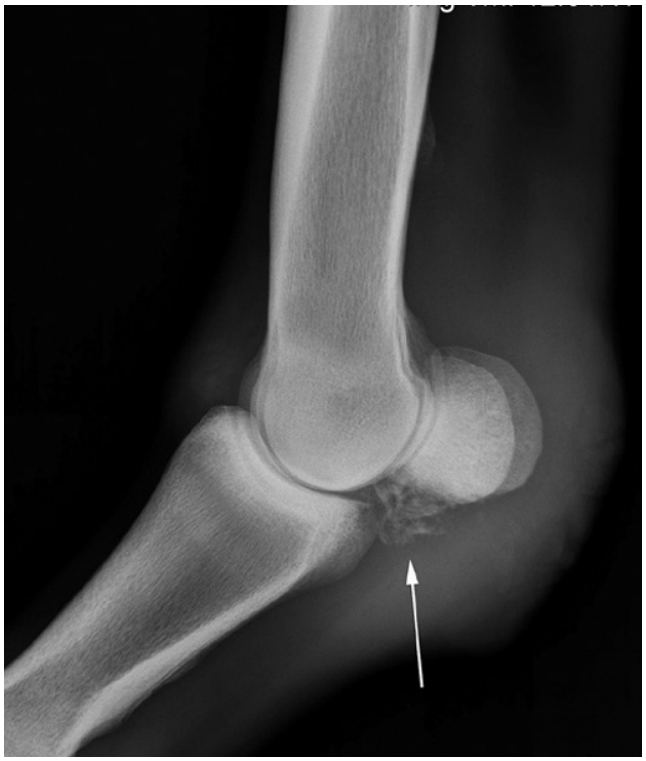


Figure 7.21. Lateromedial radiograph from a horse that was casted for 6 weeks. Notice the loss of bone density in the proximal sesamoid bones, as well as the formation of enthesophytes distally (arrow).

organization, combined with its high rate of metabolic activity, allows it to respond rapidly to many physical and biomechanical demands, especially in young animals.[62] Normal daily activity and exercise impose complex forces on the skeletal system that cause varying quantities of deformations of bone. Deformations within a bone are referred to as strains, and the local force intensities at these sites are the stresses, defined as a given force per unit area. The direction and magnitude of these stresses and strains are dependent on the geometry of the bone, the direction and magnitude of the load imposed on the bone, and the material properties of the bone. That is why joint surface geometry, limb conformation, and shoeing are so important to the pathogenesis and management of lameness. Bone in any species can undergo "remodeling" or "modeling." Equine bone remodels in response to the stresses placed on it according to Wolff's law, which in brief states that bone is laid down where it is needed and is resorbed where it is not needed. Examples of adaptive remodeling in equine bone include the cancellous portion of the proximal sesamoid bones that becomes less porous (stiffer and stronger) when trained on dirt tracks compared with untrained horses,[133] and the shape changes the metacarpus undergoes during training and racing to withstand the added forces applied to the bone.[79,83,84] Bone modeling occurs when the resorptive phase does not occur, and instead bone is laid down on top of normal trabecular structures. Examples of this are the palmar aspect of the third metacarpal condyle as it becomes dense and the radial facet of the third carpal bone

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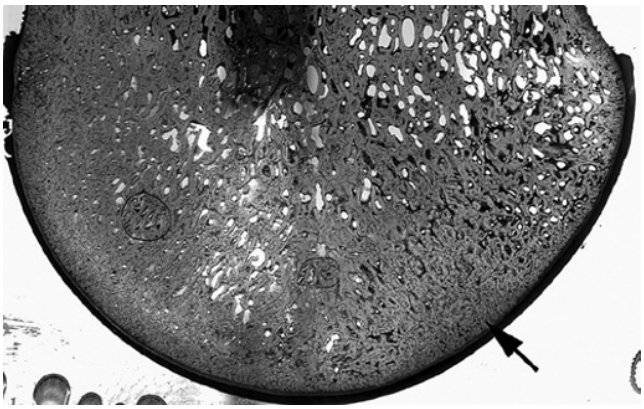


Figure 7.22. A histologic section through the palmar aspect of the third metacarpal condyle of a racehorse.

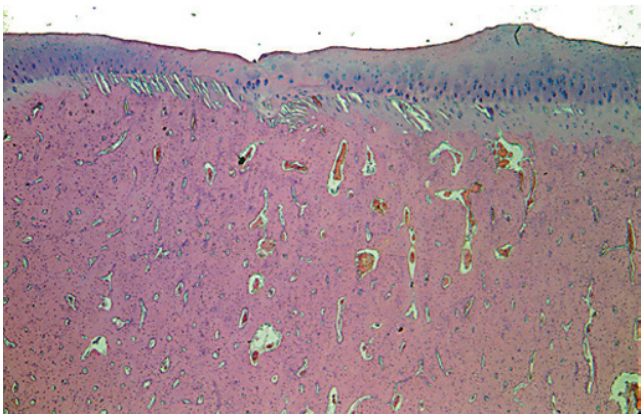


Figure 7.23. A histologic section through the palmar aspect of the third metacarpal condyles showing not only severe bone modeling but also overlying articular cartilage damage. This example appears to represent a modeling response beyond the threshold of normal adaptation to exercise.

with training[19,52] (Figure 7.22). These are only a couple of examples of bone's response to exercise, which undoubtedly occurs in every bone during training and performance. Unfortunately, there is a fine line between what is considered to be normal adaptive training (repetitive stress response) and overtraining that may be pathologic to the bone (repetitive stress injury)[106] (Figure 7.23). In addition, there is no objective means of characterizing that threshold between normal and pathologic loading.

The osteogenic response of bone to remodeling stimuli such as training or exercise appears to be most dependent on the strain magnitude and strain rate. In other words, the greater the load, the faster the load is applied, and the more times bone is loaded (repetitions), the greater will be the remodeling response. Increased magnitude of loading could be achieved by going at higher speeds or on harder surfaces (or a combination of each). Excessive repetitions of normal loads or too high of loads are thought to contribute to the development of dorsal metacarpal disease or stress or fatigue fractures in racehorses.[83,84,114] Fatigue fractures indicate that the bone has not remodeled or gained enough strength to withstand the forces (loads) applied to it.[84] In most cases, the exercise regimen should be altered to permit the bone to gain adequate strength to tolerate the higher loads. However, the clinical signs of fatigue fractures are vague, and often the horse will show no clinical signs until the fatigue fractures coalesce and cause a complete fracture. If excessive load is applied to a bone as a single event, the ultimate stresses and strains that the tissue can tolerate are exceeded, and a complete fracture occurs. Single-event trauma is a cause of complete fractures in many horses and foals. However, fatigue or stress fractures predispose to complete fractures especially in performance horses, presumably by decreasing the ultimate load that the bone can withstand before fracturing. This has been shown repeatedly at sites of injury in racehorses.[19,52] Examples include fatigue fractures of the cortical bone of the third metacarpus, the tibia, pelvic structures and humerus, and the subchondral bone of the carpal bones, proximal sesamoid bones, and third metacarpal condyles[5,65,115,118,130] (Figure 7.24). In addition to fracture, stress-induced modeling can lead to pathologic sclerosis of subchondral bone, especially in the third carpal bone and third metacarpal condyles (palmar osteochondral disease) that can lead to OA.

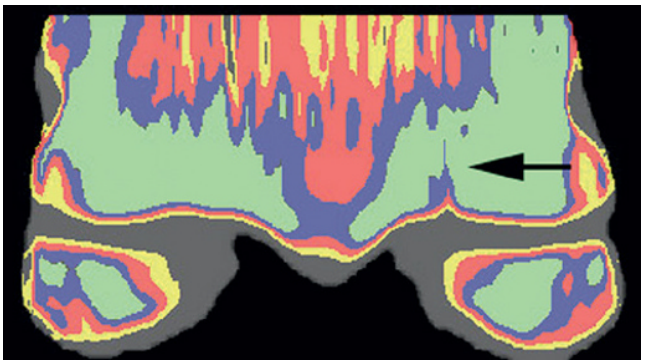


Figure 7.24. A computed tomographic image of the third metacarpal condyle of a Thoroughbred racehorse. Note the vertical demineralization in the area where condylar fractures typically occur (arrow).

Fractures

When a fracture occurs, there is usually a loss of structural continuity of the bone, and its function is impaired to varying degrees. The degree of altered function and the particular bone that is fractured often determine the type and severity of lameness. For example, a displaced fracture of the olecranon process or a fracture of one of the major load-bearing bones such as the radius or tibia usually produces a severe lameness. Conversely, a so-called osteochondral or "chip" fracture of a carpal bone produces a mild lameness that usually responds well to surgical removal, but if severe enough may predispose the horse to OA and cause a secondary lameness. However, most acute fractures will cause significant lameness regardless of their size and location. A fracture should always be considered as a possible cause of any non-weight-bearing lameness.

Fractures in horses are often due to a single high-energy traumatic event that completely disrupts the

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bone (breaks the bone into at least two pieces). In contrast to most complete fractures, fatigue fractures are due to chronic trauma that weakens the bone, rather than a single traumatic event that fractures the bone.[114] Specifically, chronic repetitive stress normally induces a bone strengthening cascade as a form of adaptation to withstand these forces (repetitive stress response). This is a normal process in all athletes, especially the horse, and creates normal patterns of bone enlargement or thickening, such as sclerosis through the processes of bone modeling and remodeling. Specifically, repetitive stress can induce the response one of two ways. Microdamage can stimulate remodeling events and lead to replacement of the damaged bone. Since osteoclastic function is more rapid than the osteoblastic response, this does create a window of vulnerability in which further high stresses can induce more damage within the relatively porous bone. The second form of bone strengthening in response to stress is through bone modeling, in which osteoid and mineral are added to existing bone to create either a dense sclerotic region (such as in subchondral bone) or geometric changes to bone architecture in order to maximize stress resistance (such as in cortical bone). This modeled bone is often more brittle than normal and in some instances can create pain and necrosis in these areas.

Fatigue fractures (repetitive stress injury) of the humerus, tibia, pelvis, and metacarpus/metatarsus (Figure 7.25) are commonly diagnosed in performance horses (primarily racehorses).[114,115] Initially these lesions cause variable degrees of lameness but may contribute to complete fracture of the involved bone with continued use.[114] Diagnosis of incomplete fractures in racehorses is critical to prevent catastrophic bone failure, but this can be difficult and the timing is critical. These horses may not be overtly lame at the time of examination, but may have a history of not working correctly, or be lame intermittently or immediately after work. If lameness is present, then diagnostic analgesia can be performed, but the proximal location of many of these fractures often precludes its use. In addition, many subchondral bone lesions may not extend into the joint early in the disease process, making intra-articular analgesia of little use.[21] Nuclear scintigraphy can be very helpful in locating suspected fatigue fractures that are not apparent on radiographs. Care must be taken in overinterpretation though, as young exercising horses will typically show sites of intense remodeling, especially in the fetlock joints.

Treatment of incomplete fractures is much less complicated than treating complete fractures and usually involves a period of inactivity combined with a change in training schedule.[119] In contrast, complete fractures of long bones of horses are one of the most difficult injuries to treat successfully, and many horses are still euthanized because of the severity of the fracture (Figure 7.26). The prognosis of repairing complete fractures in horses depends on the specific bone affected, the temperament of the horse, the age and size of the horse, the specific characteristics of the fracture, and the expertise of the surgeon, to name just a few. However, proper stabilization of the fracture for transport to a surgical facility for repair is crucial to achieving a favorable outcome, and newer repair techniques and implants have improved prognosis.



Figure 7.25. A radiograph of the third metacarpal bone from a 2-year-old racing Quarter Horse showing a fatigue fracture of the dorsal aspect of the bone (arrows).

Fracture Stabilization for Transport/Fracture Immobilization

Fractures are a frequently diagnosed problem in horses that often require emergency first aid treatment (Table 12.1). Horses are not readily ambulatory on three limbs and often become very anxious when they are unable to place weight on a fractured limb, which potentially can result in further injury. First aid measures should be directed toward minimizing further damage to the fractured limb and maintaining it in a position and condition that will facilitate repair. The goals of first aid fracture management are to prevent damage to neural and vascular elements of the limb, to prevent skin penetration of the fracture fragments or minimize further contamination of an existing wound, to relieve anxiety of the animal by stabilizing the fractured limb, to prevent deterioration of the patient's physiologic status, and to minimize further damage to the fractured bone ends and surrounding soft tissue.[11,13] Most of these objectives can be accomplished by proper stabilization or splinting of the fracture. However, fractures of the upper forelimb and hindlimb in horses are nearly

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Figure 7.26. A severely comminuted, open, displaced fracture of the distal third metacarpus. Even though the condylar portion of this fracture is common and amenable to repair (arrow), the comminuted nature of the cortical portion of this fracture makes any repair futile, especially since the area was avascular from the degree of comminution (arrowhead).

impossible to stabilize with external splints. Luckily, the bones in these locations are surrounded with large muscle groups, which inherently stabilize the fracture ends, making external coaptation less important.

Fracture immobilization serves several purposes. In horses, immobilization is more important to preserve limb vascularity than to prevent hemorrhage at the fracture site. Severe hemorrhage infrequently accompanies fractures in horses, but vascular thrombosis from continued stretching and direct trauma often lead to diminished vascularity of the distal limb. This can result in compromise to the hoof capsule and subsequent loss of the hoof. Limb immobilization also reduces the animal's anxiety enabling the horse to regain control of the limb even though the limb cannot bear weight. Once stabilized, most horses will rest the limb instead of continually trying to place it in a normal stance, which will cause further soft tissue and bone damage. Probably the most important purpose of immobilization is to prevent the development of an open fracture. Loss of intact skin coverage over a fracture predisposes the site to contamination and subsequent infection, especially if internal fixation is performed. Equine skin is thin and readily penetrated by sharp bone fragments, and there is little soft tissue support, such as muscle, below the carpus and tarsus. In general, fractures of the distal (phalanges) and upper (humerus, ulna, and femur) aspects of the horse's limbs rarely become open, suggesting that proper immobilization of fractures involving the metacarpus/metatarsus, radius, and tibia is the most critical to prevent the development of an open fracture during transport. Fractures at these sites often require emergency surgical repair to prevent open fracture configuration. The reader is referred to the section on musculoskeletal emergencies in Chapter 12 for specific details on fracture immobilization.

Fracture Healing in Horses

Fracture healing can be considered a series of processes that occur in sequence but are often overlapping. The healing process can be divided into three distinct phases: inflammatory, reparative, and remodeling.[63] During this process the bone will unite by one of two patterns: primary or direct healing and secondary or indirect healing. With primary bone healing, the bone ends heal directly by Haversian remodeling in contact and noncontact areas without the formation of a bone callus. Rigid fracture stabilization and correct anatomical reduction of the fracture are required for primary or direct bone healing to occur. With indirect or secondary bone healing, fibrous tissue or fibrocartilage is formed initially between the fracture fragments with subsequent replacement with new bone. Periosteal and endosteal callus is formed to unite the bone ends.[63] In horses, because of their large size and weight, primary healing is rare, and even with rigid internal fixation, a combination of primary and secondary bone healing is likely to occur.

Inflammatory Phase

The inflammatory phase occurs over the first 2–3 weeks after injury, and it is considered critical for the reparative phase of fracture healing that follows. During this phase the cellular mechanisms necessary for repair and the processes protecting the healing tissue from infection are activated. If the inflammatory response is impaired, tissue healing is compromised. Chemical messengers mediate the inflammatory reaction by causing vasodilation, migration of leukocytes, and chemotaxis of substances necessary for the repair process. In particular, bone morphogenetic proteins play an important role in the initiation of fracture repair.[91] The "osteoimmunological" response is being given greater investigation lately as it is key to cell signaling and recruitment for fracture healing. Polymorphonuclear cells remove debris, macrophages and monocytes stimulate osteoclastic and progenitor cell migration, and based on the integrity of the vascularity, chondrocytes and osteoblasts are stimulated to the site. The role of nonsteroidal antiinflammatory medication in fracture cases is being critically reviewed in human medicine, and its use is being limited.

Reparative Phase

During the reparative phase, the pattern of fracture healing is highly susceptible to mechanical factors such as interfragmentary motion. With spontaneous fracture healing, the initial hematoma and fibrous tissues provide

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some internal stress and support, and periosteal and endosteal callus formation provides interfragmentary stabilization, and bone union occurs by intramembranous and endochondral ossification.[63] This process can take from 2 to 12 months to be completed depending on the method of fracture fixation that was utilized, the stability of the fracture, and the size of the fracture gap (fracture displacement).

Remodeling Phase

The remodeling phase occurs during and following the reparative phase. Avascular and necrotic regions of bone are replaced by Haversian remodeling.[63] Malalignment of fracture fragments may be corrected during this phase of healing by remodeling of the fracture site and functional adaptation, particularly in young animals. With weight-bearing and loading of the fracture, bone is removed from the convex surfaces and laid down on the concave surfaces. This process tends to realign the bone after malunion (Figure 7.27). However, fracture remodeling cannot correct torsional deformities associated with fracture healing. Theoretically, bone can heal completely and regain pre-fracture strength and function.



Figure 7.27. Radiograph of the third metacarpal bone of a horse that presented several months after the fracture had been treated with external splinting. The fracture had healed but was severely malaligned.

Fracture Fixation

In horses, more than any other domestic animal one has to carefully define "successful" fracture healing. For centuries, bone has been observed to heal by production of callus, but the end result was often angulation, rotation, or limb shortening. With intra-articular fractures, a certain amount of OA was often the end result. Fixation techniques used to achieve primary bone healing with intra-articular fractures have greatly decreased the morbidity associated with OA in these cases. In addition, improved techniques in internal fixation of long bone fractures have emphasized improving the implants to withstand massive functional forces, thus preventing failure due to mechanical overload. Such implants must also be strong enough to maintain their integrity until the bone has united, without breaking under fatigue. However, despite improvements in fracture fixation equipment, anesthetic protocols, and recovery methods, successful repair of some long bone fractures in horses remains very difficult.

Stress protection is a phenomenon seen when a bone that has been rigidly immobilized by a plate(s) undergoes certain histologic events, including loss of bone mass without a corresponding reduction in size (quantitative osteopenia).[119] Stress protection results in Haversian remodeling and has generated considerable interest in man and small animals because of the potential for refracture of the bone following removal of the plate. Stress protection is almost an unknown occurrence in the horse, even in foals, because of the greater loads imparted on the implants compared to man and other smaller animals.[119] In fact, the size and weight of horses often stress the limits of stress tolerance in implants. While the emphasis of research activity in man and small animals has focused on the development of more flexible implants, in horses the emphasis has been in the reverse direction to provide stronger implants in an attempt to overcome the massive loading of the implants.

An important consideration in the horse is stress concentration. This is where biomechanical loads are concentrated in a small area of normal or weakened bone, potentially leading to complete bone failure.[119] This primarily occurs in the diaphysis of long bones but may also develop elsewhere along the bone. Examples of stress concentration include drill holes that are not filled with implants during internal fixation and vacant screw holes after implant removal (such as after metacarpal stress fracture repair).[119]Additionally, stress concentration occurs at the ends of bone plates especially if they stop in the mid-diaphyseal region of a bone and at intramedullary (IM) pin hole sites following removal of external fixators. These locations are all areas where small areas of cortical bone are absent or have been weakened and can fail if excessive loading of the bone occurs.

Compression Fixation

The use of various methods of compression in the treatment of fractures in man and animals is widely accepted. Under stable conditions it is recognized that both cancellous and cortical bones heal by primary bone

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union, without callus formation. Under these circumstances Schenk and Willenegger[102] showed that healing resulted by proliferation of new osteons growing parallel to the long axis of the bone, first through the necrotic bone ends and then across the fracture. This is termed "primary bone healing" and is the ultimate goal for repairing any fractures in horses or any species. With compression across the fracture, the mechanical stability with little or no motion between the fragments creates more favorable conditions for primary bone healing. Therefore, with internal fixation, the function of the implant (plate and/or screws) is to hold the bone fragments under compression with little to no movement until fracture union has occurred.[119]

Compression fixation of fractures is often the goal for fracture repair in horses. Intra-articular fractures when rigidly compressed usually heal rapidly without the development of secondary OA. Long bone fractures repaired with bone plates can heal without exuberant callus that would interfere with nearby soft tissue structures. However, compression fixation cannot be achieved in many cases due to the nature of the fracture and the temperament and size of the patient. In general, compression fixation and primary bone healing can be accomplished with most intra-articular fractures in horses, but is usually not completely achieved in long bone fractures. Most long bone fractures will develop varying degrees of callus formation during the healing process, which in most cases is not a major problem. Specific methods for repair of certain fractures, in particular those amenable to lag screw fixation, are discussed in Chapter 5 when a particular bone or joint is discussed. Below are general principles regarding fracture fixation in horses.



Figure 7.28. A fracture of the lateral condyle of the distal third metacarpus that requires lag screw fixation to achieve precise alignment of the joint surface.

Lag Screw Principle

One method to compress a fracture is to use a lag screw, a basic technique used routinely by carpenters and engineers. It is particularly suited for intra-articular fractures in horses where accurate anatomic alignment of the joint surface is essential to avoid secondary OA (Figure 7.28). Lag screws alone are used rarely to repair major long bone fractures in horses or even small foals, because they are simply not strong enough. Lag screws are ideally suited to repair slab fractures of the third carpal bone (Figure 7.29), condylar fractures of the distal third metacarpus and metatarsus (Figure 7.28), sagittal fractures of the proximal phalanx, and some tarsal bone fractures. Additionally, lag screws can be used in conjunction with plates to repair comminuted fractures where fracture reconstruction is performed with screws, and a plate is applied, or the lag screws are inserted through the plate holes. Varying sizes of cortical bone screws (3.5, 4.5, or 5.5 mm in diameter) can be used as lag screws. The 5.5-mm screw is being utilized more commonly in horses because of its increased strength and holding power compared with the 4.5-mm screw.

To achieve the lag screw principle, threads must gain purchase only in the far bone fragment. For cortical screws this is achieved by overdrilling the fragment next to the screw head to such a size that the screw threads will not engage this portion of the bone. This hole is termed the glide hole (Figure 7.30). If this hole is not overdrilled, when the screw is tightened the gap between the fracture fragments would be maintained and no compression would be achieved. Therefore the screw should only achieve purchase in the "far" or transcortex, i.e. cortex away from the head.

In certain bone regions, particularly in the metaphysis of bones in foals, the bone may be too soft for cortical screws to gain purchase. Cancellous screws can be used to achieve compression between fragments in these locations but should be used with caution in dense cortical bone. Therefore, whenever possible, cortical bone screws should be used in horses with the exception of young foals with very soft bone. To exert the maximum amount of interfragmentary compression, a lag screw should be inserted at right angles to the fracture plane. However, if the bone is under some axial load, the screw ideally should be inserted at right angles to the long axis of the bone. Therefore, the ideal direction to achieve maximum interfragmentary compression and resistance to axial loading is a direction between these two extremes.[119] In general two or more lag screws are used if possible to prevent rotation of the fragment. This is not always possible especially for fractures involving the small carpal or tarsal bones.

Pre-tapped cortical screws are used primarily for lag screw fixation in horses. Self-tapping screws were

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originally thought to provide poor bone holding qualities compared to the pre-tapped screws.[119] Both 5.5- and 6.5-mm screws inserted into cadaver foal metacarpal/metatarsal bones pre-tapped had significantly greater holding power than the same size screws inserted self-tapped.[134] However, Schatzker et al. and Andreas et al. in similar studies demonstrated that self-tapping screws had the same holding power and pullout strength as pre-tapped screws.[2,4,100,101] Use of self-tapping screws may be difficult in very dense cortical bone, and the screws may actually break. Self-tapping screws (4.5 mm) inserted into equine third metatarsal bones did not break, but could not be inserted continuously.[3] In addition, pre-tapped screws can be placed in the bone with greater precision, thereby producing better holding power.[100,101] In general, most cortical screws inserted into equine bone are pre-tapped. Self-tapping screws are used in locking plates for better security at the screw-plate location.



Figure 7.29. Lateral radiograph of the carpus in a racehorse following lag screw fixation of a third carpal bone slab fracture with a 3.5-mm cortical screw. Larger third carpal bone fractures in horses are often stabilized with 4.5-mm cortical screws.

Cannulated and bioabsorbable screws are also available for use in horses. Cannulated screws are hollow enabling screw placement over a guide pin that helps maintain fracture reduction and precise screw positioning during the lag screw procedure. They are available in a variety of sizes, but the concerns of using cannulated screws in horses are their questionable strength (since they are hollow) and the expense.[3,4,16] Bioabsorbable screws are made of polylactic acid derivatives and once placed would theoretically not need to be removed. They are used sparingly in horses because of the expense and their questionable strength especially in shear.[28] However, one *in vitro* experimental study using equine third carpal slab fractures as a model suggested that they have comparable biomechanical properties to 4.5-mm cortical screws.[76] A headless tapered variable pitch screw has also been advocated for use in horses.[14] The variable pitch nature of the screw allows for interfragmentary compression to occur without the presence of a screw head, which some surgeons feel can be a source of inflammation and irritation.

Other Methods of Internal Fixation

Many fractures in horses, especially those involving long bones, cannot be repaired with lag screws alone. Other methods of repairing long bone fractures include bone plating, tension band wiring (Figure 7.31), IM

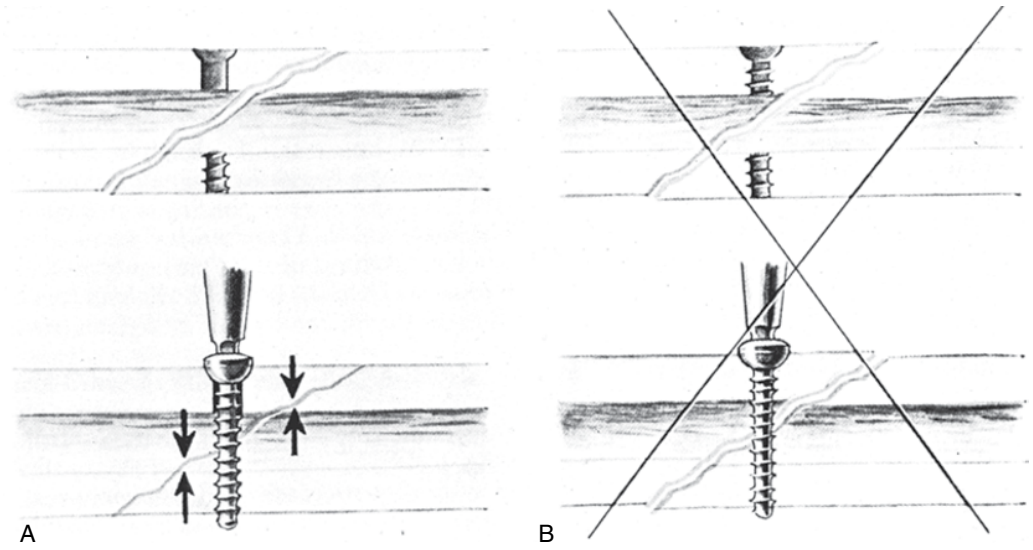


Figure 7.30. (A) Correct execution of the lag screw principle. (B) Incorrect execution of the lag screw principle. The cortex under the screw head was not overdrilled, and the gap between the bone fragments cannot close.

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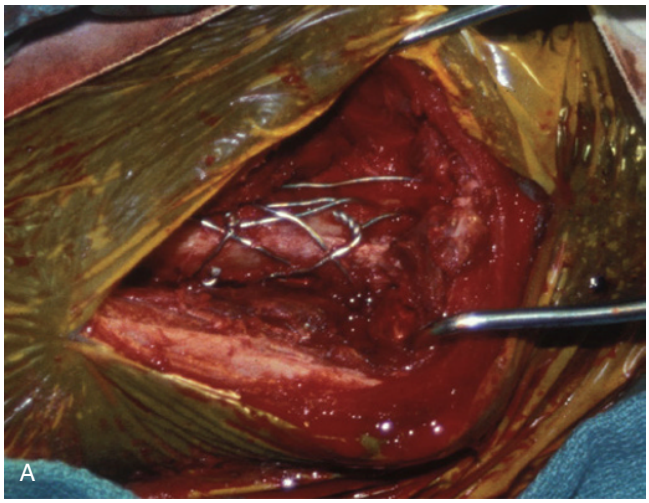




Figure 7.31. Intraoperative view (A) and postoperative lateral radiograph (B) of a fractured olecranon in a young foal that was repaired using tension band wires only. The foal did very well after surgery and the fracture healed without complications.

pinning, IM interlocking nails, and the use of external fixators.[3,4] Bone plating provides the most rigid fixation of fractures but cannot be used in all cases because of the specific characteristics of the fracture and the expense. For those fractures amenable to bone plating, one and many times two plates are used to provide adequate fixation (Figure 7.32). The plates (usually 4.5 mm in thickness) are best placed on the tension side of the bone and at right angles to each other when two plates are used. Either 4.5- or 5.5-mm cortical screws can be used to secure the plates to the bone, and these should be placed as lag screws across the fracture if possible. Special-purpose bone plates used occasionally in horses include the dynamic condylar screw (DCS), dynamic hip screw (DHS), angled blade plate (ABP), and the cobra head plate.[3,4,42] Plate luting refers to placing polymethylmethacrylate (PMMA) either under the plate or around the screw heads to further increase the strength and stability of the fixation.[81,111] Improved bone plate contact is achieved, and therefore luting is most beneficial in large horses to increase the overall strength of the implant.[81,111] However, locking plates have made the use of plate luting unnecessary. The locking plates are designed so that the screw heads fix into the plate with a screw mechanism and do not rely on friction between the plate and the bone for stability (Figures 7.32 and 7.33). Shearing of the screw heads is a common mode of failure, and this mechanism reduces the stress between the screw head and the plate, allowing for a more stable fixation. This has been used routinely in equine fracture repair and arthrodesis and lends itself well for use in minimally invasive percutaneous plate osteosynthesis.[46,59,92]

IM interlocking nails are relatively new in equine orthopedics and have primarily been used to repair humeral, femoral, and some metatarsal/metacarpal fractures.[3,70] The IM nail is placed within the medullary cavity of the bone and is transfixed to the bone above and below the fracture using bone screws.[70,71] This method of repair is thought to create a more biomechanically sound fixation by placing the implant within the bone, rather than applying bone plates to the external surface of the bone. The potential benefit of interlocking nails in equine orthopedics awaits further clinical use.

External Fixation of Fractures

External fixators or transfixation pin casts have become more widely used for fracture repair in horses over the last several years.[68,69,77] They are most useful to stabilize or prevent collapse of comminuted fractures of the distal limb where other forms of internal fixation cannot be used and to treat open fractures where placement of implants would likely result in osteomyelitis.[69,77,80,82,92,94,97] The fixator pins (usually 0.25 inch or 6.35 mm in diameter for average adult horse) can be placed above and below the fracture, but in most cases pins are placed only above the fracture (Figure 7.34). Polymethylmethacrylate or stainless steel sidebars can be used as the construct of the fixator to connect the pins if used above and below the fracture or

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Figure 7.32. This midshaft radial fracture in a foal was repaired using two locking compression plates.



Figure 7.33. A partial carpal arthrodesis performed using two locking compression plates.

a fiberglass cast is applied to the limb to support the pins.[69,77,80,82,92] In most cases of phalangeal fractures, 2–3 pins are placed in the distal to middle aspect of the metatarsus/metacarpus and a lower limb fiberglass cast is applied to the limb to serve as the sidebars of the external fixator.[68,69] The transfixation cast permits the horse to ambulate on the limb without further displacement of the fracture during the healing process. Alternatively, a specially designed external fixator for horses (Nunamaker external fixator) that does not utilize a fiberglass cast may be used.[80,82] Catastrophic failure of the bone through one of the external fixator pinholes is the most severe complication of this form of fixation. Other potential problems include infection around the pins, premature pin loosening, chronic pain associated with the pins, and prolonged fracture healing.

Fractures as a Cause of Lameness

Consequences of fracture healing may lead to subsequent lameness. This has been termed "fracture disease" in humans and is seen under certain circumstances in horses. A major cause of disability in humans following fracture healing is stiffness of joints from disuse. This is rarely seen in horses and should not be confused with stiffening due to ankylosis of joints associated with the development of OA.[119] In fact, joint laxity, especially in young animals with fractures treated with casts, seems to occur more commonly than joint stiffness in horses. However, most fractures are accompanied by varying degrees of soft tissue (muscle, tendon, and ligament) damage that may be replaced with scar tissue. Subsequently there is obliteration of normal tissue planes that may impair tendon function, produce stiffening of neighboring joints, and cause flexural deformities in growing animals. A nonunion can occur due to infection, lack of vascularity, or relative instability (Figure 7.35). This instability leads to accumulation of fibrous tissue and fibrocartilage, which due to constant micromotion cannot mineralize. A malunion of the fracture may also lead to athletic disability in some horses (Figure 7.27). In addition, implant-associated pain from bone plates or screws may cause chronic lameness or poor performance in athletic horses. In some cases, implants used for

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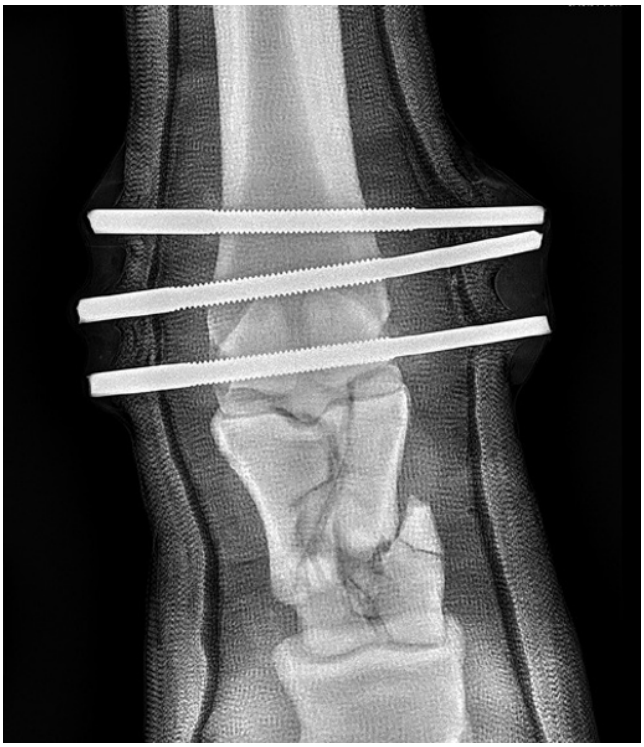


Figure 7.34. Three 0.25-in. (6.35-mm) diameter threaded intramedullary (IM) pins were placed through the distal metatarsus and incorporated into a fiberglass cast (transfixation pin cast) to prevent collapse of this severely comminuted first phalanx fracture. Two transfixation pins are usually recommended with the most distal pin placed in the center of, or slightly proximal to, the condylar fossa. *Source*: Rossignol et al.[97] Reproduced with permission of Veterinary Surgery.

internal fixation of fractures may need to be removed in performance horses to prevent these problems.

Another problem that can arise secondary to fracture fixation is the secondary problems related to casting. It is not uncommon to see significant loss of bone density, especially within the proximal sesamoid bones[35] (Figure 7.21). Clinically significant lameness unrelated to the primary disease can also result, as has been seen in several experimental studies in which lower limb casts have been applied to horses. Lameness, pain on flexion, and significant physical changes to bone density and soft tissue integrity have been observed.

Tendon and muscle flaccidity and atrophy of surrounding muscles are also seen in horses with fractures treated with external immobilization such as casts. This usually is a temporary problem that is self-correcting with time, but may lead to permanent lameness. Other aspects of so-called fracture disease in the non-fractured limb include ALDs due to excessive axial loading on active growth plates in young horses, stretching of flexor tendons and associated muscles, and support limb laminitis due to excessive weight-bearing. Support limb laminitis with rotation of the distal phalanx is unique to the horse and is a potentially devastating complication of equine fracture repair.

Infection is a serious complication of fractures that can eventually lead to permanent lameness or a nonunion of the fracture. Infection is most likely to occur with

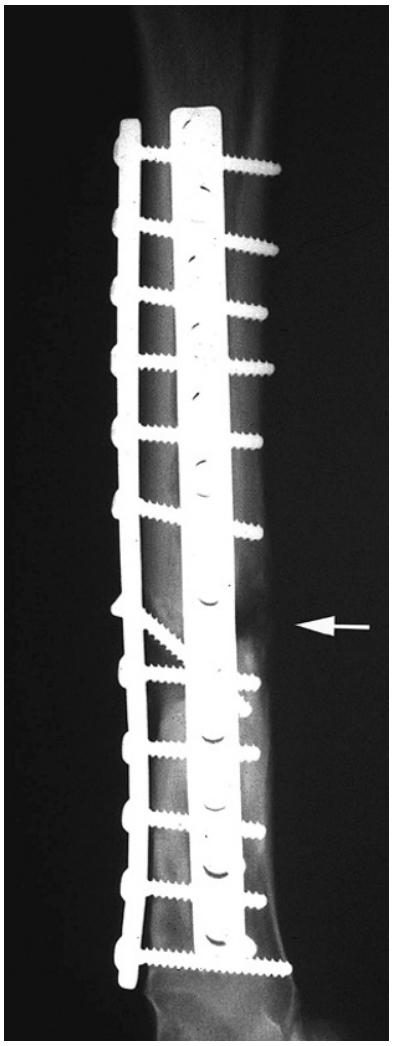


Figure 7.35. A radiograph of the third metacarpal fracture repaired with internal fixation. Note the lysis at the fracture line that is indicative of a nonunion (arrow).

open fractures or those repaired with internal fixation.[7] Severe infections may necessitate euthanasia of the animal, whereas milder more chronic infections may necessitate removal of the implants or necrotic bone to resolve the infection. Open infected fractures that eventually heal are often accompanied by considerably more fibrosis with a greater chance of loss of function of surrounding structures than closed noninfected fractures. The limb may be permanently thickened due to scar tissue and callus formation, which may lead to impaired limb function.[119]

Bone Infections

Osteitis and osteomyelitis are terms used to describe inflammation of bone involving the periosteum and connective tissues of the Haversian and Volkmann canals as well as the medullary cavity.[119] If the process begins or involves the periosteum and outer bone cortex, the term osteitis or osteoperiostitis is used. If the infection involves the medullary cavity, the term osteomyelitis is used. The prognosis and treatments for osteitis and osteomyelitis

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are quite different, and it is important to make a distinction between the two categories of bone infection.[7]

Infectious Osteitis

Osteitis commonly occurs in the extremities of the horse (mostly metacarpal/metatarsal regions) because of the sparse soft tissue coverage in this location. It is usually the result of infection from a nearby septic process or from a break in the skin.[7,10] Osteitis is seen frequently when a horse is kicked without breaking the overlying skin and may be similar to a bone bruise when no sequestrum develops. If the skin is broken, exposing the periosteum, the outer layers of cortical bone may eventually die, whereas the deeper cortical layers of bone survive due to the blood supply from endosteal vessels. For example, lacerations with bone exposure of the dorsal aspect of the metacarpus/metatarsus frequently develop osteitis and sequestration.[7,10] Bacteria that gain entrance to the bone lodge in the superficial layers of the bone, resulting in a thin layer of dead bone (bone sequestrum) within the wound (Figure 7.36). Although granulation tissue may advance over the bone sequestrum, the rate of advancement is usually slow. Occasionally granulation tissue advances under the sequestrum and extrudes it from the wound. The rate of healing of a wound can usually be accelerated by early removal of the sequestrum.

By definition the two requirements for the formation of a sequestrum are avascularity and infection. Therefore, most surgeons feel that blunt trauma to the bone cortex does not cause sequestration in the absence of infection.[7,10]



Figure 7.36. A sequestrum on the lateral aspect of the fourth metatarsal bone (arrow) secondary to trauma.

Sequestration without skin penetration does occur in horses although it is rare. In cases in which there is no break in the skin, the hematoma may become infected hematogenously leading to sequestrum formation, fistulation, drainage, and a nonhealing wound. This appears to occur most commonly with injuries to the splint bones, but most have skin wounds that lead to secondary bacterial infection. Chronic persistent drainage from any wound in the horse suggests the presence of a bone sequestrum or foreign body. Drainage will rarely subside or at least wound healing will be substantially prolonged without surgical removal of the sequestrum. This is because the pathogenic organisms reside within the necrotic bone, which is avascular, thereby resisting the animal's immune defenses.

The severity of lameness accompanying osteitis in horses is variable and inconsistent. In addition, the radiographic signs of osteitis will depend on the duration that has elapsed between the injury and time of examination. Initially there may be soft tissue swelling with evidence of bone resorption seen radiographically. At 7–14 days following the injury, periosteal proliferation may be evident. Sequestrum formation may also be visible at that time as osteoclastic resorption occurs at the periphery of the damaged bone. Radiographic evidence of a sequestrum is usually not visible for a minimum 2–3 weeks after the injury.[10] At this time, the sequestrum and the sclerotic margin around the sequestrum called the involucrum are usually visible.[7]

Occasionally an osteitis may resolve spontaneously, especially if there is no infectious component or if the sequestrum is small and extruded from the wound (which rarely occurs on its own). If bacteria and necrotic bone are present, the wound will be exudative indefinitely until the sequestrum is removed.[119] Wound debridement of unhealthy scar tissue and necrotic bone is usually required for healing to occur. Removal of bone sequestra is best performed with the animal under general anesthesia. However, thin cortical sequestra associated with avulsion injuries of the dorsal aspect of the cannon bone can often be removed in the standing sedated horse. After the surrounding granulation and scar tissue have been excised, the area should be curetted until the bone appears to be healthy. Most wounds are either closed primarily or left to heal by second intention following debridement.

Parenteral antibiotics are of limited value when used alone to treat bone sequestra because of poor penetration of the antibiotics into the necrotic bone. Antibiotics are indicated if there are signs of cellulitis (phlegmon) associated with the lesion and following surgical debridement of the wound. Occasionally a mild sequestrum can be effectively treated with antibiotics administered through regional limb perfusion, which is a technique commonly used regardless if surgery is performed. Usually a wide variety of organisms (secondary pathogens) can be cultured from the wound, and occasionally these bacteria are resistant to antibiotics that are of practical use in the horse. Culturing the sequestrum itself usually gives the most accurate diagnosis as to the causative bacteria. The prognosis for horses with osteitis and sequestrum removal is usually excellent.

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Osteomyelitis

Osteomyelitis is a more extensive inflammation of the bone than osteitis that begins within or extends into the medullary cavity. Osteomyelitis in horses can be divided into three categories based on the origin of the infection: hematogenous, traumatic, or iatrogenic.[7] Osteomyelitis from hematogenous origin occurs primarily in neonates and only rarely in adults. Traumatic osteomyelitis can occur in any age horse and is usually due to penetrating wounds or open fractures. Iatrogenic causes of osteomyelitis include surgery such as internal fixation of fractures or intra-articular injections of medications.

Hematogenous Infection

The localization of hematogenous osteomyelitis in the metaphyseal region of neonates can be explained by sluggish metaphyseal blood flow where the blood vessels form terminal sinusoids. This permits the bacteria to localize in these areas and to establish an infection (Figure 7.37). The infection in the bone spreads by way of Haversian and Volkmann's cavities, and prostaglandins are considered responsible for bone destruction.[119] Thrombosis of blood vessels also occurs as the infection spreads, producing death of the osteocytes in their lacunae.[7,119] The inflammatory process may increase pressure within the bone, further impairing blood supply. The end result is bone necrosis with possible sequestrum formation. Firth has classified hematogenous infections in foals into three categories depending on the location of the infection. S-type infections involve the synovial membrane of joints, P-types involve the physis and usually the metaphysis, and E-type infections involve the epiphysis.[29] However, these infections are not completely isolated, and foals may have multiple types of hematogenous infection at multiple sites (Figures 7.38 and 7.39). Bone infection due to *Salmonella* spp., for example, typically has multiple bones involved.[119]

Hematogenous osteomyelitis is frequently secondary to infections elsewhere in the animal's body, such as the umbilicus, gastrointestinal tract, or lungs.[7,29] Foals with a compromised immune system due to failure of passive transfer or septicemia appear to be predisposed to hematogenous osteomyelitis. Many of these foals may have multiple body systems involved with signs referable to the

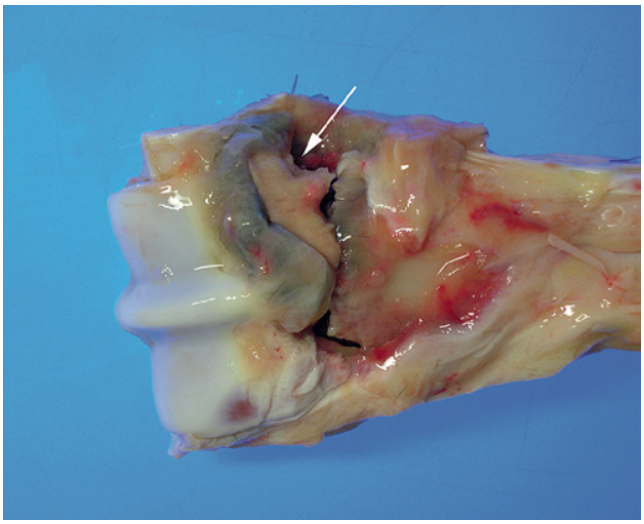


Figure 7.37. Gross image of a severely septic physis (arrow) that spread into the joint and led to a pathologic fracture of the limb.

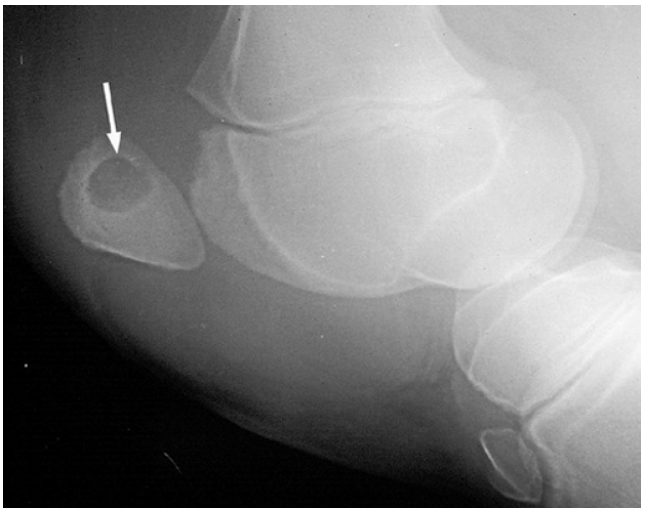


Figure 7.38. Lateral radiograph of the stifle in a foal with lameness and severe effusion of the femoropatellar joint. Lysis within the patella suggestive of hematogenous osteomyelitis was present (arrow). The caudal or ventral aspect of the patella directly below the lesion was removed using the arthroscope and the joint was lavaged.



Figure 7.39. A radiographic view of a shoulder in a foal demonstrating a metaphyseal lytic lesion (arrow). This foal also had septic arthritis of the shoulder joint and bicipital bursa.

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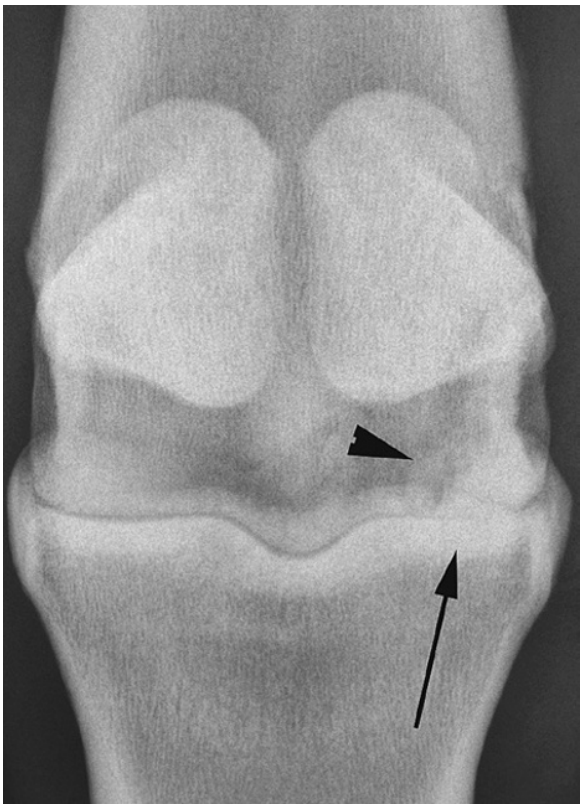


Figure 7.40. Radiographs of the fetlock joint from a mare that had a penetrating injury to the fetlock joint 2 weeks previously. Note the severe subchondral bone lysis (arrowhead) and joint space collapse (arrow) in the joint secondary to sepsis.



Figure 7.41. An oblique radiograph of the tarsus in a horse with a puncture wound and infection of the calcaneal bursa. Lysis within the proximal aspect of the calcaneus (black arrows) suggested concurrent osteitis or osteomyelitis of the bone.

infection in these regions, and, therefore, a complete physical examination is essential. However, some foals will recover completely from the initial infection only to develop bone or joint infections several days later when they appear to be very healthy. The most common bacteria that cause hematogenous infections in foals are Gram-negative enterics such as *Escherichia coli*.[7,29] Other causative organisms include *Staphylococcus* spp., *Streptococcus* spp., *Rhodococcus* spp., and *Salmonella* spp.

Traumatic Osteomyelitis

An open fracture or a penetrating wound may lead to osteomyelitis in any age horse (Figures 7.40 and 7.41). There is usually some degree of trauma to the skin and surrounding soft tissues with these injuries, and the pathogenic organisms may directly enter the medullary cavity through the open wound. Bacteria associated with these types of infections include Gram-negative enterics, *Staphylococcus* spp., *Streptococcus* spp., and anaerobes.[7,117] Infection spreads through the bone in a similar manner as for hematogenous osteomyelitis. Occasionally there is no overt break in the skin, but the necrotic tissue provides a medium for bacterial proliferation and infection develops from a hematogenous route. Avascularity is a major factor in the pathogenesis of osteomyelitis, and therefore, fractures with bone fragments isolated from a blood supply will be at risk to develop infection.[7] Treatment of an open fracture without providing absolute stability is usually futile unless the dead avascular fragments can be removed or reincorporated into the healing fracture where they can be revascularized.[119] Alternatively, some type of external fixator may be used to stabilize the fracture without disrupting the soft tissues around the fracture site.[69,80,82] In this manner, the potential for osteomyelitis is decreased because no implants are placed near the fracture, and the vascularity to the fracture is not further impaired.

Iatrogenic Osteomyelitis

The cause of osteomyelitis following internal fixation of fractures is usually due to contamination from an open wound (open fracture). Regardless of the type of internal fixation utilized, infection following repair of open fractures is much more common than following repair of closed fractures. However, contamination of the fracture during the surgical procedure can and does occur, particularly if it is prolonged (greater than 3 hours).[7] Usually the fracture hematoma and avascularity at the fracture site and the implantation of foreign material (pins, plates, screws, etc.) contribute to the development of osteomyelitis, because they provide favorable conditions for bacterial growth. Once bacteria become

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established where nutrients are available, proliferation occurs within a polysaccharide slime, forming a biofilm-enclosed colony. This biofilm or bioslime is formed by bacterial extracapsular exopolysaccharide that binds to surfaces of the implants and help maintain infection by protecting the bacteria from the host defenses. Because of this, osteomyelitis may develop despite prophylactic antibiotic coverage at the time of fracture repair especially in open fractures. Highly resistant bacteria such as methicillin-resistant *Staphylococcus aureus* or Gram-negative enterics often cause these types of infections.[7]

Clinical Signs of Osteomyelitis

Hematogenous osteomyelitis may be missed in its early stages and often presents after the lameness has become unresponsive to medical therapy. Frequently the owner feels that the lameness was due to an injury such as a sprain or being stepped on by the mare. There is usually a very severe lameness with cellulitis similar to that seen with fractures. Pain is usually elicited with direct pressure and manipulation of the joint(s) adjacent to the infection, and a fever is commonly seen in foals. Clinical signs of traumatic or iatrogenic osteomyelitis are similar and include lameness, soft tissue swelling, and retarded wound healing over the implants, drainage, and fistulation. Signs can be present as early as 7–10 days of injury or surgery or may be delayed for 3–4 weeks. Bloodwork usually demonstrates most consistently an increase in fibrinogen and occasionally increased white blood cell count. Serum amyloid A is often increased dramatically in these cases.

Radiographic Signs of Osteomyelitis

Loss of bone density due to a reduction in the calcium salt content of the bone occurs gradually with osteomyelitis. Lytic changes in the bone are not visible until 30%–50% of the bone mineral has been removed.[89] This is usually evident 10–14 days after the onset of infection (Figure 7.42). In more chronic cases there are often sclerotic margins around the lytic regions due to new bone formation. Sequestrum formation with a surrounding envelope (called the involucrum) and endosteal and periosteal thickening may also be evident. Occasionally osteomyelitis may penetrate into an adjacent joint, producing signs of a septic arthritis. With osteomyelitis following fracture repair, blurring of the cancellous trabeculation and a "moth-eaten" appearance at the fracture site will be seen on radiographs.[119] Lysis along the screw threads or under the plate may become evident with time. Usually a piece of bone that is decalcified and surrounded by a lucent zone is a sign of sequestrum formation. A zone of bone destruction adjacent to the implants typically occurs under the plate and directly along the screw threads. In more chronic cases of osteomyelitis, zones of both bone production and destruction are visible radiographically.[7]

Diagnostic Ultrasound

Diagnostic ultrasound is often helpful in characterizing fluid overlying an implant as a sign of infection. Aspiration of the fluid can be facilitated with ultrasound for culture and sensitivity. Changes in the ultrasonographic appearance can be monitored to dictate the appropriate course of therapy.



Figure 7.42. Osteomyelitis after internal fixation of a fractured third metatarsal bone. Lysis of the bone is occurring under the plate at the fracture site (arrow).

Treatment

The lameness in many foals with hematogenous osteomyelitis is frequently attributed to trauma but is, in fact, the early stages of infection. Therefore, at the time of initial examination, the infection is often well advanced with or without obvious radiographic signs, making it difficult to treat medically.[7,96] However, if acute hematogenous osteomyelitis is suspected in foals despite no radiographic signs, broad-spectrum bactericidal antimicrobials should be administered. The duration of antimicrobial use is largely empiric (usually minimum of 3 weeks) and should be based on the clinical response of the animal. However, antimicrobials alone may be unsuccessful due to the ischemic nature of the disease and poor penetration of the antimicrobial into infected avascular bone. Antimicrobials are best used early in the course of the infection and at high doses. Antimicrobials used most commonly to treat horses and foals with osteomyelitis include penicillin, gentamicin, amikacin, ceftiofur, cefazolin, enrofloxacin, and vancomycin. Nonsteroidal anti-inflammatory drugs (NSAIDs) have been beneficial

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to treat acute osteomyelitis in humans and animals in conjunction with antimicrobials.[119] Many times the pain is due to acute synovitis from infection spreading to an associated joint, and lavage of the joint can provide significant pain relief and treatment.

If there is no response to medical therapy and/or the osteomyelitis is localized, then surgery in conjunction with medical therapy is recommended. If the lesion can be accessed through a joint, which is often seen with hematogenous infections, then arthroscopy should be used to remove the damaged bone. A sample of the bone should be obtained at surgery and submitted for culture and sensitivity testing. Debridement of infected fractures or open wounds should be performed to remove avascular bone and infected soft tissue and to decrease bacterial numbers. Cancellous bone grafting is often utilized with infections of fractures to speed fracture healing. However, a major priority for treatment of osteomyelitis associated with fractures is to achieve stability of the fracture. Stability needs to be maintained not only for fracture healing but also to limit the spread of infection.[119] Loose implants should be removed, and fracture stability achieved by other means such as re-plating, external fixators, interlocking nails, external immobilization, or a combination of these techniques. If it is impossible to stabilize the fracture either with internal fixation, external fixation, or both, euthanasia may be the only alternative since it is unlikely that the infection will resolve.

Other methods used to treat osteomyelitis include regional perfusion of antimicrobials directly into the medullary cavity of the bone or within the vascular system of the limb.[127,129,130] The goal is to obtain very high tissue concentrations of antibiotics to achieve better bacterial kill. A tourniquet should be placed above and below the site of infusion and maintained for a minimum of 20–30 minutes to achieve optimal results.[7,127–129] In addition, antimicrobial-impregnated PMMA may be placed locally into the wound to achieve high antibiotic concentrations in and around the fracture.[40,116] The antimicrobials are incorporated into the PMMA during mixing and will elute from the PMMA into the wound for several days to weeks after they are placed. The PMMA is usually molded into bead or cigar shapes and placed adjacent to the implants, fracture, or site of infection.[40] The use of antibiotic-impregnated PMMA is thought to greatly improve our ability to successfully treat iatrogenic osteomyelitis in horses such as those associated with internal fixation.[40]

Prognosis

The prognosis for foals with hematogenous osteomyelitis is variable but is usually poor if multiple sites are involved. However, if the site of osteomyelitis can be thoroughly debrided, then the infection can usually be resolved. The prognosis with traumatic osteomyelitis is also variable depending on the bone involved and the duration and severity of the infection. Traumatic osteomyelitis is usually less difficult to resolve than iatrogenic infections. Osteomyelitis following internal fixation of a fracture is one of the most difficult diseases to successfully resolve in horses.[7,40] Therefore the prognosis for these animals is extremely guarded particularly for adult horses.

Infectious Physitis

Hematogenous infections of the physes are not uncommon in foals and have similar predisposing factors as other bone and joint infections in foals. The physis is often the initial location of hematogenous bacteria and infection, which can subsequently spread to the neighboring epiphysis and joint (Figure 7.37). Multiple physes can be infected simultaneously, but this is uncommon. Clinical signs, radiographic findings, and treatment are similar to other bone infections. However, foals with primary infectious physitis without accompanying septic arthritis might demonstrate a stiff gait at most in multiple limbs. Pinpoint palpation over the physis can oftentimes elicit a significant pain response, making radiographic evaluation critical to early diagnosis.

Bone Cysts

True bone cysts (here defined as those other than SCLs) that occur in other species (primarily aneurysmal and unicameral) have been reported to occur rarely in horses.[8,45,112] The most common site appears to be the mandible. Unicameral bone cysts are defined as solitary intraosseous cysts lined by thin connective tissue membranes.[45] Aneurysmal bone cysts are defined as expansile lesions consisting of anastomosing cavernous spaces filled with unclotted blood and lined with fibrous walls of varying thickness.[61]

These cysts usually contain osteoid tissue or osseous components, without elastic laminae or muscle layers.[61] Aneurysmal and unicameral bone cysts are more characteristic of true cystic lesions since they do not involve an articular surface and are usually solitary, expansile, intraosseous lesions. The majority of these true bone cysts reported in dogs and people occur in the distal or proximal metaphyses of long bones.

The cause of aneurysmal and unicameral bone cysts in any species is uncertain. Unicameral cysts are thought to result from the encapsulation and alteration of a focus of intramedullary hemorrhage supposedly from trauma. Alternatively, trauma results in a disturbance in endochondral ossification, resulting in a cystic defect within the metaphysis.[15] Aneurysmal bone cysts are generally believed to develop secondary to a preexisting lesion such as fibrous dysplasia, hematoma from trauma or bleeding disorders, or neoplasia.[113] An aneurysmal bone cyst of the distal metaphysis of the metatarsus in a horse was thought to be caused by trauma.[112] Whether the pathogenesis of aneurysmal and unicameral bone cysts and SCLs are interrelated is controversial. Most of the evidence (including pathogenesis and clinical characteristics) suggest that SCLs, at least in horses, are a distinctly separate clinical entity from aneurysmal or unicameral bone cysts.[6] Additionally, aneurysmal and unicameral bone cysts can be difficult to differentiate from some bone tumors in horses (Figure 7.43). Treatment of true bone cysts in horses usually involves surgical curettage of the lesion with or without autogenous cancellous bone grafting. Spontaneous resolution of the cyst may also occur, and some bone cysts in people respond to intralesional steroid injections.[61] The prognosis for resolution of bone cysts in horses is usually good depending on the location of the cyst.

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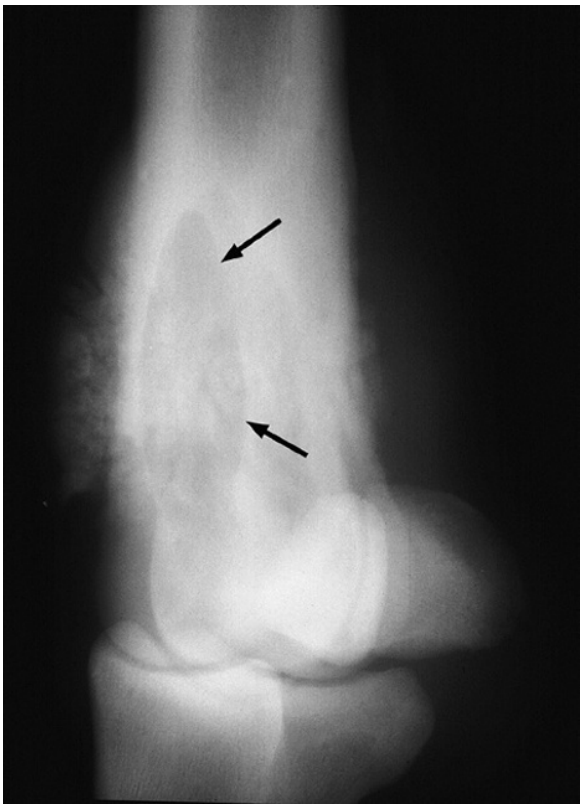


Figure 7.43. Oblique radiograph of the metatarsus in an adult horse with firm swelling of the metatarsus and mild lameness. Radiographs reveal a large lytic lesion within the bone with extensive periosteal new bone growth suggestive of neoplasia (arrows). The lesion was debrided and a biopsy suggested a possible osteosarcoma. However, this horse continued to do well with minimal progression of the lesion. Therefore, the lesion may be a true bone cyst and not a bone tumor.



Figure 7.44. Large, firm swelling of the rostral aspect of the mandible in a young Quarter horse filly that was consistent with an ossifying fibroma (arrow). A rostral mandibulectomy was performed to completely remove the tumor.

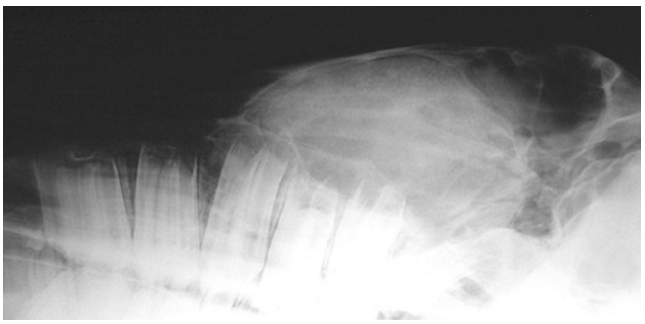


Figure 7.45. A lateral radiograph of the paranasal sinus region of a 3-year-old Quarter horse gelding revealed the presence of a large osseous mass within the maxillary sinus. A biopsy of the mass suggested a diagnosis of multilobular osteochondroma.

Bone Tumors

Primary bone neoplasia is rare in horses. The most common bone tumors that occur in horses include osteoma, ossifying fibroma, multilobular osteoma or osteochondrosarcoma, and osteosarcoma.[36] Although these tumors may develop at any location, the face and head (mandible, skull, and paranasal sinuses) appear to be the most common sites of occurrence. Osteomas are benign tumors that consist of well-differentiated bone that typically occur within the paranasal sinuses or on the skull. Ossifying fibromas are benign fibro-osseous lesions that usually involve the rostral aspect of the mandible, premaxilla, or paranasal sinuses (Figure 7.44).[36] Multilobular osteomas or osteochondrosarcomas are very rare in horses but usually involve the skull or paranasal sinus (Figure 7.45). These tumors are generally benign but locally aggressive and can be removed surgically. Osteosarcomas are malignant tumors, arising from mesenchyme, in which neoplastic cells produce osteoid or bone. In horses, the skull, axial skeleton, ribs, and occasionally the limbs are commonly affected sites. The clinical signs caused by these tumors will depend on the location and specific type of neoplasia. The diagnosis of neoplasia is usually made based on its radiographic appearance and is confirmed by histopathology of biopsy or autopsy specimens. Treatment will depend on the location of the tumor, involvement of the parent bone, and the specific type of osseous tumor present. In general, all tumors except osteosarcomas can potentially be removed successfully with minimal recurrence if complete tumor excision is achieved.[36] Unfortunately complete tumor excision is not always possible.

Bone Contusion/Bruising/Periostitis

Bone contusions from direct or indirect trauma are known to occur in people and contribute to orthopedic pain and lameness. The majority of joint pain in people is thought to originate from bone and is typically seen as bone marrow lesions of the subchondral bone with MRI.[53] With the amount of trauma that horses seem to encounter, bone bruising probably occurs much more often than we currently recognize or are able to diagnose. In fact, many of the retrospective MRI studies of athletic horses are demonstrating bone marrow lesions to be common and in many instances might be the initial pathologic lesion leading to joint or enthesis disease.[53] Diagnosing bone contusions and bruising in

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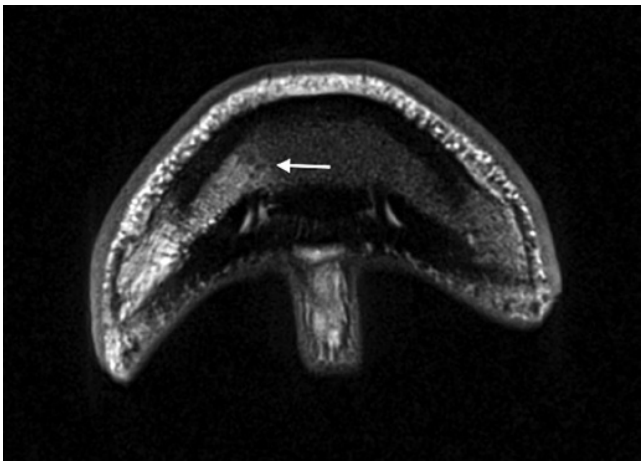


Figure 7.46. MR image of the distal phalanx demonstrating marrow edema (arrow). *Source*: Courtesy of Dr. Kurt Selberg.

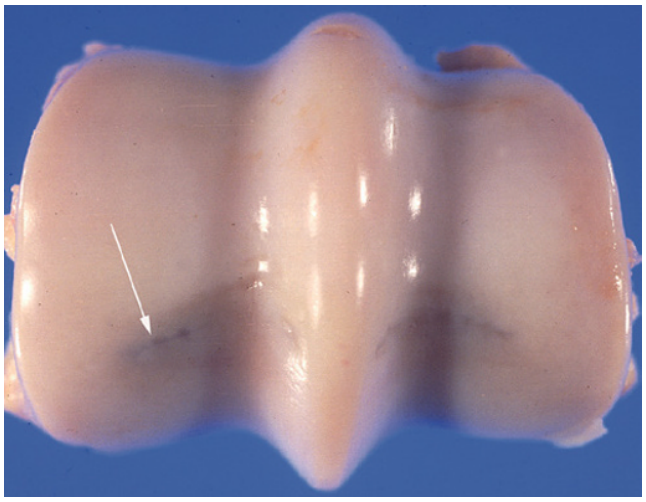


Figure 7.47. The distal aspect of the third metacarpal condyles typically shows subchondral bone bruising in racing Thoroughbred horses (arrow).

horses is very difficult and mostly subjective. This is especially true for bone pain originating in joints. Most bone contusions or bruises are diagnosed based on the history, clinical findings, and lack of radiographic abnormalities. However, MRI is now used routinely to diagnose bruising and edema within bone (Figure 7.46). With some bone contusions of cortical bone, evidence of pain can be elicited with direct pressure over the affected site. In addition, bone pain can sometimes cause more severe lameness than most soft tissue injuries, and pain is often elicited with manipulation of the affected bone or joint. Radiographs are useful only to document the absence of fractures or other abnormalities within the affected bone or joint. Nuclear scintigraphy would be useful to document abnormal bone metabolism of the affected site, but cannot be used to definitively diagnose a bone bruise or contusion. MRI and computed tomography (CT) are used in people to help diagnose bone damage/contusion and are available for use in horses at referral hospitals.[66,73,108] Positron emission tomography (PET) has also been used to identify potential lesions, but more work is needed on this modality.[109,110] Treatment of bone contusions would be similar to any type of acute musculoskeletal trauma and would include reduced activity, cold therapy, hydrotherapy, bandaging, and NSAIDs. Suspected bone contusions/bruising within joints could also be treated with intra-articular medications; however, they are somewhat limited in their effectiveness. Systemic bisphosphonate therapy has been advocated for treating bone bruising and edema; however efficacy has not been proven in horses.[50] The prognosis of horses with bone contusions is usually very good unless damage to the subchondral bone of a joint contributes to joint pathology and secondary OA (Figure 7.47).

SYSTEMIC DISEASES OF BONE

Osteoporosis

In osteoporosis, the bone mineral density (BMD) of the bone matrix is reduced. The bone becomes porous, light, and fragile and is prone to fracture. Osteoporosis in horses is an uncommon or at least an uncommonly recognized clinical problem. The term osteopenia is used if BMD is reduced, but spontaneous fractures do not occur. Clinically, osteopenia is much more commonly recognized in horses than osteoporosis.

Generalized Osteoporosis

The generalized osteoporosis seen in postmenopausal women has no counterpart in the aged horse. However, older mares may be more prone to long bone fractures during recovery from general anesthesia. Whether this may be due to declining estrogen levels associated with reproductive senescence is unknown. Osteoporosis is seen occasionally with undernutrition rather than actual deficiencies of calcium, phosphorus, or vitamin D. However, in most affected horses, osteoporosis is usually associated with a diet low in calcium, high in phosphorus, or low in vitamin D. Osteoporosis is associated with copper deficiency and chronic lead poisoning in lambs, but this has not been seen in foals.[9] A condition first recognized in Thoroughbred foals may be a manifestation of generalized osteoporosis.[26] The condition is characterized by fractures of the proximal sesamoid bones and typically occurs when foals gallop to exhaustion trying to keep up with their dams. Foals seem more prone to the condition if they are confined after birth. During this time of relative inactivity, the bones are not subjected to the stresses required to strengthen them and are a potential weak link in the skeletal system (Figure 7.21). Some underlying metabolic problem or deficiency producing osteoporosis may exist, but at this time it is undefined. Other lameness problems and unexplained fractures in horses may be attributed to generalized osteoporosis, but their pathogenesis and cause are often unexplainable. The combination of age and pregnancy have also been shown to negatively influence bone strength,[37] with increased age and parity leading to significantly decreased bone strength.

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Localized Osteopenia (Disuse Osteopenia)

This condition is fairly common, especially in horses following rigid external immobilization of their limbs (casting). Disuse osteopenia may also occur in horses with severe or chronic lameness (Figure 7.48) or neuropathies such as radial nerve paralysis where weight-bearing is reduced. With decreased weight-bearing, there is increased resorption of bone and decreased bone formation. Immobilization of the thoracic limb of a pony in a cast for 6 weeks caused a significant decrease in weight and specific gravity of the third metacarpal bone.[25] Histologically, osteopenia was caused by atrophy of osteoblasts with failure of bone apposition.[25,119] However, external immobilization of the distal limb in horses has also been shown to have only a minor effect on articular cartilage with very little clinical significance.[93] If horses are brought back into work too quickly, distal sesamoidean ligaments can tear from the distal aspect of the proximal sesamoid bones, leading to significant lameness (Figure 7.21). Osteopenia is usually more severe in young animals due to the inherent rapid bone turnover compared with the adult horse. Bone cortices become thinner and more lytic. Although this is thought to rarely contribute to a clinical problem and is easily reversed when the external immobilization device is removed and the animal commences normal weight-bearing, recent evidence suggests that the pathologic changes can be significant.

Localized osteopenia is essentially a radiographic diagnosis characterized by lack of cortical density and a more lucent appearance to the bones compared with normal (Figure 7.49).[119] The sesamoid bones are often the first bone to manifest the problem radiographically. Loss of bone that can be seen radiographically indicates an approximately 30% BMD reduction.

Further experiments have shown that treatment of osteopenia with 25-hydroxycholecalciferol has been beneficial.[25] Fortunately, localized osteopenia following immobilization rarely causes any problems. If the external



Figure 7.48. A lateromedial radiograph showing a comminuted fracture of the femur in a 23-year-old mare. This mare had severe lameness in the stifle of that limb since she was a yearling and fractured it while getting up in her stall. It is likely that the bone was osteoporotic due to chronic, reduced weight-bearing from the lameness.

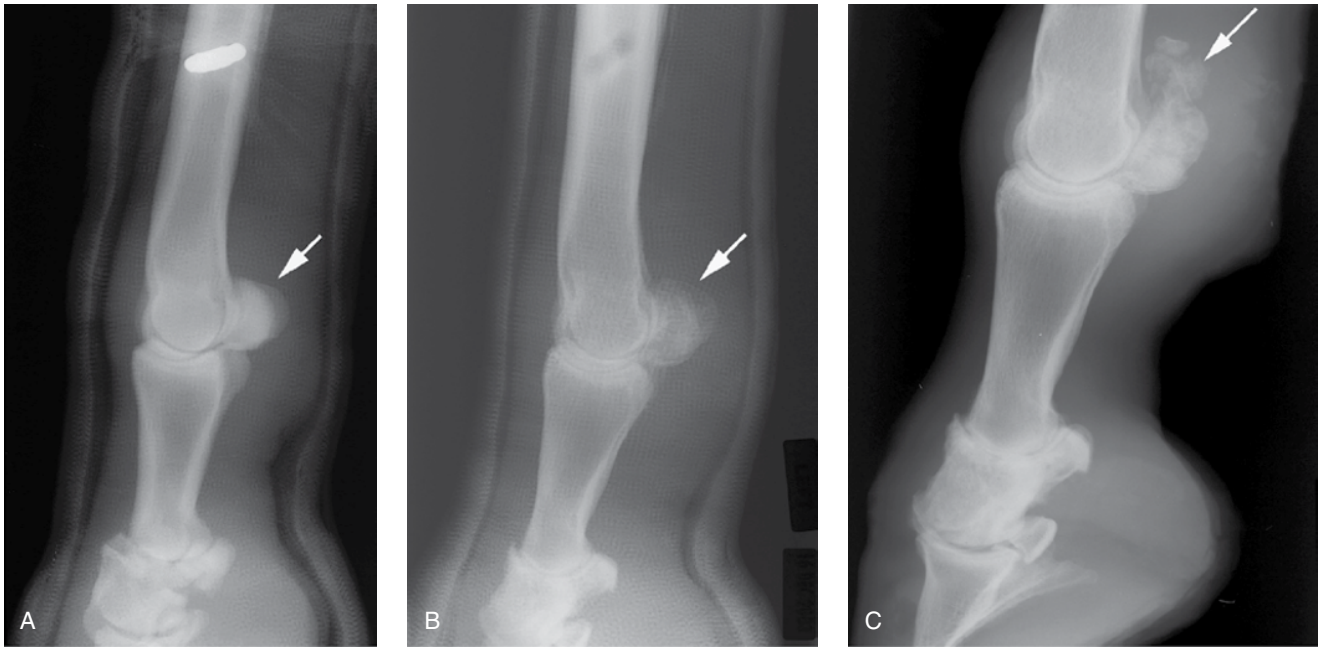


Figure 7.49. Radiographic images of a comminuted P2 fracture that was treated with a transfixation pin cast. (A) At the initial pin placement. (B) When the pins were removed 3 months later. (C) Five months after pin removal. Note the progression of disuse osteoporosis of the sesamoid bones (arrows in A, B, and C) and distal limb with subsequent fracturing of the sesamoid bones (C, arrow).

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support is suddenly removed, however, a pathologic fracture may result. To prevent this, the rigidity of the external support should be diminished gradually over time. For example, following cast removal, a cotton bandage with a PVC splint incorporated into the bandage should be used to provide less stability than a cast but more support than a bandage alone.[119] Following removal of the splint, a lighter, less rigid bandage should be used until eventually no external support is applied to the limb. It may take several months before the bone regains normal density and strength following external immobilization. Occasionally, the original BMD is never reached.

Stress protection is another example of localized osteopenia seen in fractured bones that have been repaired with rigid internal fixation devices such as stainless steel bone plates.[119] Initially, rigid internal fixation enhances healing and permits mobility of neighboring joints. Over a period of months, however, the bone under these plates becomes lytic. This produces a weakened bone with the possibility of a fatigue fracture when the plate is removed. Theoretically, plates should be removed as soon as healing has occurred. Plates with a lower modulus of elasticity have been designed to minimize stress protection, especially in people and small animals. In horses, however, osteopenia generally does not occur to the same degree because a much larger animal places a greater force on the healing bone. Therefore, stress protection due to internal fixation is of minimal clinical significance in horses. In general, bone plates and other forms of internal fixation are not removed in horses unless they contribute to infection or cause lameness in performance horses, and when they are removed, they are usually done so in a staged fashion.

Bone Fragility Disorder

Bone fragility disorder has been recently characterized by Anderson et al.[1] These horses typically present with variable degrees of lameness that is difficult to regionalize on physical examination. The key diagnostic finding is that the horses have multiple sites of radioisotope uptake, mostly in the axial and proximal appendicular skeleton, and occasionally radiographic and ultrasonographic signs of bone remodeling at various sites. Rest appeared to help some horses, but most were euthanized for humane reasons in the report.[1] Bisphosphonate therapy, namely, zoledronate, has shown some efficacy in treating signs of disease.[51]

Osteopetrosis

Osteopetrosis is a rare skeletal disease of horses characterized by an imbalance of bone apposition and resorption. It is an inherited disease of people, rabbits, mice, and cattle and may be an inherited disease in horses.[104] The underlying problem is a failure of bone resorption by osteoclasts. There is complete closure of the medullary canal at the mid-diaphysis of the bone because the canal has not been remodeled by osteoclasia during development from its embryologic state. The disease is also characterized by fractures due to a lack of a normal bone structure. Since there is no evidence of bone marrow in such bones, an anemia commonly accompanies this condition. Osteopetrosis has been reported in a Peruvian Paso foal,[104] and it may be an inherited condition in this breed. In the case described by Singer and Whitenack,[104] metaphyseal diameters of long bones were wider than normal, and the cortices were abnormally thick with complete obliteration of the medullary canal. Trabecular bone was soft and could be easily crushed, and the bones were not as strong as normal bones in an animal of this age. Large calluses were formed at fracture sites, suggesting that such fractures had occurred *in utero*. Because of the inherited nature of the disease in other species, owners should be advised against future matings.[104]

Hereditary Multiple Exostosis (Multiple Cartilaginous Exostosis, Osteochondroma)

Hereditary multiple exostosis is an inherited skeletal disorder characterized by numerous abnormal projections from growing bones that result in an abnormal bone contour.[75,102,103] The condition affects most of the long bones as well as the ribs, scapula, and pelvis in horses and is used as a model for the condition in man.[103] The characteristic swellings of hereditary multiple exostosis are usually present at birth, and the lesions are probably initiated during fetal osteogenesis. They are usually bilaterally symmetrical and consist of multiple firm bony enlargements of various shapes and sizes that are firmly attached to bone.[103] Swellings on the limbs do not appear to enlarge as the animal matures, but others such as those located on the ribs and scapulae usually enlarge until maturity is reached (about 4 years of age). Lameness, if present, is usually due to impingement of various tendons and muscle groups by the bony masses. Some horses may be presented for various joint and tendon sheath swellings.[75] Grossly the tumors adopt a variety of shapes, ranging from conical, rounded, pedunculated, multilobulated, or spur-like. Histologically such tumors appear as osteochondromas and do not appear to undergo malignant transformation.[103] They usually have a small cartilage cap covering underlying spongy cancellous bone. There is no known treatment for this condition.

Solitary osteochondromas occur more commonly in horses and usually develop on the caudomedial aspect of the distal radial metaphysis.[56,107] Solitary osteochondromas are not considered to be an inherited condition like hereditary multiple exostosis. Horses with osteochondromas on the caudal aspect of the radius usually present for lameness and swelling of the carpal canal. The masses resemble hereditary multiple exostosis, but the lesions are usually not symmetrical.[60,107] Radiographic examination usually reveals an osteocartilaginous exostosis protruding from the caudal aspect of the radius. Large exostoses may cause lameness by interfering with muscle movement such as the humeral head of the deep digital flexor muscle or pain from carpal canal effusion.[60,107] Carpal sheath effusion is usually due to the exostosis and usually will not resolve unless the bone is removed. Surgical removal of the lesion via tenoscopy of the carpal canal is now commonly performed.[107] Solitary osteochondromas may also develop in joints associated with previous trauma or surgery. Small dislodged pieces of cartilage may become trapped in the synovium,

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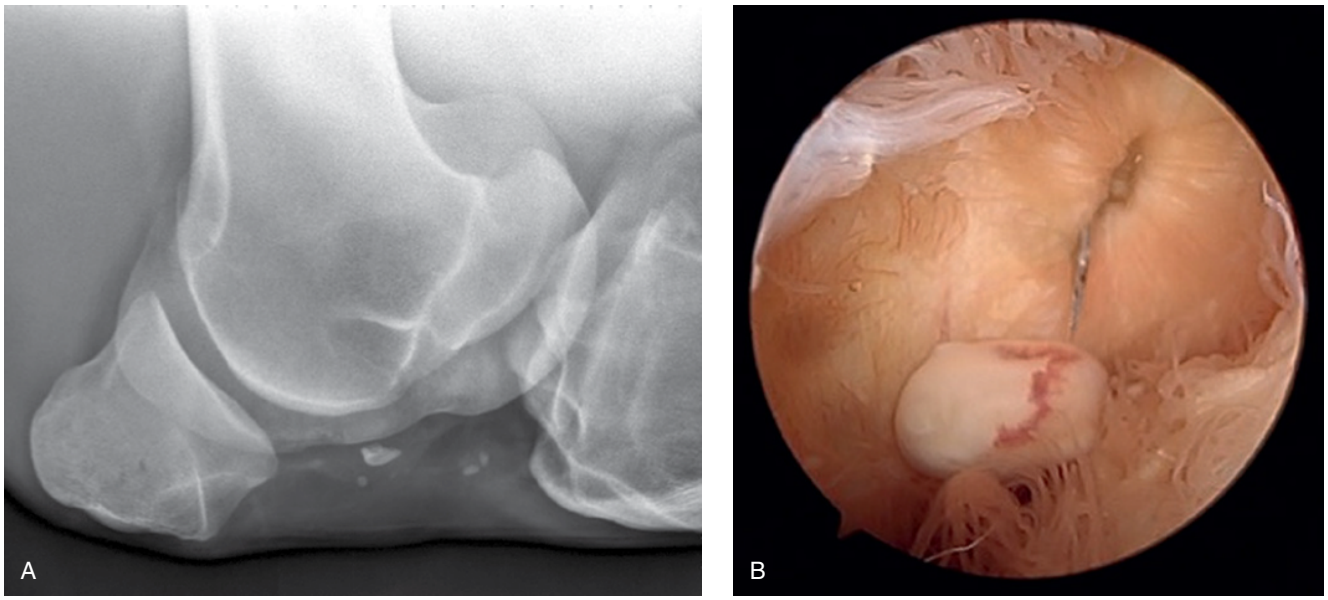


Figure 7.50. Flexed lateral to medial radiograph (A) and arthroscopic view (B) of an osteochondroma within the femoropatellar joint. The osteochondroma was a loose body within the joint and was likely contributing to joint effusion in this horse.

develop a blood supply, and grow to form osseous masses. These may or may not cause a clinical problem, but will often lead to persistent synovial effusion. The dorsal aspect of the fetlock and the femoropatellar joint appear to be particularly susceptible to osteochondroma formation (Figure 7.50).

Tumoral Calcinosis (Calcinosis Circumscripta)

Tumoral calcinosis is the formation of calcified, granular, amorphous deposits in the subcutaneous tissues that induce a fibrosing granulomatous reaction.[113] The deposits usually occur in the subcutis near joints and tendon sheaths.[22,34] It occurs infrequently in the horse although it may be more common than is actually recognized.[34] The etiology of the condition is unknown. Affected horses are usually presented for unsightly swellings that are becoming progressively larger, and lameness is uncommon. The swellings are firm and painless, and the skin is usually intact and movable over the swellings. The most common location for tumoral calcinosis lesions is the lateral aspect of the stifle, lateral to the fibula, and beneath the aponeurosis of the biceps femoris and lateral crural fascia. Of 18 cases reported in the literature, lesions occurred over the lateral surface of the tibia close to the femorotibial joint in 16 horses.[34] Radiographically, the lesions are characterized by radiopaque calcified deposits. On cut surface of the lesions, there is a honeycomb-like appearance with a calcareous, gritty deposit enclosed in a dense fibrous capsule.

The treatment for calcinosis circumscripta is surgical excision. This should be performed only in cases in which lameness can be directly attributed to the lesion. The lesion may be so firmly attached to the stifle joint capsule that it is impossible to dissect it free without opening the joint. Therefore, surgical excision of these lesions should be performed cautiously.

Osteodystrophia Fibrosa (Nutritional Secondary Hyperparathyroidism)

Osteodystrophia fibrosa or nutritional secondary hyperparathyroidism is a generalized bone disease caused primarily by a dietary calcium deficiency in the face of phosphorus excess.[18] Due to awareness and sensitivity about the condition, it is rarely seen today. It occurs in all equids, although the horse is more susceptible than its relatives.[49] It is most common in horses being fed cereal and cereal by-products such as bran (diets high in phosphorus and low in calcium), hence the name "bran disease." Addition of legume hay, which is high in calcium, to the diet can usually prevent the disease.[18,72] It is also seen in horses grazing plants high in oxalates, which chelate the calcium and interfere with the absorption of calcium. Horses in Queensland, Australia, developed osteodystrophia fibrosa when grazed on tropical grasses.[119] A subclinical form of the disease may also occur but is difficult to diagnose because the clinical manifestations are subtle.

The underlying pathogenesis in osteodystrophia fibrosa is defective mineralization of bone. The diet high in phosphorus leads to increased absorption of phosphorus and elevation of serum phosphate levels. This tends to lower serum calcium and stimulate the parathyroid glands to increase secretion of parathyroid hormone. Parathyroid hormone increases activation of remodeling, leading to resorption of bone. With bone resorption there is a compensatory replacement with fibrous tissue.[30] This causes poorly mineralized bone that is eventually replaced with cellular connective tissue. Horses of both sexes and all ages are susceptible. Lactating mares and foals appear to be at increased risk to develop osteodystrophia fibrosa.

The classic form of osteodystrophia fibrosa is called "bighead" because of the predilection of the jaws and flat bones of the skull to respond to parathyroid hormone.[31] The classic clinical signs of the disease include a

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symmetrical enlargement of the mandible and facial bones. There is loss of lamina dura around the teeth due to osteoclastic resorption of alveolar margins, and the teeth may eventually loosen. This can be identified radiographically and is one of the first signs of the disease. Swelling initially begins just above the facial crest and in the mandible producing a reduction in intermandibular space. There may also be enough swelling of the palate, maxillae, and incisor bones to produce dyspnea.

The subclinical form of nutritional secondary hyperparathyroidism may cause a nebulous shifting limb lameness in horses that is difficult to localize with standard perineural anesthesia.[119] The condition may also contribute to unexplained fractures especially in young horses. In such cases a dietary history is important in the diagnosis. Blood calcium and phosphorus levels are usually normal, and the diagnosis is usually based on analysis of the diet and response to treatment with calcium supplementation.

Hypertrophic Osteopathy (Hypertrophic Pulmonary Osteoarthropathy)

Hypertrophic osteopathy is a progressive bilaterally symmetrical proliferation of subperiosteal bone and fibrous connective tissue on the appendicular and axial skeleton and facial bones.[119] It is a relatively rare disease in the horse. The pathogenesis of hypertrophic osteopathy is still unclear at present. Classically the disease is associated with a space-occupying pulmonary lesion such as a neoplasm or chronic suppurative process such as a large abscess, tuberculosis, or a fractured rib with pleural adhesions.[67] The disease rarely occurs in horses with such thoracic lesions, but has been associated with a granular cell myoblastoma.[32] The term hypertrophic osteopathy is preferred because the disease has also been associated with intra-abdominal disorders without pulmonary involvement. Hypertrophic osteopathy has been reported in a mare with a dysgerminoma (a neoplasm of ovarian primordial germ cells) that had abdominal metastases but was free of thoracic lesions.[67]

The two most common mechanisms for the development of the disease are classified as neurogenic and humoral. The more popular neurogenic theory is based on the fact that an apparent stimulation of the vagus nerve produces an alteration of the vasculature and periosteum of bones by way of an unknown efferent pathway. Support for this theory is based on the fact that in dogs and man hypertrophic osteopathy lesions may regress following vagotomy. A humoral mechanism may also exist, since hypertrophic osteopathy has been shown to occur in people when urinary excretion of estrogen is increased. High levels of circulating estrogen have been reported in a mare with hypertrophic osteopathy, although the exact relationship between estrogen levels and hypertrophic osteopathy is purely speculative.[67]

The clinical signs of hypertrophic osteopathy are related to periosteal hyperostoses. There is symmetric enlargement of the long bones of the limb with all bones in the limb affected. There is pain and edema of soft tissues, and the horse may have a stiff gait and be reluctant to move. The joint surfaces are rarely involved; however, there may be decreased motion in affected joints as well as pain on manipulation. There may be signs referable to pulmonary abnormalities such as coughing and nasal discharge.

Radiographically, there is a generalized increase in soft tissue swelling and evidence of periostitis. Irregular new bone growth occurs, especially at the proximal and distal ends of the long bones (Figure 7.51).[119] The differential diagnosis should include fluorosis, but the gross appearance of the bones, absence of dental lesions,

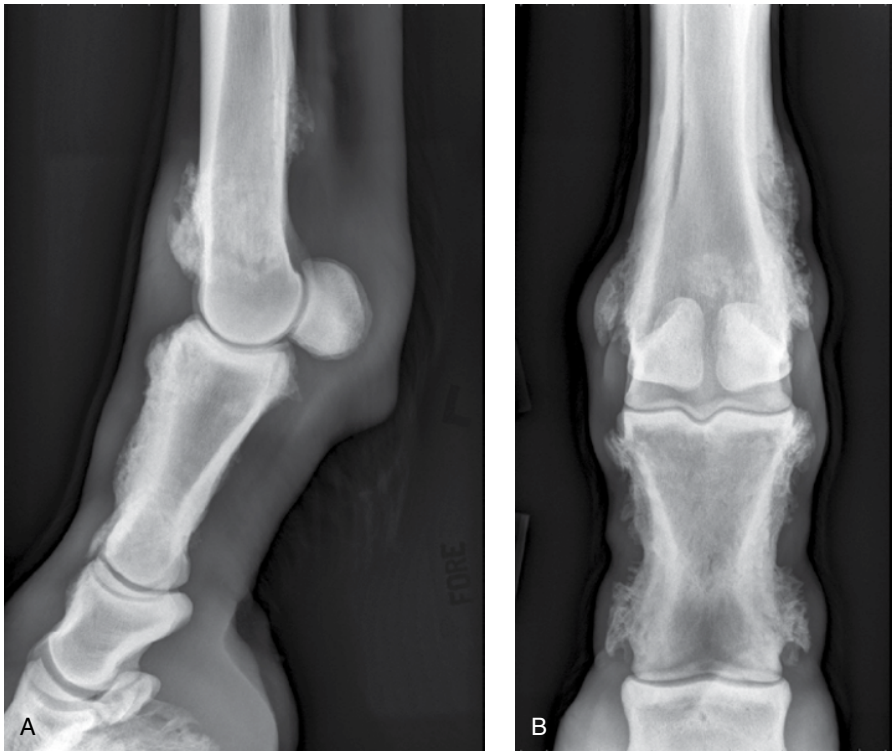


Figure 7.51. Lateral (A) and dorsopalmar (B) radiographs of the left front fetlock of a horse with hypertrophic osteopathy showing irregular new bone growth. A definitive cause was never determined.

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and blood and urine fluorine levels can usually eliminate fluorosis as the cause. Lateral radiographs of the chest or abdomen may help locate the causative lesion.

Effects of Medications on Bone

Recent research and development have demonstrated the effects of various medications on bone metabolism and bone healing. The two most investigated medication groups recently have been nonsteroidal anti-inflammatories and bisphosphonates.

Nonsteroidal anti-inflammatory medications (NSAIDs) are commonly used in horses to manage inflammation and the pain from a variety of sources. They are also used commonly to manage pain post-fracture and fracture repair. However, there has been work to show that NSAIDs can have a negative effect on bone healing, especially the "osteo-immunologic" cascade that is responsible for appropriate bone remodeling.[64] By reducing the inflammatory component of bone healing, a key signaling component is compromised, thus delaying bone healing. This has been demonstrated in several experimental models, including one study in the horse. Specifically, mineral apposition rate was reduced in horses administered phenylbutazone.[95] However, severe inflammation can impede bone and soft tissue healing, requiring significant anti-inflammatory therapy to overcome. The threshold at which NSAIDs should be used has not been determined.

Bisphosphonate therapy emerged on the equine market several years ago as a means of treating lytic lesions in the bones of horses.[50] Bisphosphonates act to inhibit osteoclast production and function. Tiludronate was initially marketed for equine use and shown in small clinical studies to be effective in managing pain in horses with caudal heel pain, bone spavin, and back pain.[17,20,33,74] The Freedom of Information report for approval of tiludronate in the United States shows that it had a positive effect on improving lameness by one grade in horses diagnosed with caudal heel pain.[120,121] Clodronate later emerged on the market, and it too showed ability to improve lameness in horses with caudal heel pain. However, the exact mechanism by which bisphosphonates work in horses is unknown. It is likely that they possess significant anti-inflammatory properties by which the action occurs, and the anti-osteoclastic effect has minimal clinical impact. Regardless of the therapeutic effects, there is concern about potential negative effects on young horses and the normal bone remodeling cascade that is necessary for physical adaptation to training. The AAEP has produced a white paper concerning the suggested use of bisphosphonates in horses, and the FDA has regulated that they should only be used in horses older than 4 years of age.

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TENDON AND LIGAMENT INJURIES AND DISEASE

Laurie R. Goodrich

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ANATOMY

Tendons and ligaments are complex structures that exist in a hierarchical structure of subunits (Figure 7.52). Grossly, tendons are made up of many fascicles, which, during further macroscopic and microscopic examination, have decreasingly sized subunits, then fibers, and finally fibrils.

The anatomic structures of note are the paratenon, epitenon, endotenon, tendon fascicles, and tendon fiber/fibrils. Paratenon refers to the loose connective tissue and vessels that surround tendons. Epitenon is outside of tendon fascicles and is continuous with the endotenon. Endotenon contains vascular and neural structures and separate cell populations. Furthermore, the endotenon may be a source for pluripotential cells. Tendon fascicles are the bundles of fibrocytes and tenocytes that are surrounded by endotenon. Tendon fibers/fibrils are made of long bundles of collagen filaments that are predominately type I collagen and have elastin and glycosaminoglycan in the extracellular matrix. Larger fibrils are stronger than smaller fibrils.[32]

Collagen fibrils have many triple helical collagen molecules that are arranged such that a characteristic banding pattern is seen on electron microscopy. Collagen molecules are secreted extracellularly through the pores of the tenocytes. Cross-linking of these molecules results in fibril formation, and these fibrils fuse as horses age, leading to larger collagen fibrils.[17,21,32]

Under a light microscope, collagen fibers have a characteristic wavy pattern that is referred to as crimp (Figure 7.53). Crimp imparts elasticity to the tendon. A decrease in crimp occurs with aging along with a greater reduction in the central fibers.[27,32,43] When the tendon is stretched, the central fibers straighten primarily, and therefore a greater load is placed on these fibers relative to peripheral fibers. This may explain the more common occurrence of tendon lesions observed centrally (core lesions). Peripheral lesions are less clearly explained.

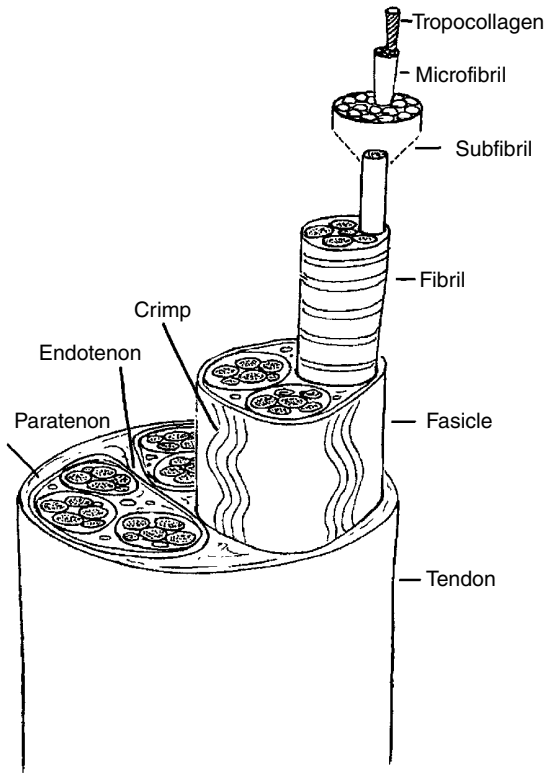


Figure 7.52. A schematic representation of the tendon hierarchy. The level of tendon fascicles can be viewed grossly, fibers may be seen microscopically, and individual collagen fibrils (tropocollagen) may be viewed via electron microscopy. *Source*: Modified from Davis and Smith.[12] Reproduced with permission of Elsevier.

Cellular Components

Tenocytes are the primary cell types found within equine tendon; they are responsible for the formation and maintenance of tendon tissue.[32] Three types are described (Figure 7.54). Type I cells have thin, spindle-shaped nuclei, type II cells have rounded or oblong nuclei, and type III cells appear as cartilage-like cells with round nuclei and visible nucleoli.[32] Proportions vary with age of the horse, site, and whether a ligament or tendon. Type I cells are more frequently found in older horses, type II cells can be found in higher numbers in young horses and in ligaments, and type III cells are found in areas sustaining higher compressive loads.[32] It is logical to assume that type II and III cells are metabolically active and maintain tendon extracellular matrix; however, type I cells most likely do this to some extent as well.

Cellular numbers vary depending on age and location within the tendon. As maturity is reached, the numbers remain constant; however, areas of tendon become acellular and have changes consistent with chondroid metaplasia such as the center of the superficial digital flexor tendon (SDFT) or the deep digital flexor tendon (DDFT) in the metacarpophalangeal region.[32] These areas are often surrounded by type II cells. Other cells found in tendon include synovial-like cells of the epitenon within the tendon sheaths and fibroblasts of the paratenon, epitenon, and endotenon. These probably have an integral role in tendon maintenance due to certain growth factors such as transforming growth factor-β (TGF-β) found in these areas.[8,18]

Tenocyte regulation has not been fully elucidated, but most likely it relies on mechanical and cytokine stimulation. Tenocytes have been shown to respond

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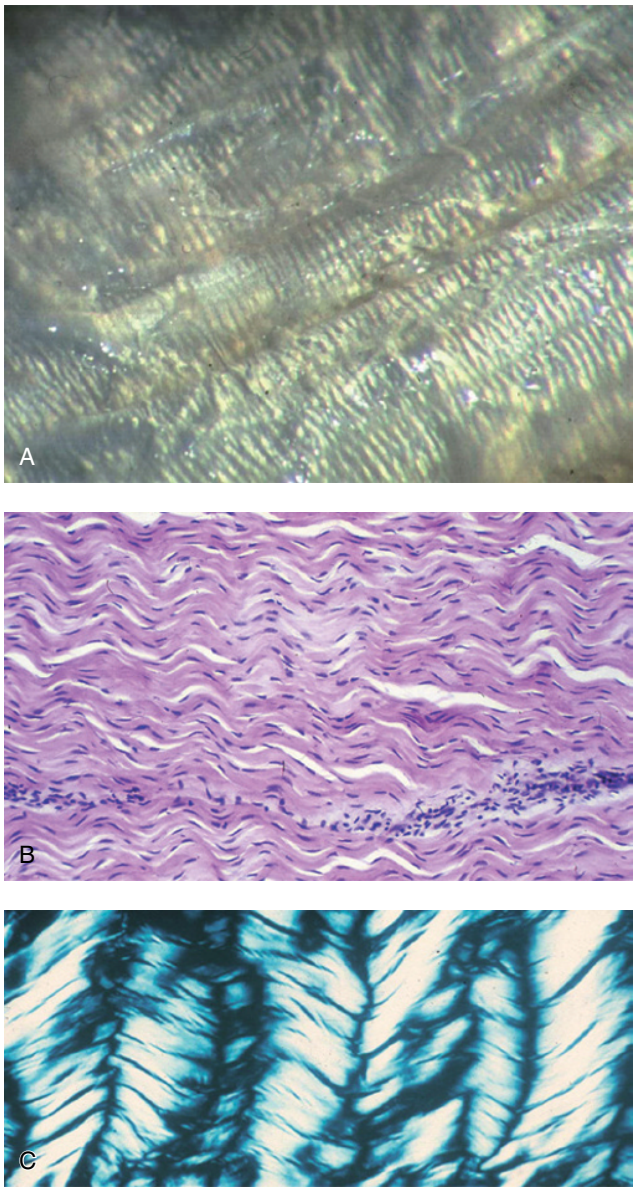


Figure 7.53. Gross (A) and hematoxylin- and eosin-stained microscopic section (B) and polarized light microscopy (C) revealing the classic crimp pattern formed by collagen fibers. *Source*: Courtesy of Dr. Roger Smith and Professor AE Goodship.

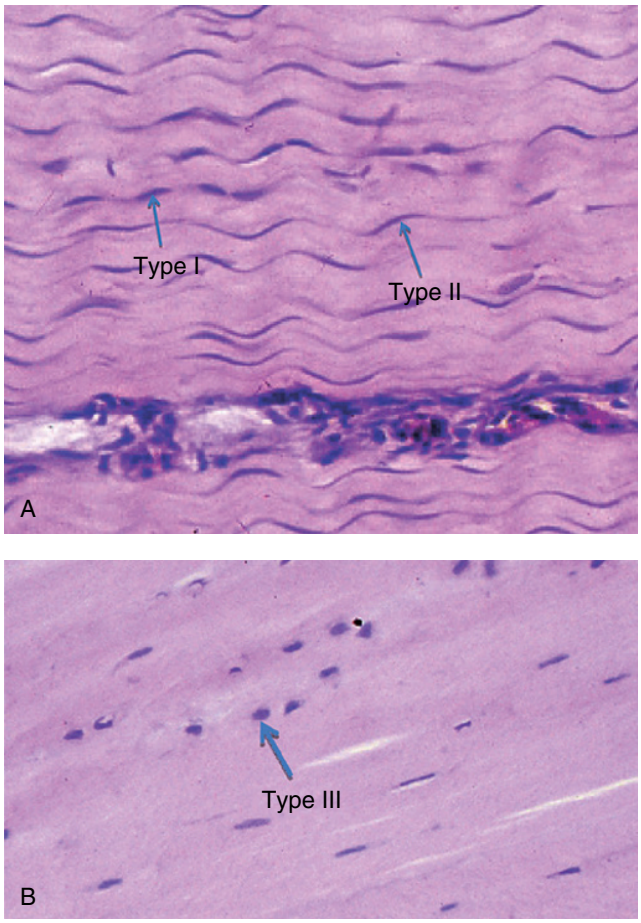


Figure 7.54. Type I, II (arrows in A), and III (arrow in B) cells found in the equine tendon. *Source*: Courtesy of Dr. Roger Smith.

quickly to mechanical stimuli in cell culture.[2] However, tenocytes in culture also require growth factors to respond to mechanical load.[17] Recent research has discovered cellular communication through cytoplasmic extensions of tenocytes, which most likely result in a coordinated upregulation in response to mechanical stimuli.[25,32]

The growth factors most noted to have anabolic effects on tendons include TGF-β and insulin-like growth factor-1 (IGF-1).[7,9] The concentration of these growth factors seems to be highest early in age and decreases with maturity. Furthermore, it is believed they are deficient in healing tendon. Further research will determine the important growth factors that are essential to tendon health and healing.

Molecular Composition

Tendon and ligaments are highly hydrated and have a water content of approximately 65% (wet weight), which makes up the majority of the extracellular matrix. The remaining content consists of collagen (30% wet weight) and noncollagenous glycoproteins (5% wet weight). Type I collagen is the predominant collagen in tendon (greater than 95%), and the remaining is type III, which is present in endotenon and increases with age. Type II collagen, the main collagen found in cartilage, can only be found at tendon insertions and areas where tendon changes direction and takes on a fibrocartilage-like character.[6] Collagen is synthesized outside of the cell as procollagen molecules. As the α-helical chains are produced and cleaved by procollagenases, they are cross-linked to form a collagen fibril.[12]

Other noncollagenous glycoproteins include cartilage oligomeric matrix protein (COMP), proteoglycans and elastin, fibronectin, and thrombospondin.[32,46] COMP has recently been studied intensely due to its perceived importance in organization of the collagen fibril framework during tendon formation and growth.[12] Recent data have shown that COMP accelerated collagen fibril formation *in vitro*.[37] As collagen molecules form a fibril, COMP does not remain bound and is displaced from the fibril. As such, levels of COMP increase during

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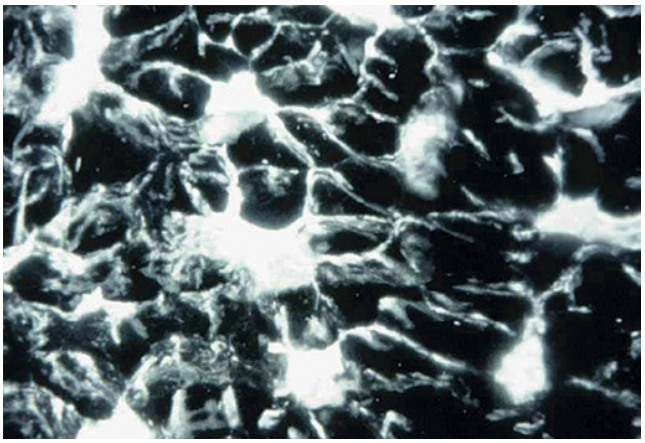


Figure 7.55. A photomicrograph of tenocytes and their extensions. The multiple connections allow the tenocytes to act in concert with mechanical influences. *Source*: From Davis and Smith.[12] Reproduced with permission of Elsevier.

growth, peak about 2 years of age in flexor tendons at the metacarpal level, and decrease once growth ceases and the animals age.[35] A correlation exists between tendon ultimate tensile strength and the level of COMP at maturity.[34]

Other noncollagenous proteins such as the small proteoglycans (decorin, fibromodulin, lumican) can be found in the regions of tension; conversely, large proteoglycans (aggrecan and versican) are found in regions of compression where the tendon changes direction over a bony prominence (i.e. the DDFT at the level of the metacarpophalangeal joint).[12,13]

Collagen fibrils are bundled together into fibers that are separated by cellular cytoplasmic extensions of tenocytes (Figure 7.55).[25] Little is known about these cells; they may have different stages of activation. Various types of tenocytes have been described above; however, it is unknown how these cells function during growth, maturity, and aging.

Associated Structures

Blood Supply

Nutrients are supplied to the tendons and ligaments through diffusion and perfusion. Diffusion primarily occurs where a sheath encloses the structure with synovial fluid, playing an important role.[32] When blood is delivered via perfusion, it originates from three separate sources, including the musculotendinous attachments proximally, the osseous insertion distally, and the intratendinous and extratendinous vessels.[24] The extratendinous blood supply comes from vessels within the paratenon (extrasynovially) or mesotenon (intrasynovially) attachments. An example of intrasynovial mesotenon is when vessels lie at the vinculum between the fetlock annular ligament and the SDFT.

Connective Tissue

Connective tissues associated with tendons include sheaths and retinaculi. The tendon sheath is comparable to the joint capsule with an outer fibrous sheath and an inner synovial membrane.[24] Sheaths of tendons are folded around the tendinous structures, most commonly in areas in which the tendon passes over joint structures and bathes the tendon in synovial fluid. Annular ligaments, or retinacula, are tenacious fibrous bands that maintain tendons in the correct position (Figure 7.56).

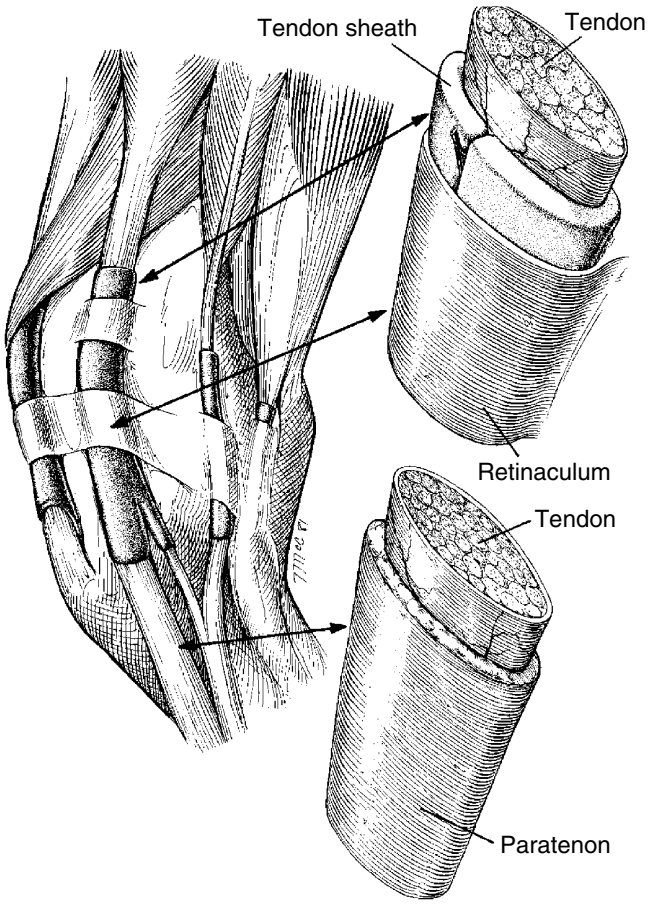


Figure 7.56. Diagram of tendons and their relationship to the retinaculum, endotenon, and tendon sheaths.

Bursae are structures very similar to tendon sheaths except that they are located between tendinous structures and bone. Similar to tendinous bursae, they have a synovial membrane and rely on synovial fluid to provide nearly frictionless movement between the tendon and the bone. Bursae that are commonly dealt with in lameness primarily include the navicular bursa, calcaneal bursa, bicipital bursa, and the olecranon bursa.

FUNCTIONS OF TENDONS AND LIGAMENTS

The elaborate anatomical architecture of tendons and ligaments is designed to allow tendons to passively transfer force generated by muscle to bony attachments on the opposite side of a joint, or joints, to provide movement.[32] Conversely, ligaments resist distraction of two bony attachments such as the collateral ligaments and suspensory ligaments.[32] Because the tendon and ligaments are on the palmar aspect of the equine distal limb, they receive significant weight-bearing loads and

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therefore provide support to the metacarpophalangeal and metatarsophalangeal joints during weight-bearing and exercise and act as force transmitters during rapid and unexpected movement.[24]

Tendons should also be considered as elastic structures that store energy for efficient locomotion.[1,32] Structures such as the SDFT provide shock absorption through the elasticity of the tendon combined with the attachment of the musculotendinous portion to the caudal aspect of the radius by the accessory or superior check ligament. The undulating gait of a horse at speed has been compared to the motion of a pogo stick with the tendons providing the elasticity of the bounce.[45] The unique structural arrangement within tendons and ligaments allows the horse to exercise at high speeds while minimizing energy expenditure.[32]

BIOMECHANICAL PROPERTIES

Biomechanical characteristics of tendons are usually described by stress–strain curves. These curves reveal the force per unit area (stress) plotted against the percentage of elongation (strain) and can be used to calculate elastic modulus (Figure 7.57). The four areas that are important to a stress–strain curve of a tendon are:

1. The toe region, where stretch is nonlinear. This represents the area where the undulating pattern of collagen fibrils are eliminated; this is also the elastic phase.
2. Linear deformation, or midsection, where the elastic stiffness of the tendon is represented.
3. Yield region, in which irreversible lengthening of the tendon occurs and the plastic phase begins.
4. Rupture, in which the collagen cross-links or fibrils sequentially rupture.

Initially, the tendon is highly compliant, but upon further extension, stiffness increases and viscoelastic (and permanent) changes occur.[24,42] It has been suggested that the second phase of the stress–strain curve for a tendon causes residual damage.[41] When the strain level is maintained between 3% and 5%, the normal linear stress–strain relationship is maintained; this is the typical strain that a tendon undergoes at a walk and trot. However, as strains reach 5% and 6%, the tendon is more viscoelastic, which results in permanent changes.[31] When the tendon reaches 10%–12% strain (ultimate tensile strain), permanent catastrophic changes such as rupture result. That said, maximum strains in Thoroughbreds at a gallop can reach 16%.[19,24] These are much greater than the strains most other species sustain at higher speeds and quite possibly explain why racing Thoroughbreds sustain a much greater incidence of disease affecting the SDFT.

The SDFT sustains loads of up to 1 metric ton at maximum weight-bearing.[32] Considering that the maximal cross section is approximately 1 cm^2 in cross-sectional area, it is little surprise that mid-metacarpal tendon injury is so prevalent. Variability in ultimate tensile force exists within any population of horses, however, with up to a twofold difference reported between weak and strong tendons.[46]

The modulus of elasticity/stiffness is represented by the force required to extend the tendon by a unit length. The stronger the tendon, the stiffer it is.[32] Hysteresis refers to the phase between loading and unloading a tendon and importantly results in loss of energy. It is estimated that approximately 5%[31] hysteresis occurs in equine tendon, increasing the temperature within the tendon core when repeated loads are applied to the tendon during exercise and possibly causing flexor tendinitis (Figure 7.58).[44]

TYPES OF TENDON AND LIGAMENT INJURIES

Three types of injury may occur in tendons and ligaments: excessive strain, physical tearing, and percutaneous injury. Overstrain may result from overwhelming the resistive strength in an acute manner, and it is believed to be the most common reason for ligament and tendon injuries in the horse.[32] Strain-induced injuries are believed to occur after a phase of molecular degeneration or inflammation that is not clinically

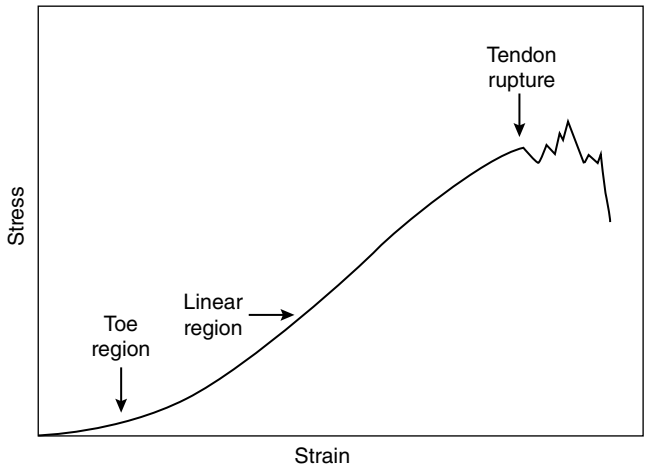


Figure 7.57. The stress strain curve for tendons. *Source*: From Goodship et al.[19] Reproduced with permission of Elsevier.

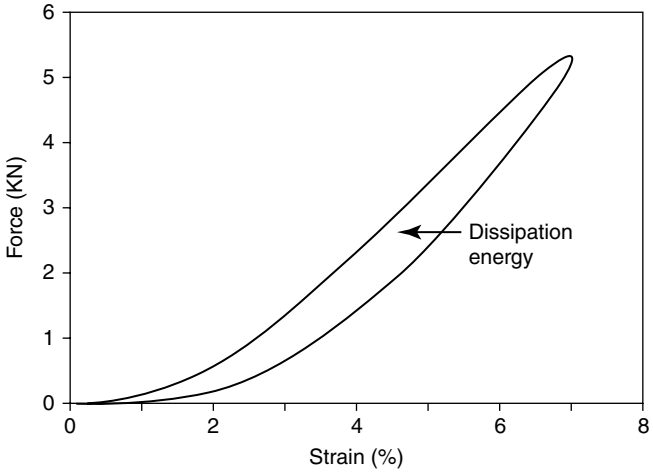


Figure 7.58. Loading of the SDFT revealing the hysteresis loop. The area within the loop represents the energy lost during one loading and unloading cycle. *Source*: From Goodship et al.[19] Reproduced with permission of Elsevier.

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evident nor produces any reparative responses, but instead progressively weakens the structure.[32] Detection of asymptomatic lesions in postmortem studies of normal horses is consistent with this observation.[40] Furthermore, many strain-induced tendinopathies are bilateral.[14] Large studies analyzing epidemiologic data in both horses and humans have shown strong correlations between age and injury rate.[22] Experimental studies and postmortem analyses of normal tendons have indicated that increasing age and exercise may induce tendon matrix degeneration instead of adaptation in the adult horse.[12,33]

Tendinopathies often begin with degeneration. Minor changes in the structural integrity predispose an already high-risk structure to injury. When the structural integrity is overwhelmed, irreversible damage ensues.[12] Palmar structures, especially at the heel, sustain load increases during heel strike, quickly placing the SDFT and suspensory ligament at risk.[19] Conversely, load of the DDFT rises during stance.

Physical disruption occurs within the tendon matrix when structural strength is overcome. Various structural breakdowns may occur, including fibrillar stretching with breakage of cross-links, fibrillar rupture, or, in severe cases, separation of tendon tissue.[12] Once this occurs, the damage signals repair processes that are common throughout the body such as inflammation followed by repair mechanisms (see phases of healing).

The risk of strain-induced injury is increased by factors such as the horse's speed, track surface (the harder the track, the higher the risk of injury), the horse's weight, fatigue level, and shoeing. Increased heel elevation has been demonstrated to be protective for the DDFT, but it increases the extension of the MCP joint.[3] Although low heels have been viewed as protective of SDFT tendinitis, epidemiologic studies have correlated an increased risk of tendon and ligament injuries with a low heel and long toe conformation.[12]

Tearing of the tendon also occurs, although with much less frequency than tendon degeneration. The most common site is the DDFT of the forelimb and occasionally the manica flexoria of the hindlimb. The mechanism of injury is not clear; however, it has been hypothesized that hyperextension of the fetlock canal combined with compression of the DDFT as it passes over the palmar aspect of the joint may predispose the structure to injury.[12] The hindlimb manica tears may be a result of ongoing tenosynovitis that frequently occurs in the tendon sheath of the hindlimb combined with hyperextension in that region.

The third common mechanism for tendon and ligament injury is percutaneous trauma. The distal limbs have minimal soft tissue coverage and commonly sustain contusion. Injuries resulting from wire, kicks, blunt trauma, and overreaching contribute to most of the percutaneous injuries seen. The most serious include those to the palmar/plantar region of the limb and tendon lacerations that are from wire. Common locations of tendon lacerations are the mid-cannon area of the hindlimb and the pastern region (including the tendon sheath) of both the front limb and hindlimb. These injuries often result in moderate to severe adhesion formation and a poor return to athleticism.

EFFECT OF AGING AND EXERCISE ON TENDON INJURY

Recently, a number of controlled studies in young and adult horses have elucidated the effect of exercise on developing and mature tendons.[5,13,23] Subjects used in these studies had no evidence of previous tendinitis nor any evidence of tendinitis induced by the exercise protocols.

Histological analysis revealed that local differences occurred in collagen fiber diameter of older but not younger exercised horses.[29] In adult tendon, a reduction in crimp occurred with aging and exercise. Exercise also induced a greater number of smaller fibrils within the central region of the SDFT. This change did not correlate with new collagen formation, indicating that an adaptive response occurred rather than an elevation in collagen.

Molecular composition changes also occurred with exercise. A reduction in GAG content and loss of COMP occurred in the center of the tendon with long-term exercise.[4] This is in contrast to an increase in GAG and type III collagen found in the SDFT of exercising horses whose tendons were collected postmortem. Because damage to these tendons was thought to result from subclinical tendon degeneration, these molecular changes most likely reflect a response to injury (or a reparative response) rather than a degenerative change associated with age and/or exercise.

Similar to cartilage, a lack of load to tendon results in a deficit of COMP levels within the tendon. However, once adequate COMP has accumulated within tendons, loading does not appear to affect the COMP levels.[32] Clinically, this implies that a deficit of exercise in the developing tendon may likely result in a tendon prone to injury due to a poorer quality of matrix since COMP levels are directly correlated to tendon strength. In contrast, too much exercise may also result in tendon injury if the strain levels of the tendon are exceeded.[5]

Exercise studies suggest that as tendon ages, degenerative changes associated with exercise accelerate. Cellular activity decreases and most likely collagen turnover slows. Growth factors such as TGF-β also decrease, which may lead to a reduced ability of the tendon to undergo a reparative response. Furthermore, less adaptive changes may occur following maturity, as evidenced by epidemiological data in horses and humans that draw a strong correlation between age and exercise and the incidence of tendon injury.[33] A recent study proved that as tendons age, there is an increase in stiffness in the interfascicular matrix that may result in tendon fascicles being loaded at an earlier point in the stress–strain curve, which leads to increased risk of damage.[40] Additionally, there is significantly less sliding at the fascicular interface in aged tendons.[40] Conversely, young growing tendon appears to be much more adaptive and responsive to loading and exercise, especially during the early stages of growth.

Among the unknown factors are the level and amount of work necessary to initiate the response. The natural exercise intensity of foals at play may be ideal to allow strain rates that boost tendon matrix production. Figure 7.59 is a hypothesized schematic representation of adaptive responses to injury in mature and immature

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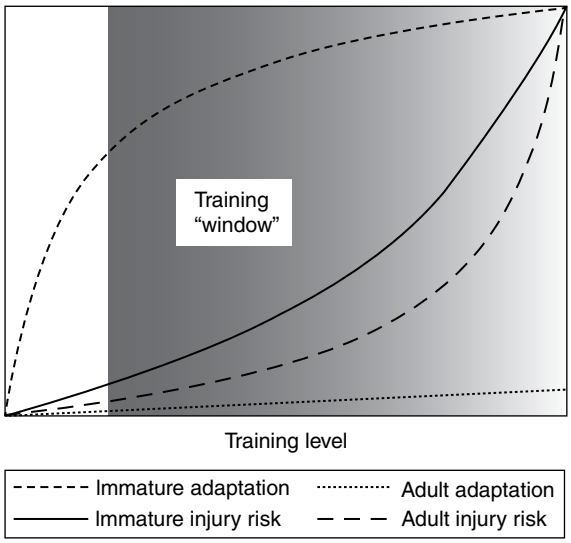


Figure 7.59. A representation of the adaptive and injury risk for growing (immature) and adult (older than 2 years of age) equine digital flexor tendons. *Source*: From Smith.[32] Reproduced with permission of Elsevier.

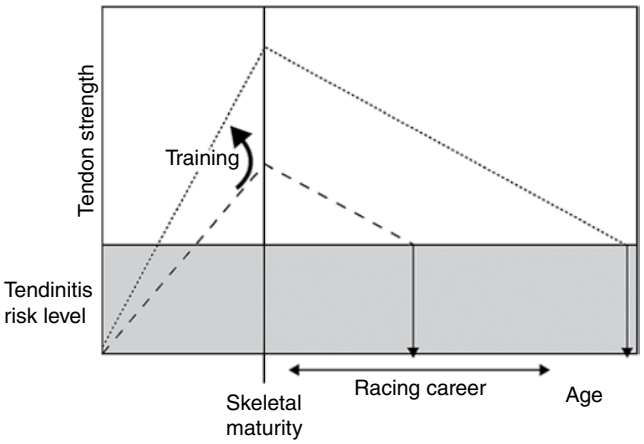


Figure 7.60. A diagram of an approach to tendinitis prevention. The dotted line represents a horse with strong tendons, in contrast to the dashed line, which represents a horse that has poor-quality tendons at skeletal maturity (approximately 2 years old). The horse with poor-quality tendons sustains tendinitis during its racing career due to cumulative fatigue damage to the tendon, whereas the horse with strong tendons, although sustaining the same degeneration, begins at a stronger point and does not suffer from tendinitis. Introduction of exercise throughout development improves tendon quality (arrow), potentially decreasing the incidence of tendinitis. *Source*: From Smith.[32] Reproduced with permission of Elsevier.

tendon. Figure 7.60 illustrates a strategy for preventing tendinitis in the horse, focusing on tendinitis risk before and after skeletal maturity.

MECHANISMS OF TENDON DEGENERATION

Mechanisms of tendon degeneration may have several etiologies including mechanical, physical, vascular, and inflammatory. Mechanical influences such as overextension or direct low-grade repetitive forces cause fatigue and microdamage, leading to degeneration. This also predisposes the tendon to clinical tendinitis when an acute supramaximal load is applied.

Hyperthermia during exercise is a physical influence that may predispose tendon to injury. Studies have determined that temperatures up to 45 °C occur secondary to galloping.[44] These temperatures are high enough to damage most other tissue types; however, *in vitro* experiments have revealed that tenocytes may resist these increases.[28] Nevertheless, the hyperemia that occurs in tendon at high speeds may damage the tenocytes or even the extracellular matrix.

Vascular disturbances are also hypothesized to contribute to tendon damage. When tendon is maximally loaded, blood flow is limited or abolished due to the compressive forces generated.[32] This may lead to a relative hypoxic state of the tendon, predisposing the tissue to degeneration. Although certain areas of tendon have poor perfusion, there is histologic evidence of an adaptive state to ischemia because fewer cells exist in these areas (i.e. the dorsal surface of the DDFT in the area of the fetlock) and matrix components in ischemic areas have compression-resistant components. In addition, these areas may receive nutrition from synovial fluid components.

Despite a reduction in blood flow to certain areas of tendon such as the DDFT in the area of the fetlock, these regions seem to be resistant to degeneration except in the most severe cases of tendinitis. Furthermore, studies have shown that mechanically reducing oxidative metabolism does not lead to abnormal cell proliferation.[4] Therefore, tenocytes, and tendons and ligaments in general, may be resistant to hypoxia compared with other tissues in the body. Although resistance may exist, however, generation of toxic free radicals predisposes the tendon to degeneration. This mechanism of injury may occur as a result of reperfusion injury when perfusion is restored.

Finally, mechanical, vascular, and hyperthermic environments may all lead to inflammation and release of proteolytic enzymes such as collagenase or aggrecanase that promote tendon degeneration. While inflammation and where it fits into tendon degeneration is yet to be defined in tendinopathy, most of the literature suggests that fibrosis of tendons results from persistent inflammation and lack of tendon healing.[10] Elevations in proteolytic enzymes result in a slowed matrix synthesis and an elevation in degradation, whereby overall degradation is high.[11,15,30]

In summary, tendon degeneration results from many factors that contribute to matrix and cellular abnormalities. Ultimately, the resident cell population cannot repair microdamage that is most likely due to deficient or absent growth factor stimuli (such as TGF-β) and cellular senescence within the tendon.[17] As loading continues and strain increases, failure of the fibrils occurs and results in tendon damage.

RESPONSE OF TENDONS AND LIGAMENTS TO INJURY: PHASES OF HEALING

When tendons and ligaments are damaged, the process of repair follows three stages of healing: the acute inflammatory phase, the subacute reparative phase, and

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the chronic remodeling phase. A subclinical phase of degeneration also exists; however, this phase is difficult to detect by clinical or ultrasonographic exam due to the minimal inflammatory reaction.

The initial inflammatory phase begins with an acute and substantial inflammatory response. The degree of inflammation depends on the severity of lesion and anti-inflammatory drugs that may be administered. This phase usually lasts 1–2 weeks and is characterized by intratendinous hemorrhage and edema, an increase in blood supply, leukocytes (initially neutrophils and then macrophages and monocytes), and proteolytic enzymes if unabated. Proteolytic enzyme release removes necrotic collagen; however, if the enzymes are not reduced with NSAID administration, their presence will result in expansion of the lesion within the inflammatory stage.

The subacute reparative phase peaks approximately 3 weeks following the initial injury.[32] This phase overlaps with the inflammatory phase and is marked by angiogenesis and the infiltration of fibroblasts in the damaged tissue. Fibroblasts originate from tendon, endotenon, and paratenon and are also delivered from the vascular origin.[7] Scar tissue is formed from the fibroblasts, which are characterized by randomly arranged collagen that is initially type III. Similar to cartilage repair, the resulting scar tissue is weaker than the original tendon tissue, and therefore predisposed to reinjury at the original site of injury. When reinjury occurs, the inflammatory and reparative phases of healing are perpetuated, furthering the damage within the tendon.[32]

The poor healing response of tendon in areas of poor blood supply and paratenon may be explained by a lack of migration of fibroblasts. Adhesions are formed following tendon injury, and although detrimental, they allow healing factors to contribute to injured tissue. Therefore, although adhesions restrict movement of tendon, they allow tendon to form reparative tissue.[32]

The chronic remodeling phase consists of a slow conversion of type III collagen to type I collagen. This process occurs over several months, and the original tendon strength is never restored. Loading (exercise) that ensues in a controlled manner enhances conversion of type III to type I collagen. It improves the alignment of the collagen fibrils in the direction of force and results in better mechanical tendon strength.

Controlled exercise programs are integral to every successful tendon rehabilitation program. It is important to use ultrasound therapy and modify the exercise program based upon the quality of repair. Table 7.1 illustrates a standard rehabilitation program with ultrasound reexamination at specified times.

Reinjury is common following repair of tendons and ligaments; it has been reported to occur in 8%–43% of racehorses.[14] Furthermore, the contralateral tendon or ligament also may become injured.[14] Although the strength of the remodeled tendon is improved 15–18 months following injury, the resulting elasticity is severely weakened, leading to elevated strains in the undamaged tendon. It is this tissue that often becomes damaged when reinjury occurs. Furthermore, if the SDFT is damaged and lengthened through the damage, the suspensory ligament may sustain microdamage and eventual injury due to increased strains.[22]

Table 7.1. A standard exercise program recommended following tendon injury.

Exercise level	Week	Duration and nature of exercise
0	0–2	Box rest
1	3	10-min walking daily
1	4	15-min walking daily
1	5	20-min walking daily
1	6	25-min walking daily
1	7	30-min walking daily
1	8	35-min walking daily
1	9	40-min walking daily
1	10–12	45-min walking daily
Week 12: Repeat ultrasound examination		
2	13–16	40-min walking and 5-min trotting daily
2	17–20	35-min walking and 10-min trotting daily
2	21–24	30-min walking and 15-min trotting daily
Week 24: Repeat ultrasound examination		
3	25–28	25-min walking and 20-min trotting daily
3	29–32	20-min walking and 25-min trotting daily
Week 32: Repeat ultrasound examination		
4	33–40	45-min exercise daily, gradually increasing in amount
4	41–48	45-min exercise daily with fast work 3 times a week
Week 48: Repeat ultrasound examination		
5	48+	Return to full competition/race training

This protocol can be modified based upon ultrasound recheck examinations.
Source: Davis and Smith.[12] Reproduced with permission of Elsevier.

The basic goals of treatment programs are to initially minimize the acute inflammatory phase, thereby decreasing the ongoing damage of tendinous inflammation and edema. Next, the goals are to implement therapies that improve collagen type I and extracellular matrix production, thereby strengthening the tendon so that the structure can withstand the mechanical forces that it encountered before the initial injury. Finally, the adhesions that may form in the healing phases significantly reduce athletic function due to pain and lameness. Therefore, minimizing inflammation, enhancing regeneration, and controlling exercise optimize the chances of successful healing of tendons. For more detail on intralesional therapies, see Chapter 8.

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MONITORING TENDON INJURY

Ultrasound examination remains the gold standard for monitoring tendinopathies. Clinical examination, while important, does not detect subtle changes and provides a poor objective assessment. Tendons and ligaments should be evaluated initially at the time of injury and then approximately 1 week following the injury due the frequent occurrence of expansion of the lesion in the first week. The prognosis for return to athleticism is determined by the appearance in the initial week.

The ultrasound exam is performed after clipping the hair and washing the limb to obtain the highest quality image. Transverse and linear images should be obtained and images should be recorded in a methodical fashion so that the images are accurately recorded. For example, during the examination, the limbs are divided into seven subunits, or, alternatively, the images are recorded as distance from the accessory carpal bones. Although one limb is almost always affected more severely than the others, ultrasound examinations should be done bilaterally due to the common occurrence of bilateral disease, especially in strain-induced tendon injury.

Ultrasound changes frequently associated with acute tendinopathy include enlargement, hypoechogenicity, alterations in tendon striated pattern viewed in the longitudinal images, and alterations in shape and margins (Figure 7.61).[12] Chronic tendinopathies are associated with enlargements or changes in echogenicity and irregularities in striations observed on the longitudinal view.

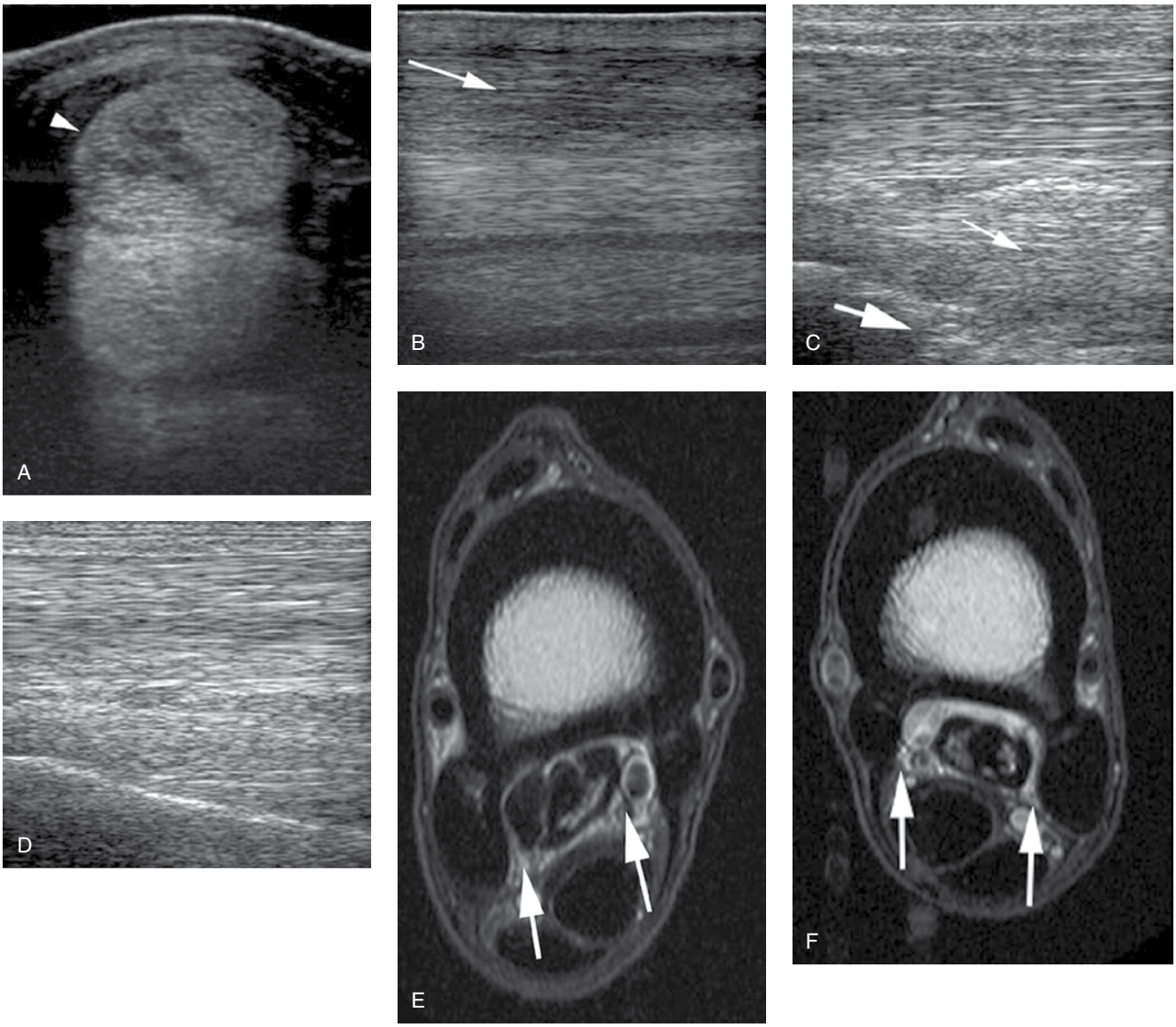


Figure 7.61. Examples of variations of tendon and ligament injuries detected on ultrasound or MRI examination. (A) Enlargement and tearing of a SDFT (horizontal image). (B) A longitudinal ultrasound image revealing tendon fiber disruption of the SDFT. (C) Fiber and bone disruption (large arrow) of a proximal hind suspensory ligament (small arrow, ligament viewed longitudinally) in comparison to (D). (D) A normal proximal hindlimb suspensory ligament. (E) MRI examination of the disrupted and enlarged hind suspensory ligament (between arrows). (F) MRI of the normal suspensory ligament of the contralateral limb. Note the space (white area) around the suspensory ligament, which is normal. *Source*: Images (A) and (B) courtesy of Dr. Natasha Werpy.

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A more recent diagnostic tool called sonoelastography is currently being investigated for diagnostic use for tendonopathy.[38,39] This technology can assess the inner stiffness of tissues and may be able to distinguish between different phases (acute, subacute, and chronic) of healing with much more accuracy than traditional ultrasound. If further investigation confirms initial promising data, this monitoring technique will be a mainstay in monitoring tendon response to rehabilitation and prognostic outcomes.

BIOMARKERS FOR TENDON DISEASE

The field of biomarkers is of keen interest to many clinicians and researchers. Accurate detection of subclinical disease contributes to improved prevention of a common career-ending disease. Furthermore, early detection of disease enables more tailored rehabilitation programs and altered training regimens. Ultrasound remains the gold standard of detection,[16] permitting a more accurate reflection of cellular disease and sensitivity to subtle changes in the tissue state. Biomarkers that detect the stage of disease may also predict optimal treatment and rehabilitation protocols and evaluate their efficacy.

Markers that have been studied intensively include collagen synthesis (carboxy-terminal propeptide of type I collagen or PICP) and degradation (cross-linked carboxy-terminal telopeptide of type I collagen or ICTP) after tendon injury.[12,20] Significant elevations in PICP concentrations have been associated with tendinitis, whereas ICTP has been unchanged in control groups. These changes reflect the ability of serum concentrations of PICP to reveal disease of tendons and indicate that these markers are not necessary bone specific.

COMP has also been intensively studied in tendon disease.[34] In one study, synovial fluid levels within tendon sheaths were significantly higher when tendons were damaged or sheaths were septic. However, serum levels were unaffected due to naturally high levels of COMP in blood. Although COMP appears to be a good marker in laboratory analysis of disease, it may not be an accurate marker for specific tendon disease.

COMMON CLINICAL CONDITIONS OF TENDONS AND LIGAMENTS

The most common tendon affected in tendinopathies is the SDFT. Although the lesion may be focal or generalized, it is usually centrally located in the tendon, just below the mid-metacarpal region of the limb. It can be focal or it may extend throughout the length of the tendon, giving rise to a palmar swelling of the metacarpal region (bowed tendon) (Figures 7.62 and 4.164). The section of the SDFT enclosed within the tendon sheath is affected much less often.

Desmitis of the accessory ligament of the DDFT can occur in association with superficial flexor tendinitis. Ponies have been observed to have a higher incidence of desmitis of the accessory ligament while rarely experiencing superficial flexor tendinitis.[12] Lameness is usually much less severe than with superficial flexor tendinitis,

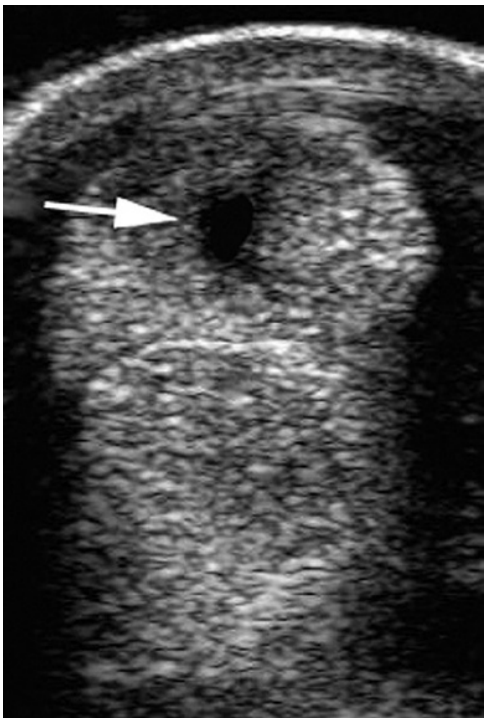


Figure 7.62. Ultrasound image of a core lesion in an SDFT. The arrow is pointing to the core lesion within the SDFT. *Source*: Courtesy of Dr. Natasha Werpy.

and swelling is often restricted to the proximal half of the metacarpus, immediately dorsal to the SDFT.

The suspensory ligament may sustain a desmitis along any section. Proximal suspensory desmitis (in both the front limb and hindlimb) is often restricted to that region, whereas midbody or branch lesions may occur concurrently or extend into each area. Branch lesions also may be found within the MCP joint and result in synovial effusion and lameness requiring arthroscopic debridement.[26] Synovial fluid may decrease the healing of these lesions as well.[12] With severe suspensory desmitis, the limb may have a characteristic dropped fetlock either standing or at the walk due to decreased support of the fetlock.

The DDFT is more frequently associated with tendinitis within the digital tendon sheath, especially of the hindlimb (Figure 7.63).[36,42,47] These lesions may be a result of single excessive load cycles.[12] Two manifestations are typically observed with one lesion found within the substance of the tendon and the other found more in the periphery (medial and lateral borders) of the tendon, usually in the region of the MCP/MTP joint.[47] Invariably, these lesions result in synovial effusion of the digital sheath, with peripheral lesions being more difficult to detect with ultrasound.

Other tendon and ligaments may sustain strain-induced injury, although the frequency of strain to the palmar soft tissue structures of the metacarpus is much more common. Ligament injuries are much more common when the joint they support (and span) is inappropriately overloaded. When this occurs, desmitis and more seriously subluxation or luxation can result (Figure 7.64). Finally, when significant tendon lacerations occur as the

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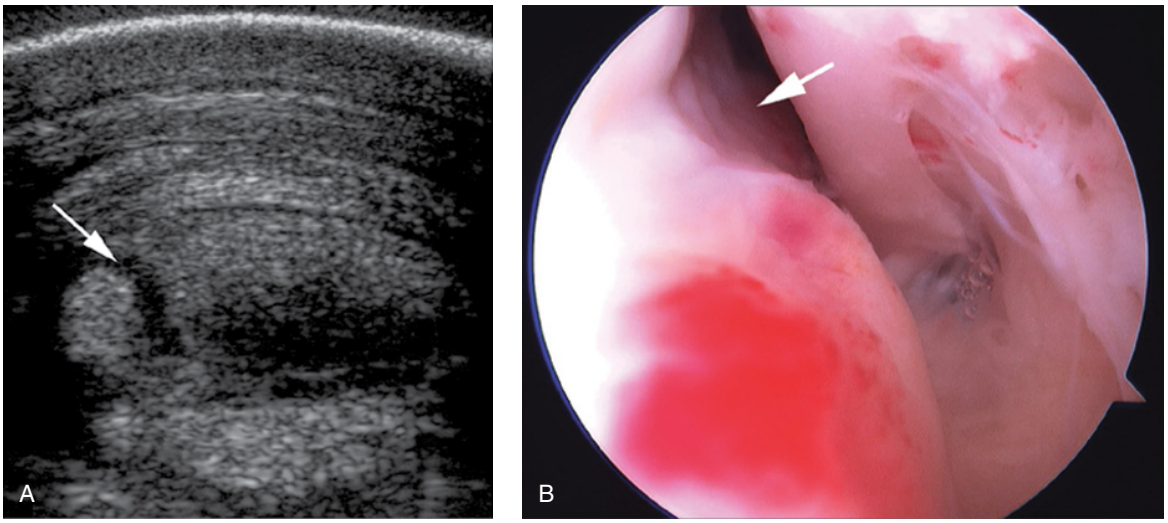


Figure 7.63. Ultrasound image of the DDFT with a linear tear located within the tendon sheath at the level of the fetlock (A) and the corresponding tenoscopic image (B). The arrow in both images points to the tear.



Figure 7.64. Radiograph of a complete fetlock luxation demonstrating failure of the collateral ligaments.

result of trauma, characteristic stances of the limb can often help the clinician determine which structure(s) has(have) been affected (Figures 12.15 and 12.16). For more information on tendon lacerations, refer to Chapter 12.

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MUSCLE INJURIES AND DISEASE

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CLASSIFICATION OF MUSCLE DISORDERS

A muscle disorder is usually suspected because of altered muscle tone, weakness and/or exercise intolerance, muscle atrophy, muscle pain or swelling, or a gait abnormality not attributed to skeletal/tendon or ligamentous structures. Horses with trauma or atrophy of individual muscle groups may show an asymmetric lameness. Those with generalized muscle soreness or myopathies affecting energy metabolism may show evidence of a mildly asymmetric gait, shifting lameness, poor performance under saddle, progressive stiffness, or inability to engage the hindquarters. Myopathies and skeletal/ligamentous causes of lameness often occur concurrently.

DIAGNOSIS OF SPECIFIC MUSCLE DISORDERS

Many times a complete examination of the musculoskeletal and neurologic systems is required to diagnose a myopathy. Myopathies can change the horse's movement to a point that lameness develops, and lameness can change muscle function to the point that soreness develops. Both possibilities should be considered. In addition, neurologic causes of muscle weakness, atrophy, and gait changes should also be fully evaluated. The topics below are key components of the examination of skeletal muscle.

History

A history of stiffness, muscle cramping, pain, muscle fasciculations, exercise intolerance, undiagnosed lameness, weakness, or muscle atrophy may all indicate a muscle disorder. Further characterization requires a detailed account of the horse's performance level, exercise schedule, previous lameness, diet, vaccination history, signs of respiratory disease, duration, severity and frequency of muscle problem, any factors that initiate the muscle problem, and all medications with which the horse is being treated.

Physical Examination

A detailed evaluation of the muscular system includes inspection of the horse for symmetry of muscle mass while standing with forelimbs and hindlimbs exactly square. Any evidence of fine tremors or fasciculations should be noted before palpating the animal. Horses originating in the Southwestern United States that have muscle pain and fasciculations should have their ears examined with an otoscope for ear ticks (*Otobius megnini*).[44]

The entire muscle mass of the horse should be palpated for heat, pain, swelling, or atrophy, comparing contralateral muscle groups. Firm, deep palpation of the lumbar, gluteal, and semimembranosus and semitendinosus muscles may reveal pain, cramps, or fibrosis. The triceps, pectoral, gluteal, and semitendinosus muscles should be tapped with a fist or percussion hammer and observed for a prolonged contracture suggestive of myotonia. Running a blunt instrument such as a needle cap or a pen over the lumbar and gluteal muscles should illicit extension followed by flexion in healthy horses. Guarding against movement may reflect abnormalities in the pelvic or thoracolumbar muscles or pain associated with the thoracolumbar spine or sacroiliac joints.

The horse should be observed at a walk or trot for any gait abnormalities and flexion tests performed for lameness. In some cases it may be helpful to observe the horse under saddle and have the owner demonstrate the exact nature of the gait abnormality or performance problem.

Ancillary Diagnostic Tests

Muscle Enzymes

The best assessment of the extent of acute muscle damage is attained by measuring serum CK activity. CK, a relatively low molecular weight protein (80,000 Da), is liberated into the extracellular fluid within hours of muscle damage, or increased cell membrane permeability, and usually peaks at 4–6 hours after muscle injury.[10] A three- to fivefold increase in serum CK from normal values is believed to represent necrosis of approximately 20 g of muscle tissue.[78] Limited elevations in CK (less than 1,000 U/L high range of normal value = 380 U/L) may accompany training or transport. Extreme fatiguing exercise (e.g. endurance rides or the cross-country phase of a 3-day event) may result in CK activities being increased to more than 1,000 U/L, but usually less than 4,000 U/L.[80] Under these circumstances, serum CK activities rapidly return to baseline (i.e. less than 350 IU/L in 24–48 hours). Recumbent animals also may have slightly elevated CK activities that are usually less than 3,000 U/L. In contrast, more substantial elevations (from several thousand to hundreds of thousands of U/L) in the activity of this enzyme may occur with rhabdomyolysis.

Serum AST is a higher molecular weight protein that has high activity in skeletal and cardiac muscle as well as in liver, red blood cells, and other tissues. Elevations in AST are not specific for myonecrosis, and increases could be the result of hemolysis, muscle, liver, or other organ damage. AST activity rises more slowly in response to myonecrosis than does CK, often peaking between 12 and 24 hours after the insult. In addition, AST is cleared slowly by the reticuloendothelial system and may persist for 2–3 weeks after rhabdomyolysis.[39,67]

By comparing serial activities of CK and AST, information concerning the progression of myonecrosis or muscle cell membrane permeability may be derived. Elevations in both CK and AST reflect relatively recent

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or active myonecrosis; persistently elevated serum CK indicates that myonecrosis is likely to be continuing. Elevated AST activity accompanied by decreasing or normal CK activity indicates that myonecrosis has ceased. The degree of elevation of CK and AST does not necessarily reflect the severity of clinical signs.

Elevations in plasma/serum myoglobin concentrations indicate acute muscle damage because this small protein leaks into plasma immediately after muscle damage and is rapidly cleared in the urine by the kidney.[30]

Exercise Response Test

Diagnosing chronic exertional rhabdomyolysis may be difficult in horses that have normal serum AST and CK when examined between episodes. In such cases, an exercise challenge can be helpful in detecting subclinical exertional rhabdomyolysis. In addition, quantifying the extent of rhabdomyolysis during mild exercise is helpful in deciding how rapidly to put a horse back into training. Blood samples should be taken before exercise and about 4–6 hours after exercise to evaluate peak changes in CK. Serum CK activity measured immediately post-exercise does not reflect the amount of damage occurring during the exercise test. Small fluctuations in serum CK activity may occur with exercise due to enhanced muscle membrane permeability, particularly if exercise is prolonged or strenuous and the horse is untrained.[27,57]

A submaximal exercise test is often valuable for detecting rhabdomyolysis because it provides more consistent evidence of subclinical rhabdomyolysis than maximal exercise tests.[67] Fifteen minutes of trotting is often sufficient to produce subclinical muscle damage in horses prone to chronic exertional myopathies.[36] If signs of stiffness develop before this, exercise should be concluded. A normal response is less than a three- to four-fold increase from basal CK. In the author's experience, horses with type 1 polysaccharide storage myopathy (PSSM1) and active recurrent exertional rhabdomyolysis (RER) are more likely to have a positive exercise response than horses with type 2 polysaccharide storage myopathy (PSSM2) or well-managed PSSM1.[38]

Ultrasonography

Physical disruption of muscle fibers and fibrosis can be detected by diagnostic ultrasonography. The typical striated echogenic pattern of skeletal muscle varies according to the muscle group, and careful comparisons must be made between similar sites in contralateral limbs, in both transverse and longitudinal images. The appearance of muscle is also sensitive to how the horse stands and whether the muscle is under tension, so it is important that the horse stand squarely and bear weight evenly. Muscle fascia appears as well defined, relatively echodense bands. Care must be taken in identifying large vessels and artifacts created by them.[58]

Relatively hypoechoic areas within muscle that lack the normal muscle fiber striation indicate acute injury. The jagged edge of the margin of the torn muscle may be increased in echogenicity. Tears in the muscle fascia may be identified. A loculated hematoma may fill the injured area and slowly be replaced by hypoechoic granulation tissue. As muscle fibers regenerate, there is a progressive increase in echogenicity. Relatively hyperechoic regions may develop due to fibrous scarring. Hyperechoic regions causing shadowing artifacts reflect mineralization.[70]

Electromyography (EMG)

Electromyography (EMG) is particularly useful for evaluating muscle atrophy, muscle fasciculations, and myotonia. Normal muscle shows a brief burst of electrical activity when the EMG needle is inserted and then quiescence unless motor units are recruited (motor unit action potentials) or the needle is very close to a motor end plate (miniature end plate potentials). Spontaneous electrical activity, fibrillation potentials, and positive sharp waves are seen in horses with denervation and myotonic discharges, or complex repetitive discharges are seen in horses with muscle conduction system abnormalities (e.g. myotonia, hyperkalemic periodic paralysis [HYPP]).[80]

Muscle Biopsy

A number of specific equine myopathies have been identified through the routine examination of muscle biopsies from horses with evidence of muscle dysfunction. Muscle fiber sizes, shapes, and fiber type distribution, mitochondrial distribution, polysaccharide staining pattern, neuromuscular junctions, nerve branches, connective tissue, and blood vessels can be fully evaluated in frozen sections using a battery of tinctorial and histochemical stains.

Inflammation, muscle fiber necrosis, cellular infiltrates, and proliferation of connective tissue are evident in formalin and frozen sections. However, there are many pathologic alterations that cannot be detected in formalin-fixed tissue but can readily be seen in histochemical stains of fresh-frozen biopsy samples.[14] These include identification of muscle fiber types to differentiate between neurogenic and myogenic atrophy, characterization of vacuolar storage material, characterization of inclusion bodies, and mitochondrial density. In addition, frozen samples may be used for biochemical analysis of substrate concentrations and enzyme activities as well as DNA isolation.

Muscle biopsies are preferably collected from abnormal/diseased muscle. A 6-mm outer diameter percutaneous needle biopsy technique can be used to obtain small muscle samples through a 1.5-cm skin incision using a local anesthetic subcutaneously. If this technique is used, enough muscle should be obtained to form a 1.5-cm^2 sample at a minimum. These samples do not tolerate shipment to an outside laboratory well. The optimum biopsy for shipment of histopathologic tissues to a laboratory is collected using surgical or open techniques, performed under local anesthesia (Figure 7.65).

Semimembranosus samples are often selected for open biopsy in cases of exertional rhabdomyolysis. Care must be exercised to infiltrate only the subcutaneous tissues, not the muscle, with local anesthetic agent. The objective is to obtain approximately a 2-cm cube of tissue without crush artifact; hence, a suitably long skin incision is required. Subsequently, two parallel incisions 2 cm apart should be made longitudinal to the muscle fibers with a scalpel. The muscle should only be handled in one corner

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Figure 7.65. Muscle biopsy of the semimembranosus muscle. Note that the sample is gently handled at one end without crushing the tissue. Samples can be wrapped in saline-moistened gauze and shipped in a hard container on ice packs for frozen sections.

using forceps, and care should be taken not to crush the tissue. The muscle sample is then excised by transverse incisions 2 cm apart, and the tissue fixed appropriately.

Routine histopathologic samples can be placed in formalin. Fresh specimens are best shipped overnight in gauze slightly moistened with saline in a plastic jar on ice packs to specialized laboratories where they are frozen in isopentane (methylbutane) that is suspended in liquid nitrogen to minimize freeze artifact. Samples that potentially may be used for biochemical analysis should be immediately frozen in liquid nitrogen.

Genetic Testing

DNA tests are available for the diagnosis of PSSM1, malignant hyperthermia (MH), HYPP, and immune-mediated myositis. Some laboratories prefer submission of a minimum of 20 mane or tail hairs pulled out with roots intact. Others analyze whole blood submitted in EDTA tubes.

MUSCLE SORENESS

Overuse and Muscle Strain

It is often difficult to assess the presence of muscle pain in horses because it is a subjective assessment and often coexists with a more easily recognized lameness. Frequently the lameness is treated without managing muscle soreness. In human athletes muscle stiffness and soreness are more easily recognized causes of decreased performance. In humans, muscle pain and stiffness are reported 24–48 hours after unaccustomed use of certain muscles; this is referred to as delayed-onset muscular soreness (DOMS). Pain resolves spontaneously, assuming the muscles are not overworked again; however, continued overstress may result in ultrastructural damage to myofilaments and eventually histological evidence of myofiber necrosis. Gradual training involving the activity that provoked the original DOMS decreases the amount of soreness associated with that condition over time. The muscles most prone to strain in horses appear to be the back muscles (Standardbreds, dressage and jumping horses), semimembranosus/tendinosus muscles (reining, roping, cutting horses), and pectoral muscles (event horses after cross-country jumping). Poor racing performance or poor jumping techniques and the horse twisting itself over jumps are common complaints in horses with sore backs.[70]

Muscle Trauma

When muscle has been strained to the point of tearing through entire muscle fascicles, hemorrhage and inflammation can result in acute muscular pain and recognizable swelling in superficial muscles. The most common sites of injury in the forelimb include biceps brachii, brachiocephalicus, pectorals, and the musculotendinous junction of the superficial digital flexor. In the hindlimb, semimembranosus and semitendinosus, adductor, gluteal, and gastrocnemius muscle injuries are common (Figure 7.66).[70] Whereas acute trauma is associated with swelling or herniation of a muscle belly through the overlying fascia, chronic injury may be associated with loss of muscle bulk, fibrosis, mineralization, or rarely ossification (Figure 7.67).

Diagnosis

An acute muscle tear may produce a palpable defect, but this becomes filled with edema, hemorrhage, and eventually granulation tissue. Localization of deeper muscle injury is more challenging, because the clinical signs are more subtle.

Pain and limitations in movement should be evaluated in a full range of passive movement of the neck, limbs, and thoracolumbar region. The horse should be observed for gait abnormalities at both the walk and the trot. However, overt lameness may not be present if pain only occurs when the muscle contracts strongly or is stretched maximally. Serum muscle enzyme concentrations are usually in the normal range for focal muscle trauma.

Treatment

After most insults, muscle will regenerate over time unless muscle architecture is so severely disrupted that fibrosis results. The choice of treatment depends upon the type of muscle injury and the stage of injury and repair. The goal is to relieve muscle spasm, decrease inflammation, restore circulation, minimize scarring,

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Figure 7.66. (A) Acute swelling of the right adductor muscles (arrow) in a mare that slipped in a muddy paddock. (B) The same muscle 2 weeks later after cold therapy and treatment with a nonsteroidal anti-inflammatory.



Figure 7.67. Severe atrophy, fibrosis, and contracture of the left hamstring region in a horse with previous trauma to the area. *Source*: Courtesy of Dr. Gary Baxter.

and maintain range of motion. Treatment modalities include cold therapy, therapeutic ultrasound or laser therapy, transcutaneous electrical stimulation, massage, passive stretching combined with box rest, and a graduated, controlled exercise program. Relief of acute muscle spasm may require chiropractic manipulation. The extent of rest depends upon the degree of tearing and hemorrhage. Once initiated, the exercise program must be carefully moderated according to the site of the injury to avoid overstress in the early stages of repair but encourage a progressive increase in strength. Aqua treadmills can provide an excellent controlled means of building muscle strength.

Muscle Ischemia

Muscle ischemia is extremely painful and occurs with acute trauma, vascular occlusion, and the crush/compartment syndrome. Vascular occlusion may be a sequela to infarctive purpura hemorrhagica, disseminated intravascular coagulation, and iliac thrombosis.

Compartmental syndrome develops when high pressure within a closed fascial space reduces capillary blood perfusion below a level necessary for muscle tissue viability. Trauma to the caudolateral compartment of the antebrachium can cause ischemia of the enclosed lateral digital extensor, ulnaris lateralis, superficial and deep digital flexor, and flexor carpi ulnaris muscles, as well as the median artery, vein, and nerve (Figure 7.68).[63] Lameness is acute and severe in onset, with reluctance to bear weight on the limb associated with firm swelling on the caudolateral aspect of the antebrachium. Treatment of compartment syndrome is by fasciotomy. In one case, incising the superficial fascia over the ulnaris lateralis resulted in immediate separation of the fascia by 3–4 cm and rapid relief of clinical signs.[63]

In recumbent or anesthetized horses, the triceps muscle, gluteal muscles, longissimus dorsi, or extensors of the hindlimb may become ischemic because of compression, prolonged hypoperfusion, and subsequent reperfusion and edema. Hypotension during surgery in which mean arterial blood pressure is less than 60 mm Hg contributes to the development of this syndrome. Clinical signs are usually evident as soon as the horse tries to stand, but may be delayed for up to 2 hours. In severe cases the horse is unable to stand.

Triceps myopathy is often characterized by a dropped elbow stance typical of radial nerve paralysis. Involvement of the gluteal muscles may result in unwillingness to bear

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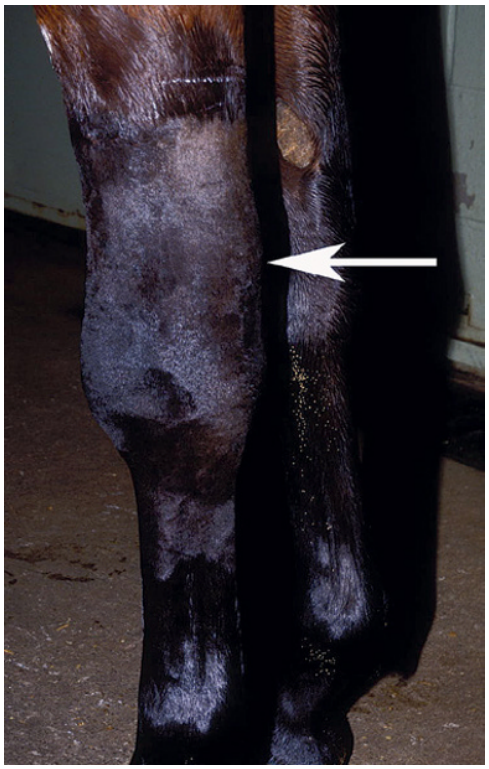


Figure 7.68. Trauma and associated swelling to the caudolateral antebrachium (arrow) that can cause ischemia of the enclosed structures, leading to signs suggestive of compartment syndrome. *Source*: Courtesy of Dr. Gary Baxter.

weight on the hindlimbs. Horses may appear distressed, with profuse sweating, tachycardia, and tachypnea. The degree of distress depends on the severity of the muscle damage. Affected muscles may feel very hard. There may be localized swelling. Neuropathy also may develop in the compressed muscles, resulting in temporary radial or peroneal nerve damage. Serum CK levels greater than 2,000 IU/L often occur within 4 hours after anesthesia as blood flow returns to the affected muscles.

Treatment of postanesthetic myopathy or neuropathy in horses is similar to that described in the section on acute exertional rhabdomyolysis. Bandaging and splinting of limbs with neuropraxia may help the horse stand and ambulate more easily, thus relieving much of the animal's anxiety. Horses that can stand on three legs have a good prognosis for recovery from myopathies or neuropathies and generally respond to treatment within 72 hours. Horses that are affected bilaterally and unable to stand have a very poor prognosis.

Muscle Abscesses

Staphylococcus aureus, *Streptococcus equi*, and *Corynebacterium pseudotuberculosis* are common causes of skeletal muscle abscessation. Abscesses develop following penetrating injuries or local or hematogenous spread of infection. Ultrasonography and culture of aspirated fluid are the best means of diagnosis in superficial sites. Deeper abscesses may be diagnosed using fibrinogen, nucleated white blood cell count, and the synergistic hemolysin inhibition test for abscesses due to *C. pseudotuberculosis*.

Clostridial Myositis

Clostridial myositis most commonly occurs following penetrating wounds, parturition injuries, castration, and especially intramuscular injections.[56] Spores of *Clostridia* species involved are found in soil and feces and may lie dormant in skeletal muscles. When tissue is traumatized by injection or injury (low oxidative potential and alkaline pH), organisms proliferate, elaborate exotoxins, and cause extensive necrosis of muscle and surrounding tissue. Death can occur quickly from toxemia with *C. sordelli*, *C. sporogenes*, and *C. chauvoei*. Cases of *C. perfringens* have a lower mortality rate and initially present with swelling and crepitus in an area in muscle, as well as moderate systemic signs of toxemia. Ultrasound is useful in identifying air bubbles in muscle exudate, and clostridial species can be identified in aspirates by fluorescent antibody staining and anaerobic bacterial culture. Antibiotic therapy, wound fenestration, aggressive surgical debridement over the entire affected area, and supportive care are the hallmarks of successful treatment.[56] High doses of intravenous potassium penicillin are recommended every 2–4 hours until the horse is stable (1–5 days) combined with or followed by oral metronidazole. Supportive fluid therapy and anti-inflammatory agents for control of pain and swelling are recommended. Extensive skin sloughing over the affected area is common in surviving horses.

Muscle Cramping

Muscle cramping is a painful condition that arises from hyperactivity of motor units caused by repetitive firing of the peripheral and/or central nervous system. The origin of the cramp in most cases is believed to be the intramuscular portion of the motor nerve. Most muscle cramps are also accompanied by fasciculations in the same muscle and normal serum CK activity. In contrast, muscle contractures, such as those seen in exertional rhabdomyolysis, are painful muscle spasms that represent a state of muscle contracture unaccompanied by depolarization of the muscle membrane. Muscle cramps can be induced by forceful contraction of a shortened muscle, changes in the electrolyte composition of extracellular fluid, and ear tick infestations in horses. Muscles may also develop intensely painful spasms, particularly following trauma to the caudal thoracic and lumbar regions. Manipulation to release muscle spasm can produce relief of pain and rapid restoration of normal performance.

Electrolyte Disturbances

Muscle cramping in endurance horses is most frequent in hot, humid weather when horses develop remarkable deficits in sodium, potassium, chloride magnesium, and calcium due to extensive sweating. Synchronous diaphragmatic flutter may be seen in association with cramping.[12] Mild muscle cramping is self-limiting, and the signs abate with rest or light exercise. Exhausted

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horses with metabolic derangements, however, require immediate treatment, including plasma volume expansion with oral or intravenous isotonic polyionic fluids and cooling (using water and fans).[11] Because most horses with this condition are alkalotic, administration of solutions containing sodium bicarbonate is contraindicated. Daily direct addition of 2 oz. of sodium chloride and 1 oz. of potassium chloride to the feed is recommended for horses with recurrent cramping in addition to electrolyte supplementation before and after endurance rides.

Muscle AtrophyDenervation Atrophy

Neurogenic atrophy results in fairly rapid atrophy of both slow- and fast-twitch muscle fibers because the normal low-level tonic neural stimulus that is necessary to maintain muscle fiber mass is absent. Complete denervation of a muscle results in more than a loss of 50% of muscle mass within 2–3 weeks. Denervation can be focal (e.g. suprascapular nerve damage causes supraspinatus/infraspinatus atrophy) or generalized with slower progression (e.g. equine motor neuron disease).

Focal denervation atrophy may occur due to nerve trauma (Figure 7.69) or focal ventral spinal cord infection from equine protozoal myelitis. If the injury is mild, then most nerves can regenerate (at about 4 mm/day) and the original continuity to the muscle can be reestablished. However, if the nerve injury is severe (e.g. transection), regrowth of the nerve to the muscle is delayed and may never occur. Furthermore, if concurrent muscle atrophy is severe, even if a functional connection is made, there may only be a limited number of fibers available for renervation. Generalized denervation atrophy occurs with diseases such as equine motor neuron disease and peripheral neuropathies.



Figure 7.69. Focal denervation atrophy of the gluteal musculature (arrow) that was attributed to nerve root trauma. *Source*: Courtesy of Dr. Gary Baxter.

Myogenic Atrophy

Muscle atrophy can be myogenic in origin, resulting from hypovitaminosis E, severe rhabdomyolysis, immune-mediated myositis, disuse, malnutrition, cachexia, and corticosteroid excess. Disuse, malnutrition, cachexia, and corticosteroid excess are characterized by slow onset of atrophy over months of exclusively type 2 fast-twitch muscle fibers and normal serum CK. Horses with rhabdomyolysis and marked elevations in serum CK activity can have a moderate onset of atrophy over a few weeks. Vitamin E deficiency causes gradual loss of muscle mass.[6,16] In contrast, immune-mediated myositis shows rapid loss of 30% of muscle mass over the topline within 48 hours. This disorder has lymphocytic infiltrates in biopsies of the atrophied muscle associated with elevated serum CK activity.[18,37]

EXERTIONAL RHABDOMYOLYSIS

Exertional rhabdomyolysis (ER) occurs in about 3% of exercising horses[13] and in a wide variety of breeds, including draft breeds, Warmbloods, Thoroughbreds, Standardbreds, Arabians, Morgans, Quarter horses, Appaloosas, American Paint horses, and many more. Terms such as tying-up, set fast, azoturia, and Monday morning disease have been used to describe this syndrome.

Classically, horses lose impulsion and develop a stiff, stilted gait, particularly involving the hindquarters. There is excessive sweating and a high respiratory rate due to pain. Horses may be unable to walk forward after resting due to firm, painful muscle contractures involving the back and hindquarters. Gluteal, biceps femoris, semitendinosus, and semimembranosus muscles are usually symmetrically affected with less frequent involvement of the forelimbs. Severe cases of rhabdomyolysis show signs of colic, become recumbent, and develop occult myoglobinuria. Attempts to move more severely affected animals may result in extreme pain, obvious anxiety, and exacerbation of the condition. Diagnosis of rhabdomyolysis is usually obvious based on the clinical signs, but measurement of serum muscle enzyme activities provides an assessment of the severity of muscle damage and confirms the diagnosis. The degree of elevation of muscle enzyme activity does not necessarily reflect the severity of clinical signs.

Treatment of Acute Rhabdomyolysis

If the attack occurs during exercise some distance from where the horse is normally stabled, the horse should not be made to walk home. If an attack occurs at a competition, the horse should be treated there and should not be transported home over a long distance for at least 24–48 hours.

The objectives of treatment are to relieve anxiety and muscle pain, as well as correct fluid and acid–base deficits (Table 7.2). Acepromazine is helpful in relieving anxiety and may increase muscle blood flow but should not be given to dehydrated horses due to hypotensive effects that produce recumbency. Alternatively, xylazine may provide short-term relief or in very painful horses, and detomidine combined with butorphanol provides

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Table 7.2. Potential medications that can be used to treat acute rhabdomyolysis in the horse.

Category	Drug	Dose	Comment
Tranquilizer	Acepromazine	0.04–0.07 mg/kg IV	May be hypotensive; use caution if dehydrated
	Xylazine	0.4–0.8 mg/kg IV	
	Detomidine	0.02–0.04 mcg/kg IV	
	Butorphanol	0.01–0.04 mg/kg IV	Use with xylazine/detomidine
Anti-inflammatory	Phenylbutazone	2.2–4.4 mg/kg IV or oral	Use caution if dehydrated
	Flunixin meglumine	1.1 mg/kg IV or oral	Use caution if dehydrated
	Ketoprofen	2.2 mg/kg IV	Use caution if dehydrated
	DMSO	1–2 mg/kg IV or oral	Use as a solution diluted to less than 20%
Pain control: CRI	Lidocaine	1.3 mg/kg IV followed by 0.05 mg/kg/min IV	
	Detomidine	0.22 mcg/kg IV followed by 0.1 mcg/kg/min IV	
	Butorphanol	13 mcg/kg/h	
Muscle relaxant	Methocarbamol	5–22 mg/kg IV slowly	
	Dantrolene	2–4 mg/kg oral QID	

excellent sedation and analgesia. NSAIDs such as ketoprofen, phenylbutazone, or flunixin meglumine provide additional pain relief. Analgesic treatment is continued to effect, but most horses are relatively pain free within 18–24 hours. For horses with extreme pain and distress, a constant rate infusion of detomidine, lidocaine, or butorphanol can make the difference between adequate time for recovery and euthanasia.

Intravenous or intragastric dimethyl sulfoxide can be used as an antioxidant, anti-inflammatory, and osmotic diuretic in severely affected horses. Muscle relaxants such as methocarbamol seem to produce variable results, possibly depending on the dosage used. Dantrolene sodium in severely affected horses may decrease muscle contractures and possibly prevent further activation of muscle necrosis. This can be repeated in 4–6 hours.

Severe rhabdomyolysis can lead to renal compromise due to the ischemic and the combined nephrotoxic effects of myoglobinuria, dehydration, and NSAIDs. The first priority in horses with hemoconcentration, or myoglobinuria, is to reestablish fluid balance and induce diuresis. In mild cases, administration of fluids via a nasogastric tube may be adequate, but generally fluids are better given intravenously. Balanced polyionic electrolyte solutions are best. If severe rhabdomyolysis is present, then isotonic saline or 2.5% dextrose in 0.45% saline may be necessary because horses often have hyponatremia, hypochloremia, and hyperkalemia.[34] If hypocalcaemia is present, then supplementing intravenous fluids with 100–200 mL of 24% calcium borogluconate is recommended, but serum calcium should not exceed a low normal range. Affected animals are usually alkalotic, making bicarbonate therapy inappropriate.

Etiology of Exertional Rhabdomyolysis

Some horses develop exertional muscle damage as a result of extrinsic nutritional or environmental factors. These sporadic cases of ER are usually amenable to treatment once the extrinsic cause of muscle damage is identified. Some horses, however, develop chronic ER, and many of these cases are due to an intrinsic and inherited dysfunction of muscle metabolism or muscle contraction.

Sporadic Exertional Rhabdomyolysis

The most common extrinsic cause of sporadic ER is exercise that exceeds the horse's underlying state of training. Horses that are advanced too quickly in their training, those that are only ridden sporadically while being continually fed full rations, and horses performing strenuous exercise such as racing or endurance riding without sufficient conditioning commonly develop rhabdomyolysis. In addition, rhabdomyolysis may be more common in horses exercising during an outbreak of respiratory disease. Both equine herpesvirus 1 and equine influenza virus have been implicated as causative agents.[23,26]

Dietary Imbalance

Horses consuming a high-grain diet appear to be more likely to develop ER than horses fed a low-grain or fat-supplemented diet.[42,43] The grain itself may not be responsible for rhabdomyolysis; however, high starch intake may trigger rhabdomyolysis in horses with particular myopathies such as RER and PSSM.

Electrolyte depletion in horses can occur due to dietary deficiency and losses in sweat with strenuous

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exercise. Sodium, potassium, magnesium, and calcium play key roles in muscle fiber contractility. With severe acute electrolyte depletion such as that found following endurance exercise, serum electrolytes may be below normal ranges.[11] With chronic dietary depletion, however, serum concentrations may not reflect total body electrolyte imbalances.

Work by Harris et al. established renal fractional excretions as a technique to evaluate electrolyte concentrations in horses with chronic ER.[28] Blood and urine samples are obtained concurrently, and creatinine and electrolyte concentrations measured in both. Serum creatinine/urine creatinine multiplied by the reciprocal for urine and serum electrolyte concentrations ×100 provides the fractional excretion of a particular electrolyte. It can be difficult, however, to obtain consistently representative renal fractional excretions of electrolytes in horses.[53] Hypokalemia has also been suggested to play a role in chronic ER, although it is not a common finding in horses consuming adequate quantities of forage.[4] Supplementation with good quality forage or 30 g of KCl/day (light salt) is recommended for horses with low renal fraction excretion of potassium.

Vitamin E and Selenium

Another postulated cause of sporadic ER is the increased generation of free radicals from oxidative metabolism associated with exercise. It has not been shown whether horses that experience repeated episodes of ER may generate more free radicals than normal horses. Primary selenium deficiency has rarely been demonstrated as a cause of ER.[60] In fact, many racehorses with chronic ER have higher concentrations of selenium and vitamin E due to zealous dietary supplementation by the owner. Adequate values for blood selenium are greater than 0.07 mcg/mL.

Horses with low serum vitamin E may present with muscle soreness, muscle fasciculations, muscle weakness, and loss of muscle mass. In early stages this may be due to a reversible myopathic process.[6] After years of deficiency, oxidative damage to motor nerves can result in generalized denervation typical of equine motor neuron disease.[16] Adequate values for serum vitamin E are above 2.0 mcg/mL.

Chronic Exertional Rhabdomyolysis

Horses that have repeated episodes of ER from a young age, from the time of purchase, or when they are put back into training after a long period of rest may have an underlying intrinsic abnormality of muscle function. Many of the horses with intrinsic muscle defects have repeated episodes of rhabdomyolysis with minimal exercise, even when the dietary and training recommendations for sporadic ER are followed. Five specific intrinsic causes of ER have been identified to date: MH, RER, PSSM1, PSSM2, and myofibrillar myopathy (MFM). It is likely that there are other specific causes that have yet to be identified (idiopathic chronic ER).

In all of these intrinsic forms of chronic ER, it appears that there are specific environmental stimuli that are necessary to trigger muscle necrosis in genetically susceptible animals. Horses cannot be cured of their susceptibility to this condition, but if the specific disease is identified, changes in management can be implemented to minimize episodes of rhabdomyolysis.

Malignant Hyperthermia

Prevalence

A genetic form of MH due to an autosomal dominant mutation in the skeletal muscle ryanodine receptor (*RYR1*) has been identified in 1% or fewer of American Quarter horses.[2]

Clinical Signs

Horses with MH may develop classic signs under general anesthesia of excessive body temperature, rigor, metabolic acidosis, and death.[1] In addition, affected horses may show signs of ER and high body temperatures. Some MH-affected horses have died suddenly after an episode of ER. The MH defect can coexist with the *GYS1* mutation for PSSM1.[45] The combination of these two mutations makes signs of ER more severe and increases recurrence of high serum CK, and horses do not respond as well to changes in diet and exercise.

Diagnosis

Genetic testing is recommended in Quarter horses and Paint horses with difficult to manage forms of PSSM or a family history of post-anesthetic complications.

Recurrent Exertional Rhabdomyolysis

Prevalence

About 5%–10% of Thoroughbred racehorses develop ER during a racing season, and 75% of these horses have more than four episodes in 4 months.[41] Approximately 6% of National Hunt horses[68] 13% of polo horses[54] and 6% of Standardbred horses[32] develop ER. Horses with a nervous disposition, especially fillies, are highly predisposed. Research suggests that a subset of Thoroughbred horses with chronic ER have a specific form of ER, denoted as RER.[35,72] RER appears to be an inherited, intermittent, stress-induced defect in the regulation of muscle contraction; however, a specific genetic cause of RER has not been established despite considerable research.[35] Recurrent episodes of rhabdomyolysis in Standardbreds and Arabian horses may be due to a similar abnormality, but this has not been confirmed.[32]

Clinical Signs

Horses with RER have intermittent elevations in serum CK activity.[55] Episodes of muscle stiffness, sweating, and firm muscle contractures often occur in horses once they become fit and are frequently associated with excitement at the time of exercise. In Thoroughbreds, rhabdomyolysis occurs most frequently during training when horses are held to a slow gallop.[41] In Standardbreds, rhabdomyolysis often occurs 15–30 minutes into jogging.[67] Obvious clinical signs are rare after racing. A history of poor performance and elevated serum AST and CK may be the only presenting complaints in some

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horses. Older Thoroughbreds used as riding horses may have very intermittent episodes of rhabdomyolysis associated with lay-up of fit horses. Event horses often develop clinical signs after the steeplechase phase of a 3-day event, or in the "10-minute box," or less commonly during the cross-country phase. Muscle stiffness and reluctance to collect may be present on a continual basis between episodes in some of these older horses. Arabian horses often develop clinical signs with little exertion, frequently in association with excitement.

Etiology

RER is characterized by abnormal sensitivity of intact muscle bundles to contractures induced by the addition of caffeine or halothane to a muscle bath.[8,35] Elevated myoplasmic calcium concentration has been reported in muscle of horses with acute ER and, in the face of caffeine, in myoblasts derived from Thoroughbreds with RER.[36,40]

There are physiological similarities between the contracture results of RER and contracture tests for MH; however, biochemical studies of isolated muscle cell membranes[79] and linkage analysis[17] do not support an identical biochemical basis for MH and RER. The increased halothane sensitivity of the muscles in RER horses may explain why many Thoroughbreds with RER develop rhabdomyolysis under halothane anesthesia.

Factors that trigger rhabdomyolysis in susceptible horses include gender, temperament, excitement, stress, dietary starch, exercise duration/intensity, season, and lameness.[66] Females are most commonly afflicted with RER (67% female to 33% male), particularly those that are 2 years of age and in race training.[41,50] Nervous horses are five times more likely to develop RER, and horses with lameness are four times more likely to develop rhabdomyolysis. Susceptible horses receiving more than 5 kg of concentrate feed (oats, corn, molasses mix) are more likely to develop rhabdomyolysis than those receiving 2.5 kg of concentrate feed/day.[43] Dietary effects of high carbohydrates in RER may in part be related to the psychogenic effects of grain on excitability. Glycogen storage does not increase substantially in horses with RER.[72] Inclement weather has been cited as a trigger of ER, and rhabdomyolysis is reported more commonly in the autumn and winter in the United Kingdom.[29]

A contribution of reproductive hormones to triggering ER has been postulated because the incidence of RER appears to be highest in mares. Many owners report that episodes of rhabdomyolysis occur most commonly during estrus, but in one study of racehorses, no direct correlation was shown between progesterone fluctuations and serum CK activity.[22] It is likely that the estrus cycle is one of many factors that combine to trigger ER in susceptible horses. Many racetrack practitioners report that the incidence of ER declines when susceptible mares are treated with testosterone. Hypothyroidism and lactic acidosis have also been suggested as a cause of ER but never substantiated.

Diagnosis

Acute cases of ER are readily diagnosed by serum muscle enzymes. Identification of RER in a horse that presents with a history of poor performance unrelated to signs typical of tying-up may be facilitated by a submaximal exercise test with pre- and 4-hour post-serum CK activity assessed. This test is useful for horses with concurrent problems but is not adequate to screen for RER in asymptomatic horses.

Muscle biopsies from horses with RER that are in training have increased numbers of centrally located nuclei in mature muscle fibers. They may have evidence of varying stages of muscle degeneration and regeneration, and they have normal to slightly increased subsarcolemmal muscle glycogen staining.[72] Histopathologic changes are often lacking in horses that have been laid up for a period of time prior to biopsy.

Polysaccharide Storage Myopathy (PSSM)

Prevalence

Recent research has shown that there are at least two forms of glycogen storage disorders in horses. PSSM1 is due to an autosomal dominant gain of function mutation in the glycogen synthase gene (*GYS1*).[49] This mutation is found in at least 20 different breeds of horses in Europe and North America, including Quarter horses; American Paint horses; Appaloosa horses; draft horse breeds including American Cream, Belgian, Percheron, Gypsy Vanner, Cob Normand, Trekpaard, Haflinger, Morgan, Mustang, and Rocky Mountain horse; Tennessee Walking Horse breeds; mixed breed horse; Cobs; Hanoverians; Rhinelanders; and Warmblood horses of unspecified type.[5,48,61] The prevalence of PSSM due to the *GYS1* mutation ranges from 8% in Quarter horses and Paints to 36% in Belgian draft horses, with no reported cases in the Thoroughbreds, Standardbreds, and Arabians tested.

PSSM2 is the term designated for those horses lacking the *GYS1* mutation that have with the histopathologic characteristic of abnormal-appearing amylase-sensitive or amylase-resistant polysaccharide in muscle biopsies of horses.[46] Importantly, the term PSSM2 does not indicate a specific etiology since no specific genetic mutations or biochemical aberrations have been described in these horses as of yet. The assessment of amylase-sensitive aggregates of glycogen is subjective and impacted by tissue handling, leaving considerable room for false-positive diagnoses and overlap with a diagnosis of RER. PSSM2 appears to account for about 25% of PSSM cases in Quarter horse-related breeds and 80% of PSSM cases in many Warmblood breeds.[46,47] In the United Kingdom approximately 35% of PSSM cases diagnosed by muscle biopsy would be classified as PSSM2.[62] The acronyms EPSM and EPSSM have also been used for polysaccharide storage myopathy (PSSM), although they do not indicate a specific genotype.[76,77]

Clinical Signs

PSSM1

Horses with PSSM1 often have a calm and sedate demeanor.[71] Clinical signs of muscle stiffness, reluctance to exercise, exercise intolerance, or overt muscle contractures and reluctance to move usually are apparent at the commencement of training.[20] Most horses have numerous episodes of ER or a consistent history of poor

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performance; however, some mildly affected horses are asymptomatic or have only one or two episodes of ER per year. In Quarter horses, PSSM1 has the highest frequency in halter horses, followed by pleasure horses and working cow horses.[64] It is uncommon in racing Quarter horses. Serum CK activities are often elevated in untreated Quarter horses, even when horses are rested, and while clinical signs are active, CK will usually increase by 1,000 U/L or more 4 hours after 15 minutes of exercise at a trot.[72] Muscle atrophy, renal failure, and severe colic-like pain are less common presenting complaints.

Draft horses with PSSM1 may be asymptomatic if not in work but may also show signs of a generalized decrease in muscle mass and weakness in the hindlimbs with difficulty rising.[74] ER can be a debilitating feature of PSSM1 in draft breeds. Although shivers was suggested to be a sign of PSSM, recent studies show there is no causal relationship.[21] Draft horses with PSSM1 often have only a mild elevation in serum CK and AST. A small percentage of Warmbloods are afflicted with PSSM1 and have an increased incidence of episodes of ER.[33]

PSSM2

In Quarter Horses and light breeds, chronic ER is the predominant clinical sign of PSSM2.[47] Elevations in CK and AST are common. One of the most common presenting complaints in Warmbloods with PSSM2 is a gait abnormality characterized by lack of impulsion and shifting undiagnosed lameness.[34] By 10 years of age, Warmblood horses with PSSM2 show signs of sore muscles, poor performance, reluctance to go forward under saddle, reluctance to collect, and in some cases slow onset of topline atrophy, particularly if taken out of work.[38] ER occurs infrequently in Warmbloods with PSSM2, and post-exercise increase in serum CK activity is absent to infrequent in Warmbloods.[38]

Diagnosis

PSSM1

PSSM1 is most readily diagnosed by genetic testing for the *GYS1* mutation performed on whole blood or hair root samples.[49] The distinctive features of PSSM1 in muscle biopsy samples are numerous subsarcolemmal vacuoles and dense, crystalline periodic acid-Schiff (PAS)-positive, amylase-resistant inclusions in fast-twitch fibers.[71] A false-negative diagnosis of PSSM1 by muscle biopsy may occur if biopsy samples are small or if horses are less than 1 year of age.[15]

PSSM2 must still be diagnosed by muscle biopsy at present.[66] Muscle biopsies from horses with PSSM2 have increased PAS staining for glycogen and aggregates of granular PAS-positive polysaccharide in the cytoplasm or under the sarcolemma. The PAS-positive polysaccharide in PSSM2 is frequently but not always amylase sensitive. False-positive diagnosis is possible for PSSM2 in highly trained horses that normally have higher muscle glycogen concentrations or in formalin-fixed sections, which show a greater deposition of subsarcolemmal glycogen in healthy horses. Other features that may be present with both forms of PSSM include muscle necrosis and macrophage infiltration of myofibers, regenerative fibers, and atrophied type 2 fibers.

Etiology

Muscle glycogen concentrations in horses with PSSM1 are 1.5–4 times normal.[71] Increased muscle glycogen concentrations in PSSM1 appear to be due to increased and unregulated glycogen synthase activity as a result of a gain-of-function mutation in the glycogen synthase 1 gene (*GYS1*). Horses with PSSM show increased glycogen synthase activity both in the basal state and when stimulated by glucose-6-phosphate.[49] Elevated insulin in the bloodstream can further enhance glycogen synthase activity. Abnormal amylase-resistant polysaccharide appears to result from production of a polysaccharide with a higher ratio of straight glucose chains, which are created by glycogen synthase, relative to branched chains, which are created by the less active glycogen branching enzyme. Rhabdomyolysis in PSSM1 appears to result from a deficiency of energy within individual contracting muscle fibers.[3] The genetic defect combined with a high-starch diet appears to produce substrate-limited muscle oxidative metabolism by both impairing production of acetyl-CoA from glycogen and preventing lipolysis and delivery of free fatty acids to skeletal muscle mediated by insulin release.[73] Clinical signs of muscle pain in horses with the *GYS1* mutation may be exacerbated by enhanced individual insulin sensitivity as well as by meals that produce elevated blood glucose and insulin levels.[59] The etiology of PSSM2 is not known at present.

Myofibrillar Myopathy (MFM)

Diagnosis

MFM is a recently identified disorder in horses presenting with exercise intolerance or intermittent ER that is defined by specific histopathology.[65,69] First and foremost MFM horses have cytoplasmic aggregates of the cytoskeletal protein desmin in scattered muscle fibers. Desmin functions to align sarcomeres at the Z-disc and tether them to the cell membrane. Other ultrastructural derangements evident in electron microscopy include disrupted myofibrillar alignment, ectopic accumulation of cytoskeletal proteins, and Z-disc degeneration. In some cases, MFM is also characterized by cytoplasmic aggregates of glycogen similar to PSSM2. This is likely because pools of glycogen form within disrupted myofibrils. MFM may represent a more extreme subset of PSSM2. Muscle glycogen concentrations in horses with MFM are similar to controls.

Warmbloods

Warmblood horses diagnosed with MFM have an insidious onset of exercise intolerance notable by 6–8 years of age characterized by a lack of stamina, unwillingness to go forward, inability to collect, abnormal canter transitions, and inability to sustain a normal canter.[65] Unresolved hindlimb lameness, stiffness, muscle pain, and, rarely, an episode of ER are reported. Serum CK and AST activities are usually within normal limits unless samples are taken in conjunction with ER.

Arabians

Arabian endurance horses diagnosed with MFM usually have a history of intermittent elevations in serum

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CK activity after endurance rides (>10,000 U/L) or during exercise that follows a week or more of rest.[69] Horses do not necessarily always show the same degree of pain, sweating, and reluctance to move as is frequently seen in other forms of acute ER. Myoglobinuria can be observed in horses with only mild muscle stiffness. Between episodes, the heart rate, lactate, and CK and AST responses to exercise are normal.

Management of Chronic Forms of Exertional Rhabdomyolysis

Expectations

Most horses with chronic forms of ER always have an underlying predilection for muscle soreness. However, with proper management, most cases can have successful competitive careers. A few RER horses are extremely difficult to manage and may be retired from racing. Most PSSM1 horses can be successful pleasure and trail horses but usually do not achieve success in upper-level dressage or fast-paced activities such as barrel racing. ER can be well managed in Quarter Horses and Arabians with PSSM2. Warmbloods with PSSM2 will improve with diet and exercise recommendations, but they may not attain a higher level of performance in dressage.

Following clinical episodes, more than 80% of PSSM1 and PSSM2 horses show notable improvement with adherence to both the diet and exercise recommendations provided below.[20,31] There is, however, a wide range in the severity of clinical signs shown by horses with PSSM1; horses with severe or recurrent clinical signs require more stringent adherence to diet and exercise recommendations to regain muscle function. PSSM1 horses that also have the mutation for MH do not respond as well to diet and exercise recommendations and may continue to develop ER with the possibility of a fatal episode.[45]

Rest

Stall rest appears to be counterproductive and predisposes RER and PSSM horses to further episodes of rhabdomyolysis. Providing daily turnout with compatible companions can be very beneficial. It decreases anxiety in RER horses and enhances energy metabolism in PSSM horses. PSSM1 horses that are confined for days following an episode of rhabdomyolysis often have persistently elevated serum CK activity. In contrast, PSSM1 horses kept on pasture with little grain supplementation often show few clinical signs of rhabdomyolysis and have normal serum CK activity.

Environment

If excitement is a triggering factor for RER, stressful environmental elements should be minimized. Many horses respond to a regular routine including feeding prior to other horses and training before other horses, especially if the horse becomes impatient while waiting. Other ways to decrease excitement include housing in an area of the barn where horses are not always walking past and next to calm companionable horses. The use of hot walkers, exercise machines, and swimming pools should be evaluated on an individual basis, because some horses develop rhabdomyolysis when using this type of equipment. Horses that develop rhabdomyolysis at specific events, such as horse shows, must be reconditioned to decrease the stress level associated with such events.

Most PSSM horses are calm and not easily stressed. They do best when turned out on large pastures without lush grass where they move about on a daily basis. Grazing muzzles may be of benefit to PSSM horses for periods when grass is particularly lush.

Exercise

Regular daily exercise is important for managing all forms of ER. Thoroughbreds and Standardbreds with RER should avoid days off exercise. This prevents the elevations in serum CK activity that occur after a day of rest.[55] Following chronic episodes of ER in Thoroughbreds and Standardbreds, mild, calm, low intensity daily exercise (less than 15 minutes) or preferably extensive daily turnout is recommended until serum CK is less than 1,500 U/L. Thoroughbred racehorses often develop rhabdomyolysis when riders fight to keep horses at a slower speed (gallop exercise); therefore, this should be avoided.[41] Standardbreds often develop ER after 15–30 minutes of submaximal jogging, and therefore interval training and reduction of jog miles to no more than 15 minutes per session is recommended.[74] For riding horses with RER, a prolonged warm-up with adequate stretching is recommended. Rest periods that allow horses to relax and stretch their muscles between 2- and 5-minute periods of collection under saddle may be of benefit. Event horses may require training that incorporates calm exposure to speed work to prevent rhabdomyolysis, as well as interval training at the speeds achieved during competitions.

Important principles to follow when starting exercise programs in PSSM horses include:

1. Providing adequate time for adaptation to a new diet prior to commencing exercise
2. Recognizing that the duration of exercise is more important than restricting the intensity of exercise
3. Ensuring the exercise is gradually introduced and consistently performed
4. Minimizing any days without some form of exercise

If horses have experienced an episode of ER recently, 2 weeks of turnout and diet change are often beneficial prior to recommencing exercise. Exercise should be very relaxed, and the horse should achieve a long, low frame without collection. For many horses this is most readily achieved in a round pen or on a lunge line. Successive daily addition of 2-minute intervals of walk and trot beginning with only 4 minutes of exercise and working up to 30 minutes after 3 weeks is recommended. Advancing the horse too quickly often results in poor adaptation or an episode of ER and repeated frustration for the owner. Warmblood horses with PSSM may benefit from long, low lunging prior to exercise under saddle.

Work under saddle after 3 weeks of ground work should be a gradually accelerating program that adds 2-minute intervals of collection or canter to the initial relaxed warm-up period at a walk and trot. Unless a

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horse shows an episode of overt ER during the initial first 4 weeks of exercise, reevaluating serum CK activity is not helpful for the first month. This is because it is very common to have subclinical elevations in CK activity when exercise is reintroduced in PSSM horses, and a return to normal levels often requires 4–6 weeks of gradual exercise.[59] Keeping horses with PSSM fit seems to be the best prevention against further episodes of ER. This gradual approach aims to enhance the oxidative capacity of skeletal muscle, which in most Quarter horses is very low but can be increased with daily exercise. The objective of enhancing oxidative metabolism is to facilitate the metabolism of fat and blood-borne fatty acids as energy substrates.

Dietary Management

A nutritionally balanced diet with appropriate caloric intake and adequate vitamins and minerals is the core element of treating all forms of ER. Dietary recommendations are summarized in Table 7.3. Diets with a restricted amount of starch and sugar and supplemental calories supplied with fat are the basis for dietary management. In general, grass hay is preferable to alfalfa hay. For RER Thoroughbreds and Standardbreds, the challenge is supplying an adequate amount of calories in a highly palatable feed to meet daily energy demands. Controlled experimental studies using Thoroughbreds with RER show that serum CK activity is significantly lower when horses are fed a specially formulated high-fat, low-starch feed rather than an isocaloric amount of high-starch grain.[42,55] Given the close relationship between nervousness and RER, assuaging anxiety and excitability by reducing dietary starch and increasing dietary fat may decrease susceptibility by making these horses calmer prior to exercise. Some racehorse trainers prefer to supplement with a titrated amount of grain 3 days prior to a race if horses are on a low-starch, high-fat feed to increase the horse's energy during the race.

For PSSM1 horses, the challenge can be to provide adequate fat for energy metabolism while preventing excessive weight gain. If horses are overweight, reducing caloric intake by using a grazing muzzle, restricting hay to 1.5% of body weight, and providing a vitamin/mineral ration balancer is recommended. The NSC content of hay should be <12% to avoid insulin release.[9] Adding excessive calories in the form of fat to an obese horse is inadvisable. Until horses are a normal weight, fat metabolism can be enhanced by riding horses after a 5- to 8-hour fast as a means to elevate plasma-free fatty acids.

Once a horse has achieved the desired body weight, feeds low in starch and sugar combined with dietary fat can be introduced. The starch and sugar content of the diet of PSSM1 horses must be managed more stringently than for RER horses (Table 7.3). Owners report that this type of diet improves clinical signs of muscle pain, stiffness, and exercise tolerance in draft horses, Warmbloods, Quarter horses, and other breeds with PSSM1 when combined with the recommended exercise program.[20,31,75] The addition of fat alone is not beneficial, and an exercise program must be instituted for PSSM1 horses to show clinical improvement. In the author's experience, there is a great deal of variation in individual needs for fat supplementation, and this should be balanced with the horse's weight.

Warmbloods with PSSM2 or MFM are fed similar NSC to PSSM1; however, there is less evidence that very low NSC or high-fat diets are the most effective means to manage these disorders. For MFM horses and horses with topline muscle atrophy, amino acid supplements can be used to enhance muscle mass.[25,65] There are an increasing number of whey-based amino acid supplements on the market for horses.

A number of well-balanced low-starch, high-fat commercial diets are suitable for horses with RER and

Table 7.3. Feeding recommendations for an average sized 1,000-lb horse with exertional rhabdomyolysis due to recurrent exertional rhabdomyolysis (RER) or polysaccharide storage myopathy (PSSM).

	RER			PSSM		
Exercise intensity	Light	Moderate	Intense	Light	Moderate	Intense
Caloric intake (megacalories/d)	20	25	33	18	23	30
Percentage of daily digestible energy as NSC	15–20	15–20	15–20	Less than 10	Less than 10	Less than 10
Percentage of daily digestible energy as fat	15	15–20	20–25	20	15–20	15–20
Selenium mg/d	2.2	2.8	3.1	2.2	2.8	3.1
Vitamin E[a] units	1,000	2,000	4,000	1,000	2,000	4,000
Daily amount (lbs) of 12% NSC, 10% fat concentrate[b]	5	7	10	4	6	8
Grass hay (lbs) with less than 12% NSC by weight	15–20	15–20	15–20	15–20	15–20	15–20
Sodium chloride (g)	34	34	41	34	34	41

[a] Natural vitamin is preferable to synthetic vitamin E due to better absorption.
[b] Concentrate such as Re-Leve made by Kentucky Equine Research for horses with exertional myopathies.

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PSSM; however, the amount of starch and sugar can vary widely, as can the type of fat. Not all of these feeds are equally effective. At present, the NSC content of equine feed products is not listed on the feed tag, and consultation with the feed manufacturer is necessary to obtain this information. Nutritional support is available through most feed manufacturers in designing an appropriate diet. There are published peer-reviewed studies to show the beneficial effect of one commercial diet on both RER and PSSM.[55,59]

Supplements

Horses require daily dietary supplementation with sodium and chloride either in the form of loose salt (30–50 g/day) or a salt block. Additional electrolyte supplementation is indicated in hot, humid conditions. Some studies suggest that electrolyte imbalances, as reflected by low urinary fraction excretion of sodium or high dietary excretion of phosphorus, may contribute to ER, although others have not found a consistent abnormality.[28,29]

A number of supplements are purported to decrease lactic acid buildup in skeletal muscle of ER horses. These include sodium bicarbonate, B vitamins, branched-chain amino acids, and dimethylglycine. Because lactic acidosis is no longer implicated as a cause for ER, it is difficult to find a rationale for their use.

Medications

Low doses of acepromazine prior to exercise have been used in RER horses prone to excitement. A dose of 7 mg IV 20 minutes before exercise is reported to make horses more relaxed and manageable.[24] Reserpine and fluphenazine, which have a longer duration of effect, are also used for this purpose. Horses given fluphenazine may occasionally exhibit bizarre behavior. Use of tranquilizers may only be necessary when horses are in their initial phase of training and accommodation to a new environment because they obviously cannot compete on these medications.

Dantrolene sodium acts to decrease release of calcium from the calcium release channel in skeletal muscle and is used to treat MH. Dantrolene given to horses with RER 90 minutes before exercise prevented abnormal elevations in CK following exercise.[52] A dose of 800 mg of dantrolene was given to Thoroughbred horses in the United Kingdom 1 hour prior to exercise and resulted in significantly lower post-exercise CK activity than a placebo.[19] It must be given 60–90 minutes prior to exercise and horses do not need to be fasted.[51]

Phenytoin (1.4–2.7 mg/kg PO BID) acts on a number of ion channels within muscle and nerves, including sodium and calcium channels. Therapeutic levels vary, so oral doses are adjusted by monitoring serum levels to achieve 8 µg/mL and not to exceed 12 µg/mL.[7] Drowsiness and ataxia are evidence that the dose of phenytoin is too high and the dose should be decreased by half. Initial dosages start at 6–8 mg/kg orally twice a day for 3–5 days. If the horse still experiences rhabdomyolysis but is not drowsy, the dose can be increased by 1 mg/kg increments every 3–4 days.

Intramuscular injections of vitamin E and selenium are used in an attempt to prevent RER. Ensuring adequate oral intake may prevent the muscle soreness associated with IM injections.

Some mares appear to exhibit signs of ER during estrus, and it may well be beneficial in these horses to suppress estrus behavior using progesterone injections. Testosterone and anabolic steroids are used at racetracks to prevent signs of RER, but the efficacy has not been evaluated.

Adjunct Therapies

Massage, myofascial release, mesotherapy, stretching, and hot/cold therapy performed by experienced therapists may be of benefit in individual cases of ER.

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8

CHAPTER

Principles of Therapy for Lameness

SYSTEMIC/PARENTERAL

Drew W. Koch and Laurie R. Goodrich

Systemic administration of medications to treat musculoskeletal diseases in the horse mainly encompasses intravenous nonsteroidal anti-inflammatory drugs (NSAIDs), intramuscular (IM) polysulfated glycosaminoglycans (PSGAGs), and intravenous (IV) hyaluronan (HA). Less commonly used systemic medications include IV bisphosphonates, and a few other miscellaneous medications covered below. Excluding NSAIDs, there has been controversy regarding the use of the following medications in the horse and whether they reach high enough therapeutic levels at the intended area to be effective. Many (or most) of the drugs or nutraceuticals have been intended for systemic administration in humans, and levels high enough to be efficacious in the horse are often in question.

PARENTERAL NSAIDS

The most commonly used IV NSAIDs are phenylbutazone and flunixin meglumine. Recommended dosages are between 2.2 mg/kg q12 hours or 4.4 mg/kg q24 hours (phenylbutazone) and 1.1 mg/kg q24 hours to q12 hours (flunixin meglumine). Ketoprofen, carprofen, and firocoxib are less commonly used intravenously with ketoprofen and firocoxib now labeled for use in the United States (Ketofen® sterile solution, Zoetis, Parsippany, NJ; Equioxx, Merial, Inc., Duluth, GA). The NSAIDs mentioned above are most commonly used orally; however, in the acute case of inflammation when pain and swelling have not yet been addressed, the IV route of delivery is more effective and yields the desired pharmacologic effect faster.

Repetitive IV injections are not recommended due to increased likelihood in developing vasculitis. Oral formulations are recommended when a venous catheter is not in place and when repetitive dosing is necessary. For more information on specific NSAIDs, mechanism of action, dosages, and indications, see the section on oral/nutritional therapy later in this chapter.

POLYSULFATED GLYCOSAMINOGLYCANS (PSGAGS) AND PENTOSAN POLYSULFATE (PPS)

PSGAGs are a mixture of low molecular weight (approximately 6–10,000 Da) glycosaminoglycans (GAGs) that are very similar in structure to chondroitin sulfate, the major GAG in normal cartilage.[10] PSGAG administration has been associated with reducing the severity of clinical signs on both human and equine patients with OA.[35,58] Beneficial clinical effects are, at least in part, attributable to anti-inflammatory effects consisting of the inhibition of prostaglandin E_2 (PGE_2), cytokine release, and stimulatory effects on cartilage matrix components.[68] Modes of administration include IM and intra-articular (IA) routes. Elevated risks have been identified with IA administration; therefore, many practitioners still prefer to administer PSGAGs intramuscularly.[10,18] The efficacy of IM administration may be lower compared with direct injection into the joint;

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anecdotally, if a favorable response to IM administration occurs, it is rapid.

The PSGAG Adequan® (Luitpold Pharmaceuticals, Inc., Shirley, NY) is produced from the bovine trachea and lung and is in a class of drugs that exhibits chondroprotective properties in cartilage. Pentosan polysulfate (PPS; in a sodium or calcium derivative), within a similar class of medications, is produced from a plant source and is not licensed in the United States, but widely used in Europe and Australia.[49] In the United States, compounded versions can be obtained for use in horses. This section focuses on the use of IM PSGAG and PPS. Please refer to the chapter on IA therapy for IA use of PSGAG.

While there has been convincing evidence of the beneficial effects on cartilage, the exact mechanism of action remains unknown. It has been theorized that PSGAGs form stable complexes with fibronectin and collagen fibers and are deposited in cartilage.[3] Studies have shown that PSGAGs inhibit a plethora of degradative enzymes that contribute to the osteoarthritic (OA) process[32] and an anti-inflammatory role based on the inhibitory effects on leukocyte migration and interleukin levels.[20,39] A biosynthetic role has also been documented in terms of its stimulation of HA and collagen synthesis.[53,70]

Adequan® was originally intended for IA use; however, alterations in the complement pathway that potentiated sub-infective doses of *Staphylococcus aureus* resulted in IM administration becoming a more popular mode of administration.[34] There is less clinical data for IM use of the drug, although anecdotal reports of efficacy abound.[43] Reports in the literature reveal that IM administration of a single 500-mg dose of Adequan® in horses resulted in therapeutic levels of the drug in antebrachiocarpal, metacarpophalangeal, tibiotarsal, and distal interphalangeal joints within 2 hours of injection.[36] This was similar to a previous report examining radiolabeled IM PSGAG administration that led to measurable levels within the joints of interest for up to 96 hours; concentration also peaked at 2 hours after administration.[8] When efficacy of Adequan®, administered IM every 4 days for seven treatments, was evaluated in a full thickness cartilage defect model in ponies, no significant cartilage healing was noted.[69] Furthermore, Fubini et al. reported an inability of IM Adequan® to have a protective effect on methylprednisolone-injected joints in ponies.[26] When PSGAG (also IM every 4 days for 28 days) was used as a positive control in a carpal chip model evaluating the benefits of extracorporeal shockwave therapy (ESWT) after 70 days, PSGAG did not improve lameness scores nor produce any disease-modifying effects when synovial fluid and cartilage were evaluated.[24]

When PPS was first studied in animal models, a multimodal effect on proteoglycan preservation and production and inhibition of enzymes responsible for cartilage degradation were found.[27,29,31,51] *In vitro* studies using equine chondrocyte monolayers and articular cartilage explants indicate that both PSGAG and PPS stimulated proteoglycan production in cell monolayers; however, no difference in production was seen with cartilage explants.[21] This may suggest that common formulations might not be able to penetrate the extracellular matrix, bind chondrocytes, and stimulate proteoglycan production in the live animal.[21] While these aforementioned studies have shown inhibition of proinflammatory and degradative enzymes involved in progression of OA, other studies have not measured any significant difference.[11] When examined in the cartilage chip model,[50] significant improvement in cartilage fibrillation scores was noted in the PPS treatment group, which may indicate its benefit is likely through its disease-modifying effects.

Clinical Indications

Despite lack of scientific data to support IM administration of Adequan®, some clinicians believe that it has a beneficial effect on certain horses. In a survey of over 400 equine practitioners, IM Adequan® was one of the most commonly administered medications (not in the steroid category) in horses and was administered as a prophylactic therapy for OA.[18] The current dosing recommendations are 500 mg IM every 3–5 days for 5–7 treatments.

Data is currently lacking in the benefit of IM PSGAG administration. At this time, most veterinarians that recommend IM Adequan® do so for preventive therapy. IA use has shown greater benefit and is covered in the section regarding IA therapy. PPS has consistently shown greater benefit than Adequan® when administered IM, with current recommendations to administer 3 mg/kg IM once a week for 4 weeks.[49]

HYALURONAN

Hyaluronan (or hyaluronic acid, HA) is a large unbranched non-sulfated GAG composed of repeating units of D-glucuronic acid and *N*-acetyl glucosamine. It is an integral component of synovial fluid and articular cartilage.[23] HA actions in the synovial joint include increasing viscosity of synovial fluid, lubricating unloaded joints, restoring the rheologic properties of synovial fluid, and most importantly, inhibiting inflammation.[32,55] Many of these effects appear to depend on molecular size of the HA molecule administered, with molecular weights of $0.5–1 \times 10^6$ Da having the greatest ability of decreasing inflammation.[30]

IV administration of HA is noninvasive and a route that can be used to treat multiple joints. The benefits are most likely due to the effects in the synovial membrane because plasma half-life is less than an hour and is no longer detectable after 3 hours.[44,57] The effects of IV administration may be due to the fact that the synovial membrane is highly vascularized, which may allow greater exposure to synoviocytes than IA administration. The anti-inflammatory effects may also be due to the binding characteristics of HA to CD44 receptors present on several cell types within the synovial membrane, causing an inhibition of the expression of various cytokines.[66]

The only study to evaluate IV administration of HA in the horse used an osteochondral chip model of OA.[40] Horses treated with IV HA had reduced lameness, better synovial membrane scores, significantly lower total protein concentrations, and most importantly reduced prostaglandins in joints compared with horses receiving saline. Interestingly, HA did not have any effects on GAG content, synthetic rate, or morphologic scoring in articular cartilage, which lends credence to the theory that HA has mainly anti-inflammatory effects in joints.

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Intrasynovial injections are likely the most efficacious route of administration; however, the desire to treat multiple joints simultaneously has driven clinicians to use IV and oral preparations. Indications for IV HA are similar to those for IM PSGAGS in that IV HA reduces inflammation in one or multiple joints or prevents an inflammatory process in sport horses that are in heavy use. Although IA administration is the most effective way to deliver this drug to the joints, IA and IV administration are often concurrent. IV HA's effect is most likely through its anti-inflammatory nature and not through disease-modifying effects against the progression of OA.

In a recent review of the use of HA in all facets of clinical and benchtop research,[6] mention of the possibility of IV therapy was not made. At this time, IV use has only been evaluated in dogs and horses, and no human IV formulations exist. At this time, very little evidence-based medicine has been used to evaluate IV HA other than those listed previously. Likely, HA does have some beneficial anti-inflammatory effect, and anecdotal efficacy has been noted in our practice experience. At this time, IV HA appears to be safe, but whether true benefit is noted with its use remains to be shown.

Currently, the only labeled IV HA product on the market for equine is Legend® (Merial Inc., Duluth, GA). Current recommendations by the manufacturer include 40 mg intravenously weekly for three treatments. Another product, Polyglycan® (Bimeda, Inc., Oakbrook Terrace, IL), is licensed as a medical device and labeled for intrasynovial injection; however, it has been used off-label for IV and IM injection.[18] Although it has been used off-label, when a recent study examined its IV use, significant osteophytosis was identified on radiography in the treatment group compared with the control group in the equine osteochondral carpal chip fragment model.[25]

BISPHOSPHONATES

Bisphosphonates have been used for decades to inhibit loss of bone mass in human patients with osteoporosis associated with age or steroid administration and for rheumatoid arthritis.[12] Bisphosphonates inhibit osteoclast-mediated bone resorption,[12] a property that may play a role in conditions such as OA and navicular bone edema/sclerosis. Three studies in horses report the effects of the bisphosphonate tiludronate in clinical diseases such as navicular disease, vertebral pain related to OA of the thoracolumbar column, bone spavin, and bone loss due to cast immobilization.[13–15] All three studies reported reduced lameness, back soreness, and loss of bone with IV administration of the drug.

A second FDA-approved bisphosphonate licensed for treatment of navicular syndrome in horses, clodronate (Osphos®, Dechra Veterinary Products, Overland Park, KS), has emerged onto the market in recent years. With a similar mechanism of action to tiludronate, clodronate has also been hypothesized to be analgesic through inhibition of glutamate transport.[63]

A recent multicenter, double-blind, placebo-controlled clinical trial using clodronate showed that in horses with navicular syndrome confirmed with diagnostic analgesia and radiography, 75% saw at least one grade improvement in lameness at day 56 compared with 3% in the placebo group.[22] At day 180, 65% were still considered to have an improvement in their lameness. Clodronate has also been reported to successfully treat an acute, non-weight-bearing lameness in a horse that was diagnosed with coffin joint effusion and bone marrow edema of the second phalanx.[38] Clodronate was administered IM daily for 10 days at each recheck, and the horse's lameness improved.

More widespread use of bisphosphonates in horses has created concerns regarding their use in growing animals and in horses that have undergone therapy prior to sustaining a fracture. In one study examining healing and functional outcome in distal radial fractures in postmenopausal women undergoing bisphosphonate therapy, no significant difference was noted in healing or functional outcome between the treated and control group.[64] Other work in rabbit, dog, and sheep models, however, have mostly shown no effect of bisphosphonate therapy on fracture healing or that callus size and strength could be improved at initial stages of healing.[2,5,33,54] However, two studies noted long-term, high-dose therapy could delay healing but have no effect on mechanical integrity[46] or slow long-term callus remodeling.[48] These models all used varying bisphosphonates – incandronate, zoledronic acid, alendronate, ibandronate, and pamidronate – all of which are not currently licensed in equine medicine.

Due to the lack of available data, off-label use or use of bisphosphonates in young, growing horses cannot be recommended at this time. Further studies regarding long-term effect on bone density, ability of bone to heal after administration, and duration of action after administration are still needed.

Current recommendations for Osphos® administration are 1.8 mg/kg by IM injection with a maximum dose of 900 mg per horse (e.g. 900 mg dose standard for a 500-kg animal); recommendations to divide the dose between three sites are also given (Osphos®).[a] In a study of 146 horses, 10 horses developed mild signs of colic within the first 2 hours of treatment, but all resolved with minimal treatment.[22] For this reason, premedication with an NSAID is not recommended and, in our experience, rarely required. Due to renal excretion,[56] baseline creatinine values are recommended prior to starting a course of bisphosphonates. Concurrent administration with other nephrotoxic medications should also be avoided.

TETRACYCLINES

The anti-inflammatory effect of tetracyclines, independent of their antimicrobial role, has been well documented for multiple disease processes.[4,41,52,71] When more recently evaluated in a rodent model of acute inflammation, both minocycline and doxycycline were found to be effective through inhibition of TNF-α and nitric oxide.[45] Furthermore, and more relevant to equine practitioners, their use as disease-modifying osteoarthritic drug (DMOAD) has also been established.[7,60,65,72]

When these studies were translated to *in vitro* work with equine synoviocytes, doxycycline was noted to be chondroprotective through inhibition of specific matrix metalloproteinases (MMPs) that are known to cause

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progression of OA; their effect was also measured at a dose lower than what's required to inhibit bacterial growth.[19] Follow-up work from the same group reported that orally administered doxycycline was able to penetrate and accumulate within the synovial fluid,[62] and when administered to horses orally at 5 mg/kg every 24 hours for four doses, synovial fluid obtained from treated horses had significantly reduced MMP-13 expression.[47] At that time, the authors supported the use of doxycycline as a disease-modifying drug for OA.[47]

ESTROGEN

Estrogen therapy has been used to treat intermittent upward fixation of the patella in horses.[17] It has been hypothesized that estrogen affects muscle cell metabolism and muscle tone leading to the anecdotal benefit sometimes reported. However, no studies currently exist examining whether this therapy is beneficial as a medical treatment. Recommended therapy is 1 mg of estradiol cypionate IM for every 45 kg of body weight (i.e. 11 mg/500 kg) once weekly for 3–5 weeks.[17] Concurrent anti-inflammatories along with exercise to strengthen the quadriceps muscles are usually recommended as well.

METHOCARBAMOL (ROBAXIN)

Methocarbamol, marketed under the trade name Robaxin in the United States and Canada, is a centrally acting muscle relaxant commonly prescribed for various inflammatory and traumatic muscle disorders and to treat muscular spasms in the horse.[42] Methocarbamol produces variable results, but it seems to be most beneficial in horses that are prone to rhabdomyolysis or back soreness secondary to musculoskeletal disease of the axial skeleton or hind limbs. At our hospital, methocarbamol is commonly prescribed in conjunction with gabapentin in horses with suspected axial skeleton disease with associated secondary epaxial muscle sensitivity. Horses are treated for 4–8 weeks, while IA therapy (articular process joints, sacroiliac joint, etc.) is allowed time to improve the horse's comfort. Previous publications have reported a dose range of 4.4–55 mg/kg.[42] While an IV formulation can be obtained, oral tablets are now available for use in veterinary medicine.

GABAPENTIN

Gabapentin, an analog of γ-aminobutyric acid (GABA), has been shown to have analgesic effects through modulation of voltage-gated calcium ion channels.[28] It is used as an antiepileptic medication and for the treatment of neuropathic and chronic pain in humans.[67] Very little data exists to support its analgesic properties in animals,[1,9,61] although small numbers of case reports have found it beneficial as one component of multimodal analgesia for head shaking in temporohyoid osteoarthropathy (THO) and for chronic pain in cases of laminitis.[16,59]

At our hospital, gabapentin is most commonly prescribed in conjunction with methocarbamol to combat chronic back soreness secondary to pain associated with the axial skeleton or as a component of multimodal analgesia for musculoskeletal pain refractory to NSAID and opiate treatment. Dosages range from 5 to 20 mg/kg PO two to three times daily with dosages being titrated to effect.[37]

Manufacturer's Details

[a]Osphos®, Dechra Veterinary Products, Overland Park, KS.

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TOPICAL/LOCAL

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Topical and local therapies are commonly used to treat equine musculoskeletal diseases, both acutely when inflammation is most pronounced and chronically when ongoing inflammation results in soreness and/or lameness. Although NSAIDs are commonly used orally to control musculoskeletal inflammation and intravenously in the acute stages of inflammation, clinicians often look for local modes of therapy to control associated edema and release of inflammatory mediators. The need for systemic NSAID therapy can be reduced and associated edema and tissue damage minimized with effective use of topical therapy.

TOPICAL NSAIDS

A topical formulation of diclofenac liposomal cream (DLC; Surpass®, Boehringer Ingelheim Vetmedica, Inc., St. Joseph, MO) is now approved for treatment of horses with OA.[19] The drug comes as a cream that is applied locally to areas of inflammation. Studies in horses revealed that when the drug was incorporated into liposomes, it readily penetrates skin and significantly attenuated carrageenan-induced local production of prostaglandin E_2 (PGE).[8] Furthermore, minimal concentrations of diclofenac were detectable in plasma. In a study of 24 horses that had induced OA in the middle carpal joint, application of 7.3 g of DLC twice daily (label dose) to the skin had significant improvement in lameness, significantly less carpal bone sclerosis, and overall gross cartilage erosion compared with control.[19] A study reporting the serum and urinary concentrations at 1, 2, and 4 times the recommended dose applied over 10 days revealed slow absorption and elimination of the cream. This study is a useful resource to determine withdrawal times in order to prevent an inadvertent positive drug test.[3]

In the United States, diclofenac-based topical ointments are the only labeled products for topical use in people. In the European Union, ibuprofen creams and gels, ketoprofen gel, felbinac gel and cutaneous foam, and piroxicam gel are labeled for use. One review concluded that diclofenac and ketoprofen gel displayed the most meaningful analgesia in mild to moderate cases of hand and knee OA, had few systemic adverse effects, and had modest efficacy in chronic pain.[2] However, efficacy does appear to be limited in patients with more than one joint affected by OA.[2] Currently, diclofenac is the only NSAID labeled for topical use in horses and has modest data to substantiate its use in decreasing pain associated with synovitis in horses. No studies evaluating its use in chronic OA in horses have been documented.

Clinical Use

Surpass® is used commonly in clinical practice. It can be used under a bandage, and there are currently no reported adverse effects. Although the dose is 7.3 g BID, some clinicians use this drug once daily or every other day for less acute inflammation. It is used for OA, tendinitis, and various musculoskeletal inflammatory conditions. Concurrent use of this product with systemic NSAIDs is common and seems to be very effective for a variety of inflammatory conditions.

TOPICAL FIRST AID (COLD THERAPY AND BANDAGING)

With acute inflammation associated with tendon injury or joint trauma (capsulitis, synovitis), initial cold therapy using ice boots, cold water hosing/hydrotherapy, or medical devices that provide cold therapy and compression (Game Ready®, Alameda, CA) are useful therapies. Cold compression in the early stages of inflammation retards the inflammatory processes of exudation and diapedesis and reduces local circulation, tissue swelling, and pain.[23,33] The application of cold therapy as a primary treatment for most acute joint or tendon injuries is commonly practiced and is extremely beneficial. Following an initial cooling out phase, usually 48–72 hours, warm hydrotherapy is often used to relieve pain and tension in tissues and stimulate the vasodilatory effect to aid with fluid resorption and the stimulation of phagocytic cells.[33] These effects aid in increased local circulation, encourage muscle relaxation, and improve the metabolic rate of reparative cells and enzyme systems.[23]

Bandaging and pressure wraps are also commonly used to decrease edema formation and generalized swelling that results from inflammatory mediators released from the torn tendon or joint capsule. Further benefits may include stimulation of mechanoreceptors that aid in reduction of pain sensation.[33] Recent work has compared a standard cotton compression or light adhesive tape bandage at multiple sites and the effect of walking on pressure maintenance.[9] Cotton compression bandages applied to the limb below the carpus maintained their pressure, while those applied directly over the carpus and tarsus quickly lost pressure after walking a short distance. Light elastic bandages over the carpus, however, maintained their pressure after walking. Double-layer cotton compression bandages nor the addition of the "inner sanctum" to a standard cotton compression bandage at the distal limb did not improve bandage pressures any further. The authors concluded that modifications to the standard cotton compression bandage were of no additional benefit, while light elastic bandages over the carpus were resistant to pressure changes with ambulation and may be adequate for edema reduction.[9]

A survey of equine veterinary groups revealed that multiple modalities are used across both North America and Europe to combat injury and performance issues

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associated with musculoskeletal injury.[44] Controlled hand walking, therapeutic shoeing, ice, compression bandaging, platelet-rich plasma (PRP), therapeutic exercises, interleukin-1 receptor antagonist protein (IRAP), stretching, and cold water therapy were used by over 80% of respondents; to a lesser degree, heat, acupuncture, and massage were also used.[44] Use of these modalities is dictated by ease of use, whether specific personnel are required for their application (e.g. a licensed veterinarian) and time commitment. While evidence-based medicine for their benefit is lacking for many of these modalities, anecdotal beneficial effects have kept many of them within the armamentarium of both equine veterinarians and horse owners alike.

DIMETHYL SULFOXIDE

The chemical solvent dimethyl sulfoxide (DMSO) has been used alone or mixed with corticosteroids to treat soft tissue swelling and inflammation resulting from acute trauma.[33] Its main benefit is considered to be reduction of edema;[45] however, some work has suggested analgesia through blockade of C-fiber transmission and/or blocking sensory neurons.[16,41] Additionally, DMSO has been shown to possess superoxide dismutase activity and inactivate superoxide radicals. Further, the drug enhances penetration of percutaneous steroid when mixed with DMSO, and when cortisone was dissolved in DMSO, the dilution of cortisone necessary to stabilize lysosomes was reduced from one-tenth to one-thousandth.[45] If applied under a bandage, it should be used with caution due to its predilection in causing skin irritation.

The pharmacokinetics of DMSO in the horse has been established.[38] However, studies investigating the anti-inflammatory or analgesic effect in horses or companion animals are still lacking. Continued use in the horse without objective data of its efficacy is likely.

EXTRACORPOREAL SHOCKWAVE THERAPY

Extracorporeal shockwave therapy (ESWT) is used by veterinarians to treat many different musculoskeletal conditions in horses. Extracorporeal shock waves are acoustic waves generated outside the body, characterized by transient high peak pressures, followed by negative pressure, and then return to zero pressure. Pressures reached by current equipment range from 10 to 100 MPa with a rapid rise time of 30–120 ns and a short pulse duration (5 μs). Other variables include energy level, pulse frequency, and depth of penetration. Originally designed to treat bladder and ureteral stones (lithotripsy), it has also been used to treat horses with suspensory ligament desmitis, OA of the low-motion joints of the tarsus and dorsal metacarpal disease.[29,32]

The exact mechanism of action that ESWT has on specific tissues has yet to be determined. Through its ability to cause cavitation, or the rupture of gas bubbles within the tissues, it is postulated that it has effects on the tissues that are influenced by pressure, energy flow, and pulse frequency of the machine generating the waves.[32] Work in laboratory animals has demonstrated various dose-dependent effects, including microfracture of the cortical bone, medullary hemorrhage, subperiosteal hemorrhage, and stimulation of osteogenesis. Work in tendons has also demonstrated various dose-dependent effects from hematoma formation and tendon cell damage to increased glycosaminoglycan and protein syntheses.[7,31] Analgesic properties attributed to ESWT have also long been recognized, and concerns over its use at racetracks have stimulated debate due to its abilities to reduce or eliminate pain from an injury that may become catastrophic if the horse continues to race. Two studies have been performed in the equine that have detected decreases in nerve conduction properties.[6,32] One of those studies reported observations of significant disruption of the myelin sheath with no evidence of damage to Schwann cell bodies or axons following treatment with non-focused ESWT.[6] Further, bone effects have also been documented in the horse in which both focused and radial shockwaves resulted in an increase of microcracks on the dorsal surface of the third metacarpus and metatarsus.[11]

Several musculoskeletal conditions in horses have been treated with ESWT including the bucked shin complex, tibial stress fractures, proximal sesamoid bone fractures, incomplete proximal phalangeal fractures, subchondral bone pain, insertional desmopathies (most notably proximal ligament suspensory desmitis), impinging dorsal spinous processes, OA of the distal hock joints, navicular disease, and superficial digital flexor tendonitis.[31] Clinical trials have been inconclusive in the horse because the type of machine used varied as well as the energy levels, pulse frequency, depth of penetration, and number of treatments.

One of the most common clinical entities in which shockwave therapy has been utilized is proximal suspensory ligament desmitis. Data from clinical trials in both Germany and the United States revealed a 70%–80% success rate of return to work 6 months following focused or radial shockwave therapy.[31] Other work, however, reported return to work at 6 months to be more in the range of 55%–60%, and follow-up at 1 year showed much worse prognosis, especially for hind limbs.[28] Another popular use of shockwave therapy is in the treatment of navicular syndrome. In a clinical model of induced OA in the middle carpal joint of horses, no disease modification was seen with ESWT. Significant improvement in analgesia was observed in horses.[18] Most likely, this effect is due to the nerve sheath alterations observed in other studies.[6]

Use of ESWT will most likely continue in equine practice due to anecdotal evidence of its use in proximal suspensory ligament desmopathy and improvement in lameness scores in horses with induced OA. To this day, large clinical trials are still lacking. With the advent of a specialty college focused in rehabilitation medicine, controlled studies to validate the appropriate use and ideal settings, units, and specific diseases that will benefit from this treatment modality are much more likely to occur.

REGIONAL PERFUSION

Intravenous regional limb perfusions (IVRLPs) of the equine limb have become a standard of equine veterinary medicine over the last decade, with a multitude of

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publications within the veterinary literature investigating the ideal medication, concentration, and method of application for their use in a variety of disease processes. Nomenclature from early work in both human and veterinary medicine, however, made specific route of medication delivery difficult to ascertain. Those first to perform "regional limb perfusions" did so through direct antibiotic delivery within the medullary cavity in an effort to allow retrograde flow into the venous system to perfuse the limb (see below). In many instances, these were often referred to as intravenous therapy. However, today, most regional limb perfusions (RLPs) traditionally use a peripheral vein – and at times an artery – for application of intravascular therapy, while intramedullary application is seldom utilized.

Our understanding of IVRLPs has grown dramatically over the last several years. Current recommendations for antibiotic administration vary from tourniquet times of 10–30 minutes[24] and perfusate volume of 10–60 mL[22] and typically include an aminoglycoside – and more specifically amikacin – as the class of antibiotic administered due to their concentration-dependent and post-antibiotic effects.[10,21,22,24,30,35,40] Many other classes of antibiotics have been explored[12,26,36] but are likely poor candidates due to insufficient time above minimum inhibitory concentration (MIC) to cause beneficial effects.[35] RLPs have also been used in horses to provide distal limb contrast for MRI,[1] for mesenchymal stem cell therapy,[39,42] and for anti-inflammatory and antifungal treatment with DMSO and amphotericin B to combat pythiosis.[13,14] When multiple antibiotics have been used in combination or antibiotics with a local anesthetic, a reduction in antibacterial efficacy has not been measured.[10,12]

THERAPEUTIC ULTRASOUND, LASERS, AND ELECTROMAGNETICS

The use of therapeutic ultrasound, lasers, and electromagnetics is common in the horse industry. Horse owners that have done extensive investigation on the Internet where anecdotal reports abound will frequently request equine clinicians input on this therapy. Therapeutic ultrasounds emit unfocused sound waves that may have the potential to penetrate underlying tissues. The exact mechanism of action is unknown, but purportedly, thermal energy is produced as the sound energy contacts the tissues. *In vitro* studies have reported alterations in blood flow, angiogenesis, collagen and protein synthesis, and osteogenesis. Clinical trials have shown conflicting results in treating tendon injuries. Therapeutic ultrasound did not improve muscle regeneration in injured tissue nor alleviate chronic heel pain in people.[5,15] One trial in horses reported improved healing characteristics 4 and 6 weeks following tendon injury.[34] Until more clinical trials become available, use of this treatment will remain empirical.

Therapeutic laser therapy is based on light that is emitted from the instrument, which is a focused beam of photon energy. The power output of lasers used in physical therapy is in the milliwatt range (low). Mechanisms of action of therapeutic lasers are thought to include alterations in mitochondrial enzymes, cytochromes within the electron transport chain, and cellular calcium channels.[5] Cellular alterations may trigger mitosis and cellular proliferation, which may have biomodulatory effects on circulation and analgesia. Classification of lasers is defined by wattage output. Most used in clinical practice fall into the class IIIb criteria, which are those that emit power of 5–500 mW.[27] When used in a therapeutic setting, these lasers are commonly referred to as low-level laser therapy (LLLT) or cold lasers because their use does not generate noticeable heat to the patient. Class IV lasers are of greater wattage and have started to find their place on the human and veterinary market for treating injuries deeper within the body due to their higher output.[27]

A previous study examining its use of LLLT in the treatment of wounds in the horse showed no benefit.[37] The only benefit that has been shown in the horse is through the application of LLLT in the treatment of periodontal defects. Furthermore, when LLLT was used for treatment of surgically created wounds in dogs, no improvement in subjective healing nor wound size was observed compared with control groups.[20]

Magnetic and electromagnetic therapy remains a controversial subject. A large amount of anecdotal information is available, especially by equipment manufacturers. Ironically, this information is highly available to lay horseman, despite a lack of technical information, understanding, and directions for use. In general, an electromagnetic field is created by passing an electric current through a coil of insulated wire. When piezoelectrical properties of bone where discovered, evidence became available, which supported the use of electromagnetic fields on bone healing.[5] Limited studies are available in horses. Some benefit in fracture healing was observed after 2 hours of daily therapy.[4] Conversely, pulsed electromagnetic field therapy delayed tendon healing in experimentally created defects in the superficial digital flexor tendon (SDFT) in horses.[43] In a rabbit study, however, beneficial effects were demonstrated in tendon healing.[17] Since the last edition of this book, more recent work in human medicine has shown an added benefit when electromagnetic therapy was used in combination with shockwave therapy in rotator cuff tendinopathy.[25] However, no further elucidation on mechanism of action has been published, nor any further evidence-based studies performed in equine medicine. As with therapeutic ultrasound and therapeutic laser therapy, magnetic therapy applications will most likely continue to be touted by the lay horseman until clinical trials are available to prove or disprove efficacy.

COUNTERIRRITATION

Topical blistering and pin firing have long been part of veterinary medicine, but fortunately, they are rarely practiced today due to the lack of clinical data as well as their inhumane ramifications. They are mentioned in this section only to acknowledge their common usage in the past. Topical blisters historically were made of iodine or mercuric iodide and were mainly used on splints, sore shins, or curbs. More severe blistering agents were made from cantharides or croton oil. The technique involved application to the skin and light exercise.

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Firing most typically involves the use of a pinpoint hot iron applied to the skin over the affected structure. Most commonly, firing is used for bucked shins and plantar ligament desmitis (curb) when the condition has not responded favorably to other forms of treatment. The expectations that counterirritation may result in an improved fiber pattern with crimp and collagen within tendons are curious at best. Beyond enforced rest and low-grade exercise, the benefits of the treatment on underlying structures are most likely minimal or nonexistent. Occasionally, the technique continues to be used, but with the retirement of veterinarians who commonly utilized this method, it is seldom practiced. At this juncture in veterinary medicine, when a greater push for evidence-based medicine is becoming standard, counterirritation has little evidence to support its use, is antiquated, and can no longer be recommended.

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INTRASYNOVIAL

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Intrasynovial therapies are used to diminish the inflammatory response in synovium, cartilage, tendon (or tendon sheath), or meniscus. An effective therapy should halt progression of degradation of these structures and restore the normal intrasynovial environment, which should alleviate pain. Equine practitioners currently have several options available to treat intrasynovial inflammation. Intrasynovial therapies, specifically corticosteroids, are used frequently in horses to minimize or control pain associated with synovitis and osteoarthritis (OA).[20] New therapies are also beginning to be used intrasynovially as potential disease-modifying agents of osteoarthritis (DMOAD); their reputed properties are prevention, retardation, or reversal of morphologic cartilaginous lesions of OA. While claims of these properties exist, evidence-based clinical studies about their true abilities to reverse OA are still in their infancy. The intrasynovial therapies covered in this section are the most commonly used treatments currently available for equine practitioners.

CORTICOSTEROIDS

Corticosteroids (CS) are the most potent anti-inflammatory drugs available that decrease the catabolic effects of joint disease. Historically, their use has been highly controversial due to the negative effects associated with overuse and high dosages utilized in the past.[78] Current clinical studies suggest judicious use of CS may be extremely beneficial and can result in long-lasting pain relief and control of inflammation.[36,66]

Mechanism of Action

CS have powerful inhibitory effects on inflammation through stabilization of cellular lysosomal membranes, reduction of vascular permeability and leukocyte adherence to vessel walls (margination), inhibition of platelet aggregation, and leukocyte diapedesis. Furthermore, they inhibit prostanoids and nitric oxide, mediators integral to the progression of osteoarthritic lesions.[9,56] CS are considered to be disease modifying through their actions on the abrogation of important mediators of inflammation, interleukin-1 (IL-1), and tumor necrosis factor-α (TNF-α). Degradative enzymes such as matrix metalloproteinases (MMPs) and other related proteinases are also decreased through the actions of CSs which alters the progression of an inflamed joint toward osteoarthritic conditions.[27,60]

Detrimental Effects and Concerns Regarding Intra-Articular Corticosteroids

The literature is replete with reports describing the deleterious effects of CS on cartilage. They include decreased chondrocyte size, loss of glycosaminoglycans (GAGs) and decreased GAG synthesis, inhibition of proteoglycan synthesis, chondrocyte necrosis, and hypocellularity.[14,25,61,103] Particularly at high concentrations, CS inhibit production of important components of cartilage such as proteoglycans, collagen, and hyaluronic acid, and furthermore result in chondrocyte necrosis.[25,111] It is believed the high concentration and extended exposure used in earlier studies resulted in the detrimental effects of CS on cartilage. As more realistic clinical models have emerged, such detrimental effects are less noted, and more cartilage-sparing effects have been observed with use of low-dose CS.[66,89] The emerging stand by many clinicians seems to be that intra-articular (IA) steroids can be extremely beneficial when used judiciously. This has been supported in the human literature as well, which has shown that CS have a time- and dose-dependent effect on articular cartilage, with beneficial effects occurring at low doses (<2–3 mg/dose or 8–12 mg/cumulative total dose) and durations and detrimental effects at high doses (>3 mg/dose or 18–24 mg/cumulative total dose) and durations.[117]

Postinjection flare associated with CS use in the horse is rare and usually self-limiting but associated with some discomfort, necessitating nonsteroidal anti-inflammatory drug (NSAID) therapy. The inflammation is believed to be caused by the microcrystalline characteristics of CS preparations, as well as the dose and particular ester that is injected; however, the incidence of postinjection flare has decreased with the advent of branched-chain esters.[113] More concerning is the potential for infection following CS injections, and a reaction to medication should be treated as a septic process until proven otherwise. While the incidence is low, 1 case per 1,279 joints injected in one study,[106] the occurrence of infection can be devastating and expensive to treat; therefore, strict adherence to aseptic technique is mandatory, and the combination with an antimicrobial is commonly practiced.

It is important for practitioners to be aware that CS injections associated with sepsis may take up to 10 days to become apparent due to their powerful anti-inflammatory properties. When faced with determining between a CS flare and joint sepsis, a synovial aspirate should be performed to determine a leukocyte count and differential consistent with bacterial infection. Clinicians should bear in mind that CS can falsely lower white blood cell count post-CS injection.

Laminitis has been associated with CS administration, and although rare, its occurrence subsequent to IA steroids can be devastating. In a study in which 205 horses were injected with triamcinolone at a dosage between 10 and 80 mg/treatment, only 2 of 205 horses developed laminitis, and 1 of those cases was considered unrelated to the treatment because the laminitic event occurred several months following the treatment. This study reveals the low incidence of this complication,[80]

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with earlier studies and anecdotal reports suggesting a total cumulative dose of less than 18–20 mg triamcinolone acetonide (TA) to be safe.[13,50]

Finally, there has been concern regarding the use of CS in Thoroughbred racehorses due to a positive correlation between local corticosteroid injections (LCIs) and subsequent musculoskeletal injury rates, most likely due to progression of the condition that prompted treatment.[118] A retrospective cohort study comparing the rate of musculoskeletal injury among Thoroughbred racehorses after receiving LCI to those not being treated with LCI reported a 3.3–4.6 times greater incidence rate of musculoskeletal injury in treated versus untreated horses; however, due to study design, a direct assessment of the independent effect of LCI could not be performed.[118] The risk of musculoskeletal injury in that study also appeared to rise with subsequent treatments, with the carpus and fetlock joints being at the highest risk of injury post-medication.[105,118]

Another study investigating the association between intrasynovial medication and fracture risk in racehorses reported serious musculoskeletal injuries in 3% (44/1,488) of horses following intrasynovial medication, with ≥3 previous intrasynovial medication sessions resulting in an increased hazard of sustaining a fracture within 56 days of treatment.[105]

While additional research regarding the relationship between intrasynovial use of CS and the risk of subsequent musculoskeletal injury is still warranted, the greater hazard of injury identified in the current literature is more likely dependent upon identifying horses at risk for musculoskeletal injury rather than a direct adverse effect of the CS.[118] Therefore, a thorough examination, including diagnostic imaging, should be performed to determine whether to treat a problem with LCI and continue to train and race the horse.

Clinical Choices

Despite decades of CS use to treat IA inflammation in the horse, the choice of formulation, dose, and frequency of administration remains somewhat empiric. Currently, the most commonly used formulations are TA, methylprednisolone acetate (MPA), and betamethasone acetate (BA).[30] Less commonly used CS include isoflupredone acetate and flumethasone. In a survey of more than 400 equine practitioners, TA was found to be more commonly used in high-motion joints and MPA in low-motion joints.[30]

Clinicians currently use lower dosages of CS than in the past because they seem to result in similar inhibition of inflammation with fewer detrimental effects on cartilage metabolism. Doses vary depending on the volume of synovial fluid in the joint, severity of inflammation, and the number of other joints requiring treatment. The formulations in Table 8.1 represent currently used CS and the associated dosages most commonly administered.

Triamcinolone Acetonide

Both *in vitro* and *in vivo* studies have demonstrated that low doses of TA can inhibit degradative metalloproteinases with minimal negative effects on extracellular matrix of cartilage.[42,97] Studies suggest that a dose of 6–12 mg/joint should be adequate for anti-inflammatory effects, with one study demonstrating that 12 mg of TA administered twice, 2 weeks apart, minimized the development of OA secondary to osteochondral chip fractures.[42] These effects were also noted in the contralateral joint, and subchondral bone remodeling or fragility was not detected.[66] Another study provided further evidence for the anti-inflammatory effects of IA TA (9 mg dose) with significant changes in inflammatory gene expression; however, downregulation of collagen gene expression was also observed and warrants the careful use of TA in horses.[70]

In contrast, 12 mg of TA administered at 3, 5, and 7 weeks into normal joints significantly altered cartilage metabolism both in injected and remote joints.[22] Although the joints in this study were not inflamed, it emphasizes that TA may change regulatory processes important in cartilage metabolism both in the injected joint and remote joints. The remote effects of TA have not been observed with other CS.

Additionally, an intrasynovial structure may be injected with a CS and anesthetic as a combination of treatment and diagnostic aid. Recently, a study found that the combination of TA and mepivacaine does not alter the potency or duration of TA's action.[67]

Methylprednisolone Acetate

MPA has been studied extensively both *in vitro* and *in vivo*. Various beneficial and detrimental effects have been noted in cartilage, subchondral bone, and synovium.[21,29,97] Beneficial effects of MPA include reduction in the transcription of harmful molecules such as

Table 8.1. Commonly used intra-articular corticosteroids.

Corticosteroid	Trade name	Manufacturer	Drug conc. (mg/mL)	Dose (mg)	Duration of action
Triamcinolone acetonide	Kenalog-10 Vetalog	Bristol-Myers Squibb Boehringer Ingelheim	10 6	6–18	Medium
Methylprednisolone acetate	Depo-Medrol	Zoetis US	40 or 20	40–100	Long
Betamethasone acetate/ sodium phosphate	Celestone Soluspan BetaVet	Merck Inc. Luitpold Pharmaceuticals	6	3–18	Medium to long

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IL-1β, TNFα, MMPs, and others that directly cause matrix degeneration. Harmful effects include chondrocyte necrosis, inhibition of proteoglycans, and procollagen synthesis.[77,112] The dose of MPA seems to predict the fine balance between inhibition of inflammation with overall beneficial effects of the joint or destruction of matrix and disruption of normal cartilage metabolism. Studies regarding what level of MPA inhibits the destructive cascade of inflammation while preserving the normal joint environment are still ongoing.

Studies suggest that more physiological doses of between 10 and 40 mg/joint inhibit inflammation and preserve normal joint environment.[29,84,111] Furthermore, it has been suggested that the propensity for matrix changes tends to occur immediately following injection (softening), with inhibition of biosynthesis and evidence of matrix damage seen after intense exercise (running) within 6 days' post-MPA; therefore, it is recommended that horses be prohibited from applying excessive loads (exercising at more than a trot) for 6 days after injection with MPA.[29]

In contrast, a study performed in equine middle carpal joints found that microhardness of subchondral bone did not change with repeated injections of MPA and treadmill exercise; however, this study was done in normal joints.[85] One study in horses that had osteochondral fragments induced in the middle carpal joints, had MPA injected at 100 mg, and underwent exercise on a treadmill not only revealed lower prostaglandin E_2 (PGE_2) levels but also exhibited articular cartilage erosion and morphologic lesions associated with IA MPA.[43] In a survey of more than 400 practitioners regarding the use of CS, 75% revealed that they use MPA in low-motion joints, 36% use a dose of 20–40 mg, and 44% used 40–80 mg/joint injection.[30]

Betamethasone

Similar to MPA, betamethasone (BA) is considered an intermediate-to-long acting glucocorticoid, with intrasynovial levels falling below the level of detection between 14 and 21 days after a single IA injection (9 mg).[69] One clinical study that utilized the osteochondral chip model in the middle carpal joint with 15.9 mg of BA administered to the joint at 2-week intervals followed by treadmill exercise revealed no statistically significant differences between treated and control joints at 56 days, but there were trends toward decreased GAG staining in treated joints.[36] Despite these findings, *in vitro* work still indicates potential detrimental effects as measured by suppressed proteoglycan synthesis at medium to low physiological doses.[41] Collectively, the scientific literature on BA demonstrates time- and dose-dependent deleterious effects on articular cartilage and chondrocytes, with chondrotoxicity, loss of cartilage proteins, and progressive gross damage observed at higher doses.[117]

HYALURONAN

HA, or hyaluronan, is a large, unbranched, non-sulfated GAG composed of repeating units of D-glucuronic acid and *N*-acetyl glucosamine and is secreted by the type B synoviocytes of the synovial membrane.[63,95] HA serves various important functions in the joint, such as viscoelasticity to the joint fluid and boundary lubrication of the IA soft tissue. HA may also influence the composition of synovial fluid through steric hindrance of active plasma components and leukocytes from the joint cavity.[63] Furthermore, HA appears to modulate the chemotactic response within the synovial membrane by reducing cell migration and decreasing rates of diffusion and flow of solutes.[62]

HA is locally synthesized by chondrocytes and is the backbone of the proteoglycan aggregate in the extracellular matrix. The compressive stiffness in articular cartilage depends on the integrity of the matrix proteoglycans.[52]

Mechanism of Action

Although the exact mechanism of action is unclear, studies suggest that exogenously administered HA supplements replace the actions of depleted or depolymerized endogenous HA in the synovial fluid, which restores viscoelasticity, steric hindrance, and lubrication of the articular soft tissues. *In vitro* studies suggest additional effects, including the ability to inhibit macrophage chemotaxis, reduce lymphocytic ability to proliferate and migrate, reduce the formation and release of prostaglandins from macrophages during phagocytosis, and scavenge oxygen-derived free radicals and degradative enzymes.[10,37,52,92] Equine synovial fluid exhibits poor lubrication properties within the acute postinjury phase, with diminished concentration and molecular weight of HA suggested as the cause.[5] HA also possesses direct analgesic properties that seem to be partially attributable to reductions in the sensitivity of articular nerve endings.[20,54] Furthermore, it is suspected that exogenous injection of HA into osteoarthritic joints increases synthesis of high molecular weight HA by the synoviocytes.[63]

Many clinical studies in horses have supported HA administration in joint disease.[6,7,46,76,98,99] While the criteria for treatment success vary, the overall clinical impression is favorable. Inherent to clinical trials, duration of posttreatment observation periods is varied and short in some studies. Nevertheless, parallel clinical trials in people also indicate symptomatic efficacy measured by improvement in pain, activity level, and function, especially following use of high molecular weight products.[2]

Molecular Weight and Efficacy

The controversy continues regarding efficacy and molecular weight of high versus low molecular weight HA. Proponents of the viscosupplementation theory believe higher molecular weight products are more effective,[90] while others minimize this importance of size and suggest the activity of HA is mediated pharmacologically rather than physically.[8] A study evaluating the effects of joint injury on the boundary lubrication properties of synovial fluid found that relative to normal equine synovial fluid, there was diminished concentration and molecular weight of HA in equine synovial fluid from joints with acute injury.[5] This same study also showed restoration of the boundary lubrication function of the synovial fluid following *in vitro* supplementation with

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Table 8.2. Commonly used hyaluronan preparations.

Trade name	Concentration (mg/mL)	Manufacturer	Molecular weight (Daltons)[a]	How packaged	Current recommended dose (mg) per joint
Hyvisc	11	Boehringer Ingelheim	2.1×10^6	2-mL syringe	22
HY-50	17	Dechra	7.5×10^5	3-mL syringe	51
Hyalovet	10	Boehringer Ingelheim	$5–7.3 \times 10^5$	2-mL syringe or vial	20
Legend[b]	10	Merial Inc.	3×10^5	2-mL vial	20
HyCoat (used IA)	5	Neogen	$>1.0 \times 10^6$	10-mL vial	30
MAP-5 (used IA at this dose)	10.3 (2 mL) 5 (10 mL)	Bioniche	7.5×10^5	2- or 10-mL vial	20

[a] Manufacturer's reported molecular weight.
[b] Marketed for IV and IA use.

high molecular weight HA. There was no significant difference in HA molecular weight between synovial fluid from normal joints and those with chronic injury. Table 8.2 lists commonly used HA products, their molecular weights, and current recommended dosages.

HA can be expensive for owners, and therefore, use in joints with advanced disease may yield less satisfying results compared with joints with incipient disease. This is consistent with human medicine, where people with less severe joint disease demonstrate greater improvement to IA HA compared with those with more advanced radiographic changes.[114]

CORTICOSTEROID AND HA COMBINATIONS

The combination of HA and steroids to treat inflammation intrasynovially is commonplace.[15,26,74] Synergistic effects have been reported in human patients, and similar effects have been seen *in vitro* in horses.[75,119] Many clinicians believe that the combination allows a smaller dose of CS to be administered with the HA, although further clinical studies are needed to determine efficacy of combinations. The combination of HA and CS is also thought to provide a longer clinical effect than either injection alone, especially in high-motion joints.

A recent open-label multicenter clinical trial involving 80 horses with clinical joint disease found that the IA combination of 12-mg TA and 20-mg HA was associated with a lower short-term success rate (64.1%) compared with 12-mg TA alone (87.8%).[55] In this study, short-term success was defined as greater than or equal to a 2-grade reduction in lameness (0–5 scale) 3 weeks after injection. Furthermore, this reduction in short-term success rate for the combination treatment group was associated with advanced age (≥13 years). This same study revealed no significant difference in medium-term outcome (3 months' posttreatment), with approximately 50% of horses returning to their previous level of exercise regardless of treatment group. The reason for the difference in the observed outcome is unknown.[55] One speculation includes the presence of unknown drug interactions; however, further evaluation of the combination of HA and TA is warranted. At this time there is minimal clinical evidence to support combining HA and TA in horses.

IA POLYSULFATED GLYCOSAMINOGLYCANS

Polysulfated glycosaminoglycans (PSGAGs) principally comprise chondroitin sulfate, a GAG found in the aggregating proteoglycan of cartilage. It is a semisynthetic preparation from bovine trachea and is reported to have chondroprotective and anti-inflammatory properties as well as the ability to induce articular cartilage matrix synthesis and minimize matrix degradation.[110]

Mechanism of Action

The exact mechanism of action remains unclear. The drug most commonly is administered intramuscularly as IA administration was initially associated with joint infections due to a reduction in complement activity in the joint. IA administration is now most commonly combined with 125–250 mg of amikacin to deter any infection, and this practice appears to be effective.

Many *in vitro* studies mimic the IA administration of PSGAGs because it is unlikely that concentrations injected intramuscularly can translate to similarly elevated concentrations in the joint fluid. *In vitro* studies and *in vivo* IA administration have revealed a significant ability of the drug to decrease lameness, modify OA through reducing bone remodeling, promote synthesis of endogenous HA, and inhibit mediators of inflammation, specifically PGE_2 production. Additionally, it has been found to reduce the levels of MMPs that rise in response to production of IL-1β.[20,49,52] Most recently, IA PSGAGs were compared with HA in an osteochondral chip fragment model. While HA significantly reduced fibrillation of the cartilage, PSGAGs were demonstrated to reduce joint effusion and reduce synovial membrane

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vascularity and subintimal fibrosis compared with control horses when the drug was administered weekly for 3 weeks.[46] However, neither PSGAGs nor HA had a beneficial effect on lameness. It was concluded that in the osteochondral fragment model, both drugs had beneficial disease-modifying effects. Furthermore, in a study that compared PSGAGs with IA CS (MPA 40 mg), a regimen of weekly IA PSGAGs for three treatments gave significantly improved results (67%) compared with one treatment of CS (46%) in terms of returning horses to performance.[73] It has also been suggested that, using the osteochondral fragment model, the IA combination of TA (5 mg) and PSGAG (250 mg) is less effective than IA PSGAG used alone when administered at weekly intervals for five treatments.[48]

Clinical Considerations

PSGAG is most likely more efficacious if administered IA; however, due to the drug's ability to potentiate sub-infective doses of contaminant bacteria, this route of administration should be accompanied with amikacin in order to obviate this risk.[57,58] Currently, the approved frequency of IA administration is three to five injections (250 mg each) at weekly intervals.[1,93] It may be most efficacious in joints with known or suspected articular cartilage pathology.[1]

POLYGLYCAN®

Polyglycan®, which is not yet licensed for use as an IA medication, is a patented formulation composed of HA, chondroitin sulfate, and *N*-acetyl-D-glucosamine and has been used clinically in horses IA for viscosupplementation. This product is currently used extensively in equine practice; however, only one randomized blinded placebo-controlled study has been performed to assess its efficacy. Using the OA osteochondral fragment model of the middle carpal joints, horses that received Polyglycan® IA (5 mL) at days 0, 7, 14, and 28 showed significant improvements in lameness, bony proliferation, and degree of full thickness articular cartilage erosions observed grossly.[47] Treatment with Polyglycan® resulted in a mild transient reduction in lameness throughout the study, with the greatest improvement (30% reduction) noted on day 42. When compared with the use of HA alone in one *in vitro* study, cells treated with HA had significantly better morphology scores; however, Polyglycan® resulted in superior suppression of lipopolysaccharides (LPS)-induced production of proinflammatory cytokines, providing further support for its protective potential.[68]

AUTOLOGOUS CONDITIONED SERUM

Autologous conditioned serum (ACS, also called interleukin receptor antagonist protein, IRAP) has been used extensively in practice to treat intrasynovial inflammation, especially in cases that are refractory to IA CS. Although clinical impressions have been favorable, this method of treatment is not widely used due to cost. IRAP, a substance that inhibits IL-1 activity, decreases the progression of joint disease and is believed to be present in high amounts in ACS.[45] Specifically, it has been reported that production of IL-4, IL-10, IRAP, and other growth factors is stimulated through incubation of whole blood with medical-grade glass beads exposed to chromium sulfate.[82]

A study in which ACS was injected into the middle carpal joints on day 14, 21, 28, and 35 following induction of OA through a chip fragment model revealed significant clinical improvement in lameness and reduced gross cartilage fibrillation and synovial membrane pathology.[45] This same study detected elevated levels of IRAP in the synovial fluid of the horses injected with ACS at 70 days' post-OA induction.[45] Clinical trials in humans with OA have indicated that people injected with ACS experienced greater reduction in clinical signs and improved quality of life compared with people whose joints were injected with HA.[11,116] Significant pain reduction has also been seen following treatment of patients with varying degrees of hip OA with ACS alone, with no increase in beneficial effects seen when ACS was combined with cortisone or cortisone + recombinant IRAP (rIRAP).[12] Use of this medication requires incubation of equine serum with beads coated with chromium sulfate to obtain the ACS solution. The ACS is usually administered IA weekly for three to four treatments, depending on the quantity of ACS obtained. Treatment with ACS prior to injection of other biologic agents is increasing in practice, although further clinical trials are necessary to evaluate the benefits and effectiveness of this technique.

PLATELET-RICH PLASMA

Platelet-rich plasma (PRP) is an increasingly popular intralesional therapy used in equine practice to enhance the regeneration of injured tissues with poor healing capabilities (i.e. limited blood supply). However, IA use of PRP has recently emerged as a potential treatment of OA both in humans and horses.[19,33,34,39,71,83,94,101] PRP is a platelet-concentrated, autologous blood product created through various centrifugation processes that delivers increased platelet-derived growth factors (PDGF) to the site of injury.[40,107] Various cytokines detected in PRP that can potentially promote healing include transforming growth factor-β (TGF-β), PDGF, insulin-like growth factor (IGF-I, IGF-II), fibroblast growth factor (FGF), epidermal growth factor, vascular endothelial growth factor (VEGF), and endothelial cell growth factor.[40] *In vitro*, PRP has been shown to induce chondrogenesis by stimulating proliferation of chondrocytes and extracellular matrix synthesis of proteoglycans and type II collagen,[3] to induce chondroprotection by increasing the production and secretion of HA from synoviocytes,[4] and to inhibit inflammatory processes in chondrocytes.[19]

Due to the limited number of *in vivo* studies, IA PRP is not yet a standard treatment for OA but has shown potential as a therapeutic option in refractory cases. In both horses and humans, IA PRP appears to incite an acute, short-term inflammatory response within the injected joint;[83,94,104,107] however, studies evaluating its use in healthy equine joints have found IA PRP to be safe without long-term detrimental effects.[83,101,107] One clinical study evaluated PRP for the treatment of refractory

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OA in endurance horses and reported that 80% of horses were able to return to regular work following treatment.[91] While largely positive, variable results have also been reported in the human literature.[33–35,40,71,94,96]

AUTOLOGOUS PROTEIN SOLUTION

Autologous protein solution (APS, Pro-Stride®) is a newly developed, modified blood product generated from a dual-device system that concentrates plasma and WBC proteins and enriches platelet growth factors. APS is meant to achieve the goals of both ACS (IRAP) and PRP through a two-step centrifugation process.[17] This process separates a small volume of plasma concentrated with platelets and WBCs, which is then filtered through polyacrylamide beads to desiccate and further concentrate growth factors and plasma proteins.[17] Additionally, APS can be drawn, processed, and injected stall side without the need for incubation or processing.

A prospective, randomized, placebo-controlled clinical trial that evaluated the efficacy of a single IA injection of APS in horses with OA in various high-motion joints found that treated horses had significant improvements in lameness grade, asymmetry indices of vertical peak force, and range of joint motion by 14 days' post-treatment.[17] In this group of horses, APS had a greater likelihood of a therapeutic response in horses with a lameness score <4, <10% vertical force asymmetry, or absence of marked osteophyte formation, subchondral sclerosis, or joint space narrowing.

Current research on APS in horses has been limited; however, the benefits described in the previous study have been validated in both canines and humans. In a study of 21 dogs with OA of the stifle or elbow, a single injection of APS resulted in reduced pain and lameness scores and increased weight-bearing associated with the OA-affected joint at 12 weeks' postinjection.[115] A study involving 41 human patients that received a single injection of APS for knee OA showed significant clinical improvement in pain scores as well as reduction in bone marrow lesion size on MRI and osteophyte formation 6–12 months after injection.[72]

BONE MARROW- OR FAT-DERIVED MESENCHYMAL STEM CELL THERAPY

The use of bone marrow-derived mesenchymal stem cells (BMSCs) or fat-derived mesenchymal stem cells (MSCs) has grown in popularity in equine practice in the last decade. It is believed that these cells possess anti-inflammatory properties and may also contribute to healing of musculoskeletal tissues by becoming incorporated into the repair tissue.[87] A recent retrospective study of 40 horses that received IA injection of BMSCs that had been expanded to approximately 10–20 million cells revealed a return to athleticism in 72% of all horses with IA injuries, ranging from mild to severe.[31] IA administration of BMSCs postoperatively for stifle lesions has also been reported in horses, with improved ability to return to work (75%).[32] Although more is known regarding the efficacy of MSCs in tendon injuries compared with synovial disease, early results suggest that these cells may also have a place in intrasynovial therapies.

The concomitant use of CS and MSCs has been shown to have a detrimental effect on cell viability, with both MPA and TA resulting in the rapid death of significant numbers of MSCs, and should not be performed.[28] Similar effects have been shown when combining cultured BMSCs with therapeutic concentrations of aminoglycoside antimicrobials (150-mg gentamicin or 250-mg amikacin), resulting in >95% cell death within 2 hours of incubation.[18] Based on these findings, the co-injection of BMSCs and aminoglycoside antimicrobials is not recommended. The same study reported minimal effects of HA (22-mg total dose) on the viability and proliferation of cultured BMSCs, with a trend noted for mild stimulation and enhanced secretion of immunomodulatory mediators.[18]

Concentrated bone marrow aspirate concentrate (BMAC) is another autologous source of MSCs being investigated for the treatment of OA and regeneration of cartilage in osteochondral defects. While the overall concentration of MSCs in BMAC is limited, BMAC also serves as a source of growth factors important for the induction of chondrogenesis.[64,100,102] BMAC has been shown to improve cartilage healing in horses following microfracture repair of cartilage defects. Horse treated with BMAC had improved structure, matrix composition, and integration of the cartilage repair tissue into the surrounding normal cartilage.[38] Overall, similar positive results have also been published in the human literature for treating OA in the knee.[23] Although further work is needed, current evidence suggests that BMAC has the potential to be an effective, minimally invasive option to help repair cartilage defects and treat OA in the horse.

POLYACRYLAMIDE HYDROGEL

Polyacrylamide hydrogel (PAAG) is a nontoxic, nonimmunogenic, nondegradable, and biocompatible polymer gel consisting of 2.5% cross-linked polyacrylamide with a long-lasting viscous effect shown to support cellular growth and augment tissues.[109] This product has historically been used as a bulking agent to enhance soft tissue integrity in human medicine.[16,86,88] Just recently approved for use by veterinarians in the United States, PAAG has been a newly investigated therapeutic option for treating OA in horses.

An international multicenter study that evaluated the efficacy of IA PAAG in horses with OA showed that PAAG significantly alleviated lameness and joint effusion, with 59% of horses sound 1 month after treatment.[109] Improvement in the number of non-lame horses and reduction in joint effusion were observed over time, with 82.5% of horses sound after 24 months. Furthermore, the effect of treatment increased over time with no significant side effects observed in treated joints. A comparative prospective study by the same author also demonstrated that horses with OA treated with PAAG had significantly improved clinical signs when compared with horses treated with TA combined with sodium HA.[108] A preliminary field trial that evaluated the efficacy of a 4% PAAG in osteoarthritic horses

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showed decreased lameness with no adverse effects within the first 3 weeks of treatment, and this effect was sustained until the 90-day follow-up evaluation.[79] Limited information can be taken from this study as follow-up was not continued beyond 90 days and there was no control group for comparison.

Incorporation of PAAG into the synovium after injection into normal and osteoarthritic joints has also been demonstrated in horses, with evidence of integration, proliferation, and invasion of synovial cells into the gel by day 14.[24] This was followed by the formation of a vascular, sub-synovial connective tissue layer by day 30 that was covered by a synovial lining. This histological appearance persisted up to 2 years postinjection in equine joints. Further clinical studies to explore potential effects on synovial inflammation and pain are warranted.

MISCELLANEOUS

Other modes of therapy that control intrasynovial inflammation are currently being investigated. Gene therapy modalities engineered to overproduce beneficial cytokines such as IGF-I and IRAP are still being developed.[44,53] Clinical trials will clarify the utility of these therapies.

Atropine is an anticholinergic drug that is occasionally used intrasynovially to reduce joint effusion. Its efficacy in a mouse model was proven by reducing induced joint effusion.[81] It has been used with some success in the horse; however, its use for reduction of joint effusion remains empirical. It is primarily used to reduce effusion in synovial cavities (mostly joints and tendon sheaths) that have not responded to other forms of therapy. The dose used is 7-mg IA, and if used for treatment of hygroma, it should be combined with bandaging.[65] Horses should also be observed for colic following IA injection.

INTRASYNOVIAL THERAPY: PRACTICAL CONSIDERATIONS

Intrasynovial therapies are an integral part of the treatment of lameness in horses. Of course, success of the therapy depends on the correct synovial structure being medicated, and that decision should be based on an accurate lameness examination and appropriate imaging. Furthermore, success of diminishing lameness also depends on the severity of the disease in the synovial structure being medicated and the efficacy of the medication being injected. For example, a recent study found a direct correlation between the success of intrabursal injections and the severity of the disease seen on MRI, with less severe disease having greater long-term success in treatment.[15] There are no accurate reports of how long a horse should be rested following CS injections; however, common practice is to combine the intrasynovial injection with at least 4–5 days of rest because the mechanical integrity of the cartilage has been shown to be compromised for several days following injection.[29] It is recommended that horses be returned to exercise slowly, over 1–2 weeks.

Finally, as previously mentioned, sepsis may be a complication with any intrasynovial medication, and it is paramount for the clinician to be aware of any worsening of lameness following medication of a synovial structure. A survey of 241 equine veterinarians revealed an incidence of 2.1 septic joints per 10,000 IA injections, with significantly lower infection rates when veterinarians prepared their own injection sites and had <20 years of practice experience.[51] Strict adherence to aseptic technique should also be practiced. Clipping the hair is not mandatory,[59] and a survey of veterinarians actually suggested that synovial infection rates were significantly higher for veterinarians that routinely removed the hair at the injection site.[51] Nevertheless, clipping the area of injection is still recommended by the author if winter hair growth precludes the ability to adequately prepare the skin site – due to the devastating consequences of synovial infection.

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INTRALESIONAL

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INTRODUCTION

Therapies that are injected intralesionally are usually directed at healing tendon or ligament. They are most often intended to augment the healing processes locally by providing the necessary components of healing to the tissue being treated. Alternatively, they may act locally to either reduce inflammation and/or signal the cellular and molecular components of the injured and surrounding tissue to begin the reparative processes. During repair of these tissues, injured elastic fibers are replaced with modified fibrous scar tissue, resulting in repair that is suboptimal. The quality of repair varies greatly depending on the severity of lesion, the inherent healing properties of the individual, the rehabilitation program, and the local environment of the lesion. Some injuries may repair and resolve with enough mature collagen so that they return to normal size, with sufficient remodeling that results in parallel alignment of the fibers within the tissue. Other injuries form a scar, with a resulting increase in size of the tendon/ligament, poor or random fibrous tissue alignment, and peritendinous/ligament fibrosis.

Intralesional approaches are directed at maximizing the chances for a more physiologically functioning tendon. Along with intralesional therapies, rehabilitation and constant ultrasonographic monitoring must accompany any treatment. Treatments usually reduce the acute inflammatory response and hemorrhage in the acute phase and improve fiber alignment during the reparative phase. Ultimately, the goal is to maximize the chances for a tendon or ligament to repair with adequate strength and elasticity for a return to a similar level of performance with minimal risk for reinjury.

HYALURONAN AND POLYSULFATED GLYCOSAMINOGLYCANS

Hyaluronan (HA) has been investigated for tendon injuries and adhesion prevention throughout the last three decades. Its use is predicated on its abilities to decrease inflammation and to prevent adhesion formation through its mechanical characteristics.[27] Further, it was believed to stabilize the fibrin clot early in wound healing, enhance the migration and replication of fibroblasts in the wound,[15] and improve fiber and biochemical characteristics of tendons.[46] The use of polysulfated glycosaminoglycans (PSGAG) intralesionally is based on the known abilities of this drug to inhibit many of the enzymes associated with connective tissue degradation. *In vitro* studies have shown that PSGAG are capable of inhibiting lysosomal elastase, cathepsins G and B, lysosomal hydrolases, keratin sulfate glycoanhydrolase, serine proteases, neutral metalloproteinases, beta-glucuronidase, alpha-glucosidase, beta-*N*-acetylglucosaminidase, and hyaluronidase.[2,6,31]

Recent clinical studies have reported no difference in outcome or reinjury rate in performance horses (race, national hunt, dressage, and jumper horses) when horses were injected with intralesional HA, PSGAG, or conservative management.[12,28,29] While intrasynovial and systemic administration of both HA and PSGAGs is still used clinically, the intralesional administration previously investigated is no longer the treatment of choice for intralesional tendon injury.

AUTOLOGOUS CELL THERAPIES: MESENCHYMAL STEM CELL THERAPY, AUTOLOGOUS BONE MARROW ASPIRATE, TENDON-DERIVED PROGENITOR CELLS

The use of mesenchymal stem cell (MSC) therapy in veterinary orthopedics has been described including treatment of subchondral bone cysts, bone fracture repair, and cartilage repair.[26,32] However by far the most frequent use of MSCs has been in the treatment of overstrain-induced injuries of tendons in horses.[35] Although the mechanism of action in how these cells influence their "trophic" activity is still under intense investigation, many models of animal studies suggest that MSCs secrete bioactive molecules that (1) inhibit apoptosis and limit the field of damage or injury, (2) inhibit fibrosis or scarring at sites of injury, (3) stimulate angiogenesis and bring in a new blood supply, and (4) stimulate the mitosis of tissue-specific and tissue-intrinsic progenitor cells.[7] Historically, in untreated racehorses, the reinjury rate in tendons sustaining superficial digital flexor (SDF) tendonitis is approximately 56%.[12] Due to the scar tissue formation, the primary need to restore functionality to tendons has encouraged the development of regenerative strategies. Regenerative therapies require an exogenous cell source. Tendons and ligaments do not have a lack of cellular inflammation following injury however; cells actually involved in the synthesis of new tissues are mostly locally derived. Transplantation of MSCs into various injured skeletal tissues has been shown to promote healing, and the use of autologous cells has a benefit since they do not incite an immune response from the host.[8,14,22] Current theories on how transplanted MSCs act in tendon when injected intrathecally are that they either differentiate into cells capable of synthesizing tendon matrix or they secrete important factors that induce adjacent cells to synthesize tendon matrix.[35]

Currently, two techniques exist for MSC transplantation using intrathecal injections. One has used non-adipocyte-cell mixture from fat, and the second has used cultured bone marrow aspirate. The current widely used fat-derived stem cells do not involve a culture step and therefore have the advantage of cheaper cost and speed of preparation (cells are returned to the practitioner with 48 hours). However, the cell mixture is believed to

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be heterogeneous with regard to cell type. A small controlled study using adipose-derived cells in a collagenase model of tendon injury reported some benefit in terms of crimp pattern.[33] Another study reported a return to athleticism in 14 out of 16 horses with SDF tendonitis with no comment on reinjury rate.[9] Up until now, no head-to-head comparison studies of adipose-derived to bone marrow-derived MSCs have been performed in clinical cases of naturally occurring disease.

The culture-expanded bone marrow-derived MSC therapy involves aspirating bone marrow from the sternum or tuber coxae, transferring to a laboratory for culture and expansion, and then implanting the cell population (approximately $10–50 \times 10^6$) under ultrasound guidance.[20] Continued investigation of tissue uptake of these cells has shown retention of the stem cells within the lesion for up to 9 weeks when injected intralesionally.[18] This technique has been widely used in the United States, the United Kingdom, Europe, and Australia.

Currently, long-term follow-up is encouraging. In a 2008 study, over 168 racehorses undergoing this regimen were shown to have an 18% reinjury rate.[41] This is in contrast to a 56% reinjury rate for horses receiving no intralesional therapy. Two smaller-scale studies one in research horses[39] and one in 11 racehorses[34] also reported improved tendon repair. In a more recent study by Godwin et al., 141 racehorses with overstrain injuries of the SDFT were treated intralesionally with bone marrow-derived MSC and followed with repeat ultrasonography. A re-injury rate of 27.4% was reported for this population after a 2-year period.[19] No adverse effects were reported in these studies other than ultrasonographic evidence of needle tracks.

The efficacy of administering stem cells via regional limb perfusion (RLP) has also been evaluated via technetium-99m-labeled cells, which has shown a homing effect to the region of injury.[10] Additionally, the efficacy of intra-arterial administration of MSCs has recently been investigated. Technetium-labeled MSCs injected into the median artery via ultrasound guidance were shown to perfuse into the entire distal limb via scintigraphic imaging.[13] Longer-term evaluation of tissue uptake when administered via RLP or intra-arterially has yet to be investigated.

In addition to autologous stem cell usage, in which a sample is harvested from the patient, cultured, and reinjected intralesionally, investigation of the safety of allogenic stem cell use has been reported. In a retrospective study of 164 horses receiving bone marrow-derived stem cells from a single donor, 10/230 injection sites were reported to have adverse reactions which is not dissimilar to allogeneic administration.[43] Adverse reactions were defined as pain, swelling, heat, or lameness associated with the injection site. Seven of these reactions were in soft tissue injections.[43]

Tendon-derived progenitor cells (TDPC) have also been investigated in recent years as an alternative to bone marrow or stem cell therapies. Cells are harvested from a donor tendon site, such as the lateral digital extensor tendon, and then cultured for intralesional injection. In a recent study model using collagenase-induced tendon lesions, improved tensile strength and fiber alignment were shown in the TDPC-treated groups as compared with the saline-treated controls.[11] Studies now need to be performed comparing the efficacy of TDPC with bone marrow-derived and adipose-derived MSCs.

BLOOD-DERIVED BIOLOGICS: PLATELET-RICH PLASMA, AUTOLOGOUS CONDITIONED SERUM, AUTOLOGOUS PROTEIN SOLUTION, AND BONE MARROW ASPIRATE

Platelet-rich plasma (PRP) has been used for treatment of soft tissue injuries and bone healing and has been investigated for regeneration of bone, cartilage, tendon, and ligament. The reason behind the use of PRP comes from the knowledge that growth factors are released from platelet α-granules including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), fibroblastic growth factor (FGF), vascular endothelial growth factor (VEGF), insulin-like growth factor-I (IGF-I), and epidermal growth factor (EGF). Many animal models have demonstrated positive effects of these growth factors, both alone and in concert with each, in enhancing cellular migration and proliferation, angiogenesis and matrix deposition in tendon and wound healing and repair.[1,3,38]

Current studies report beneficial effects of PRP on equine ligament and tendon both *in vitro* and *in vivo*.[30,42,44] *In vitro* studies support the belief that PRP supplies high levels of growth factors and the presence of these growth factors results in higher type I collagen and cartilage oligomeric matrix protein (COMP) and reduced levels of inflammatory mediators such as matrix metalloproteinases (MMP) 3 and 13. A recent *in vivo* study reported improved repair of the SDFT when core lesions were induced mechanically.[5] When lesions that were injected with PRP were compared with lesions injected with saline, tendons had better mechanical characteristics as well as higher collagen, glycosaminoglycan, and cellularity. These findings correlate with the *in vitro* effects reported. A more recent randomized controlled study was performed using intralesional injection of either PRP or saline in 20 horses with naturally occurring tendinopathies. At 12-month follow-up, 80% of the PRP-treated horses had reached their previous or a higher level of performance as compared with 50% in the control. However, after 24 months only 60% of the treated horses and 40% of the control were still used at this level.[17] Additionally, in a retrospective review of 127 National Hunt racehorses, intralesional PRP was not superior to other treatments in the studied population, which included controlled exercise and bar firing.[45]

PRP is also commonly used in conjunction with extracorporeal shockwave therapy (ESWT) (see section on topical/local). An *in vitro* study showed that concentrations of TGF-β_1 and PDGF-ββ were significantly increased when PRP was exposed to ESWT; however this model has not been validated *in vivo*.[40]

Autologous conditioned serum (ACS) has become popular for its anecdotal anti-inflammatory effects and rapid improvement in lameness. ACS is similar to PRP in its rapid processing and ease of acquisition. Whole blood is processed via exposure to glass beads, which stimulates

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the expression of anti-inflammatory cytokines, including IL-1Ra and IL-10.[24] In a recent randomized controlled study,[16] naturally occurring tendinopathies of the forelimb SDFT were treated with either ACS or saline.[16] In ACS-treated limbs, lameness and swelling were significantly decreased, while ultrasonography revealed a decrease in size and increased echogenicity in the treated groups following a single intralesional injection. Recurrence of tendon injury was not reported in any of the horses in this study up to 2–4 years' post-diagnosis. Five of eight ACS-treated horses returned to previous levels of work, while four of five control horses also returned to previous work.

A newer class of biologics, autologous protein solution (APS), contains high concentrations of growth factors and anti-inflammatory cytokines, such as IL-1 receptor antagonist, IL-10, IGF-1, and TGF-β, and has had promising results intra-articularly.[4] APS has a similar processing time and centrifugation style to PRP. APS is distinct in that a second centrifugation step further concentrates the cell solution in the presence of polyacrylamide beads resulting in a solution with higher concentrations of interleukin-1 receptor antagonist proteins than PRP.[23] To date, APS has not been investigated intralesionally.

Autologous bone marrow aspirate (ABMA) has been used in recent years as an inexpensive and fast intralesional treatment modality, by which stem cells and growth factors may be administered into a lesion without conditioning or culture. In a recent study of racehorses with SDFT injuries treated with intralesional ABMA at the time of desmotomy of the distal accessory ligament of the SDFT (DAL-SDFT), Thoroughbreds showed an 82% return to racing with 59% starting five or more times. Of Standardbreds with similar lesions and treatment, 76% had one or more starts, and 62% had five or more starts.[37] The success rates of horses treated with ABMA are improved over previously reported rates of Thoroughbreds undergoing DAL-SDFT alone (72% return to racing and 48% with five or more starts).[25]

CORTICOSTEROIDS

Perilesional corticosteroids are occasionally used acutely to treat tendonitis/desmitis. However they are considered contraindicated, especially for long-term usage, because they may delay collagen formation.[36] However, some clinicians use a single perilesional dose of triamcinolone (6–9 mg) or methylprednisolone acetate (40 mg) perilesionally in horses with peripheral tendon lesions or suspensory ligament desmitis. Results using this therapy vary and should probably be reserved for tendon or ligaments in which minimal or no structural damage is seen ultrasonographically. Furthermore, dexamethasone acetate has been shown to differentiate tendinous stem cells into fatty tissues, cartilage-like tissues, and bony tissues, which may put tendinous structures at risk of rupture.[47] Anecdotally in the horse, dexamethasone acetate has been associated with dystrophic mineralization that has been noted following injection of this corticosteroid into soft tissues.

Numerous other uses have been described for local injection of corticosteroids in areas of inflammation, such as over splint bones, muscle/back soreness, and various soft tissue inflammatory conditions or trauma. The choice and amount of steroid seem to be empirical and clinician dependent; however, this does not seem to greatly impact the efficacy of the injections.

MISCELLANEOUS

Sarapin is occasionally utilized as an intralesional treatment for lesions associated with chronic nerve pain. Sarapin is an extract derived from the pitcher plant and is believed to have a numbing effect in chronic pain. The only study performed in the horse however could not detect analgesic effects of sarapin in a model of acute pain using heat.[21] Nonetheless, this therapy is commonly utilized to treat various forms of desmitis and muscle soreness with a widely believed suspicion that a temporary analgesic effect is produced. Sarapin is often combined with other medications such as corticosteroids and is used primarily to treat lameness conditions of the axial skeleton such as back and sacroiliac problems.

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ORAL/NUTRITIONAL

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Lameness is one of the most prevalent diseases, which affects the equine industry. It is considered the most common and important performance-limiting problem in horses. This section discusses oral formulations used to treat the causes of lameness. Specifically, it focuses on oral formulations of nonsteroidal anti-inflammatory drugs (NSAIDs) and the most common compounds used in equine nutraceuticals.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

NSAIDs are a large group of drugs with differing degrees of analgesic, anti-inflammatory, and antipyretic properties (Table 8.3). In a survey of horse owners and trainers, 96% of respondents stated that they use NSAIDs, and 82% said they administer them without consulting a veterinarian.[101] However, NSAIDs are also heavily prescribed by veterinarians as demonstrated in an observational study of multiple practices in multiple countries where clinicians prescribed NSAIDs in the United States (42.4%) more than those in Canada (34.2%), or in the United Kingdom (28.6%).[39] Oral formulations come in many varieties such as pills, paste, granules, and powder, allowing the consumer to choose which formulation is best for each individual. Some of these medications can also be administered systemically via intramuscular or intravenous injection (Table 8.3). In general, NSAIDs act by inhibiting cyclooxygenase (COX) enzymes that convert arachidonic acid into prostaglandins and thromboxanes. There are two well-known isoenzymes of the COX enzymes, COX-1 and COX-2.[162] In general, COX-1 is considered the housekeeping isoenzyme responsible for producing prostaglandins involved in normal physiological functions such as gastric and renal function and hemostasis.[100] The COX-2 isoenzyme is considered to be an important inducible mediator of inflammation in several organs and is primarily responsible for the inflammatory pathway.[100] However, the concept that COX-2 is purely involved in inflammation has changed since the COX-2 isoenzyme has been found to be constitutively formed in the brain, spinal cord, kidney, uterus, and some other regions.[134] In fact, one study demonstrated that in volume-depleted horses a COX-2 preferential agent (meloxicam) had similar adverse renal effects as the nonselective NSAID, phenylbutazone (PBZ).[117]

The variation in efficacy and toxicity of the different NSAIDs is closely related to inhibition of the different COX isoenzymes (Table 8.3). Some are more potent inhibitors of COX-1 than COX-2, some equally inhibit both, and others inhibit COX-2 more than COX-1.[100] In general, the anti-inflammatory and analgesic properties of NSAIDs are believed to be mainly due to inhibition of the inducible COX-2, whereas the adverse effects seem to be caused by inhibition of the constitutive COX-1.[96] This has led to the development of NSAIDs with a selective inhibition of COX-2. In humans, COX-2-selective inhibitors demonstrated adverse cardiovascular and thromboembolic effects, but horses do not appear to be predisposed to these adverse effects.[165] It is also important to note that COX-2 selectivity is only achieved when administered at correct dosing; overdosing can lead to similar adverse effects of nonselective NSAIDs.[165]

Recommendations for NSAID usage should be formulated in attempt to minimize adverse effects when it is determined that an oral NSAID may be a beneficial

Table 8.3. Commonly used NSAIDs and their modes of action, formulations, and doses for equine musculoskeletal disorders.

Name of NSAID	Primary inhibitory action	Available formulations	Recommended dose
Phenylbutazone	Cox-1 and -2	Powder, tablets, paste, and injectable solution	2.2–4.4 mg/kg SID or BID
Flunixin meglumine	Cox-1 and -2	Paste, granules, and injectable solution	1.1 mg/kg SID
Acetylsalicylic acid	Cox-1 > Cox-2	Gel, powder, granules, paste, and tablets	25–35 mg/kg SID or BID
Meclofenamic acid	Cox-1 and -2	Granules	2.2 mg/kg SID for 5–7 days
Naproxen	Cox-1 and -2	Granules, tablets, and suspension	10 mg/kg SID or BID
Firocoxib	Cox-2	Paste, injectable solution, and tablet	0.1 mg/kg SID, 0.09 mg/kg SID IV
Carprofen	Cox-2 > Cox-1	Tablets and injectable solution	0.7 mg/kg SID
Vedaprofen	Cox-2 > Cox-1	Gel and injectable solution	2 mg/kg loading dose followed by 1 mg/kg q12 h
Meloxicam	Cox-2 > Cox-1	Suspension, injectable solution, oral solution, and tablets	0.6 mg/kg SID

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treatment option. In general, oral NSAIDs should be used at the lowest effective dose and frequency as possible, for the shortest period of time. Special consideration should be used when treating susceptible breeds (ponies), foals, and systemically ill, dehydrated, or old horses. NSAIDs are commonly used in performance horses for recovery of minor injuries. However, most sport horse disciplines have rules about using NSAIDs before or during competitions. See each individual governing body for guidelines regarding appropriate therapeutic use of NSAIDs for that discipline.

Phenylbutazone (Butazolidin®, Butatron™, Bizolin®, Phenylbute™, Phenylzone®, Equiphen®, Butequine®, Superiorbute®, and Equizone 100™)

PBZ is an acidic, lipophilic NSAID that is classified as an enolic acid and is metabolized in the liver and excreted in the urine. Following oral administration, PBZ is well absorbed in the stomach and small intestine.[94] The plasma half-life after oral administration has been reported to be between 6.2 and 6.7 hours.[75] However, absorption can be influenced by the drug's formulation and the route of administration.[4,75] For example, the paste formulation offers a greater rate and extent of absorption than tablet or powder formulations, and peak plasma concentrations and onset of action can be delayed by administration close to feeding.

Oral PBZ is an inexpensive NSAID that has potent pain relief, antipyretic, and anti-inflammatory properties. It provides pronounced analgesia and reduction in inflammation in many common lameness problems, including naturally occurring chronic osteoarthritis (OA).[36] It is often administered prior to surgery to minimize pain and inflammation associated with orthopedic surgical procedures. However, studies have demonstrated that oral PBZ may have negative effects on cartilage and bone metabolism. A study in healthy horses receiving oral PBZ reported a decrease in bone activity (decrease in mineral apposition rate and a reduction in the magnitude of healing) compared with horses not receiving PBZ.[124] In another study, PBZ administered (4.4 mg/kg) orally twice daily for 14 days to healthy horses resulted in significant decreases in proteoglycan synthesis by suppressing chondrocyte metabolism in articular cartilage explants.[8] Recovery of chondrocyte metabolism took 2 weeks to normalize after cessation of treatment. In contrast, no significant adverse effects were identified using systemic cartilage and bone biomarkers when PBZ (4.4 mg/kg) was administered orally twice daily for 3 days followed by a lower dose (2.2 mg/kg) for 7 days in healthy horses.[48]

Oral PBZ has been widely studied for its toxic effects on horses. It is considered relatively nontoxic at repeated doses of 2.2 mg/kg twice a day or less.[137] Clinical signs reported with PBZ toxicity in horses are anorexia, depression, loss of weight, abdominal edema, hypoproteinemia, leukopenia, anemia, and death within 4–7 days.[132] The gastrointestinal (GI) tract is the most commonly affected area following oral administration, causing ulcers (oral, esophageal, gastric, cecal, and right dorsal colon) and a protein-losing enteropathy.[132] Renal papillary necrosis has also been described and may be due to the inhibition of prostaglandins that maintain renal blood flow and/or direct toxic effect of the drug or its metabolites.[84,117] Because of this narrow margin of safety and adverse effects, special considerations or alternative therapies should be considered when horses require long-term administration. Often in severely affected orthopedic cases, horses can be safely maintained on 2.2 mg/kg/day of PBZ for months when gastroprotectants are used concurrently.

Combination of oral PBZ with other NSAIDs may enhance the analgesic properties of these drugs in performance horses.[74,120] Some studies have shown a better clinical improvement in lameness when a combination of drugs is used than when PBZ is used alone,[74,120] whereas others have not.[47] Caution should be used when combining NSAIDs to minimize the potential toxic effects.

Flunixin Meglumine (Banamine®)

Flunixin meglumine is a derivative of nicotinic acid that exhibits analgesic, anti-inflammatory, and antipyretic activity. After oral administration the drug is rapidly absorbed with a peak in plasma levels within 30 minutes, and the plasma half-life is approximately 1.6 hours.[20] This rapid absorption may be important in minimizing potential ulcerogenicity.[20] The onset of action after oral administration occurs after 2 hours, with the greatest effect between 2 and 16 hours; some activity may persist for up to 30 hours.[62] Similar to PBZ, recent feeding delays absorption.[157] Oral administration of flunixin is relatively safe. In one study, three times the recommended dose given orally for 15 days failed to induce toxic effects.[62] However, oral administration of flunixin at 1.1 mg/kg/day for 30 days in foals resulted in oral and gastric ulceration.[140] In addition, high doses for long periods can cause GI intolerance, hypoproteinemia, and hematological abnormalities.

Oral flunixin meglumine can be used to treat musculoskeletal disorders in horses,[62] but because of its cost compared with PBZ, oral formulations are used infrequently. In general, oral flunixin has been used mostly to treat musculoskeletal injuries associated with soft tissue structures such as muscle and tendons. However, there are no studies indicating that it is better or worse than other NSAIDs at controlling pain and inflammation associated with the musculoskeletal system. When flunixin is combined with other NSAIDs, some studies have shown enhanced analgesic properties of these drugs in performance horses compared with when used alone,[74,120] whereas others have not.[47]

Acetylsalicylic Acid (Aspirin)

Aspirin is a weak acid that reduces platelet aggregation and has analgesic, anti-inflammatory, and antipyretic properties. It is only available in oral forms and has a limited clinical use in the horse. It is best absorbed in the acidic environment of the upper GI tract. In ponies, oral administration of salicylate demonstrated rapid absorption with a peak in plasma levels within an hour, with the highest concentrations of drug attained in the liver, heart, lungs, renal cortex, and plasma.[31] Aspirin affects platelet function at low dose rates, reducing clotting times and thrombus formation.[16] Aspirin has been reported to decrease platelet numbers

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and prolong bleeding time in horses with doses between 12 and 24 mg/kg.[16,70,141] These effects can occur after a single dose and can last as long as 48 hours.[16,70] Therefore, caution should be used and close monitoring performed when using this drug on horses before surgery or with bleeding tendencies or anemia. Due to its effect on platelets, aspirin has been historically used for the treatment of navicular syndrome and chronic laminitis.[97] However, its therapeutic benefits for these diseases are not well defined.

Meclofenamic Acid (Arquel)

Meclofenamic acid (MA) is another oral nonselective NSAID used to treat lameness and chronic musculoskeletal conditions such as navicular disease, OA, and laminitis in horses.[25] Compared with other NSAIDs, MA has a slow onset of action of 36–96 hours for full effect.[25] It is not clear whether feeding time dramatically affects absorption because one study demonstrated that fasted and nonfasted ponies had similar oral absorption,[131] whereas another study demonstrated that plasma levels could be delayed by feeding.[136] The reported bioavailability after oral administration varies between 56% and 90%.[68,131] MA may prove to be useful for OA because high levels can be found in synovial fluid and articular cartilage.[25] High doses produced clinical signs of toxicity similar to those of PBZ (at a dose 13–18 mg/kg).[84,138]

Naproxen (Equiproxen, Naprosyn)

Naproxen is a nonselective NSAID with analgesic and antipyretic properties that has been used primarily to treat myositis and soft tissue problems.[27,138] In a study using an equine myositis model, oral naproxen was reported to be superior to oral PBZ, with faster relief of inflammatory swelling and associated lameness.[69] After oral administration the bioavailability of the drug is approximately 50% with peak plasma levels reached at 3–4 hours; the plasma half-life is approximately 4 hours.[138] Naproxen has a wide margin of safety. Oral administration of four times the recommended dose for 42 days did not cause signs of toxicity.[138] In another study, administration of the recommended dose of naproxen for 14 days, followed by twice the dose for another 7 days, did not alter plasma protein concentrations.[133] Even though this drug has been marketed for soft tissue conditions, successful usage in the human field for treating joint pain may suggest that it can be used to treat inflammatory swelling and associated lameness.[69,97]

Firocoxib (Equioxx®)

Firocoxib is a COX-2-selective NSAID that has been approved by the FDA for controlling pain and inflammation associated with OA in horses. The bioavailability after oral administration in horses is 79%, with a time to peak concentration of 3.9 hours and elimination half-life of 30 hours.[60,80,81,85] It is metabolized in the liver and excreted in the urine.[85] It is well distributed in the body, including synovial fluid, liver, fat, kidney, and muscle.

Firocoxib is available in two oral formulations (paste and tablets). When a single dose of 57 mg was administered to 6 horses, the paste revealed a better bioavailability, higher C_{max}, shorter T_{max}, greater area under the concentration curve (AUC), and greater inhibition of prostaglandin E_2 (PGE_2) when compared with the tablets.[60] The efficacy of different doses of firocoxib in horses with chronic lameness was assessed using objective assessment with a force plate, showing that a dose of 0.1 mg/kg orally once a day was found to be most effective in reducing chronic lameness in horses.[3] When administering this oral dose (0.1 mg/kg SID) over multiple days, it has been shown that it can take up to 7 days to reach steady state.[85] However, if a loading dose (0.3 mg/kg) is administered 24 hours before starting a multidose regimen at 0.1 mg/kg, the steady-state concentration of the drug can be achieved after the first day of treatment.[26]

In a study of horses with chronic OA and navicular syndrome,[36] no significant differences in clinical improvement were found between horses treated for 14 days with firocoxib compared with PBZ treatment. However, a greater proportion of horses treated with firocoxib had improvement in scores for pain on manipulation or palpation, joint circumference, and range of motion compared with PBZ. In another study of a large number and cross section of horses with musculoskeletal pain or lameness associated with OA, firocoxib paste significantly improved lameness scores, comfort, and mobility in horses with the most change occurring within the first 7 days of treatment.[112]

Compared with other nonselective NSAIDs, firocoxib is relatively safe. No clinical and biochemical signs of NSAID toxicity were reported when using the recommended dose (0.1 mg/kg).[43] A pharmacokinetic and safety study was performed in neonatal foals, where 36-hour-old neonates received 0.1 mg/kg orally SID for 9 days; it showed that firocoxib was rapidly absorbed with no reportable adverse effects.[63] In a postoperative *ex vivo* study, firocoxib action on COX-2 was similar to flunixin meglumine in that they both prevented its increase 24 hours after surgery; the firocoxib group demonstrated a sparing effect of COX-1 activity with greater activity than flunixin.[38] However, it has been reported that an oral dose of 0.1 mg/kg SID for 10 days can still develop gastric ulceration, but the severity of the ulceration being less severe than with PBZ administration (4.4 mg/kg PO SID).[121] In addition, co-administration with another NSAID can eliminate the benefit of the COX selectivity; adverse renal issues were present when oral firocoxib (0.1 mg/kg SID) was combined with oral PBZ (2.2 mg/kg SID) for 10 days.[79] For this reason, some equine governing bodies are no longer permitting the combination use of firocoxib with other NSAIDs.[165]

The main downfall to firocoxib is that it is more expensive than PBZ. Therefore, the economic feasibility must be discussed with the client. This can often lead to off-label use of the chewable canine tablets by owners due to the substantial difference in cost between the formulations for the different species. Because of this, one study examined the effect of 57 mg of the canine tablet or the equine paste administered orally to horses for 7 days.[5] The collected blood was challenged with lipopolysaccharides (LPS) to induce PGE_2. There was no difference between formulations in reducing PGE_2 production or in plasma firocoxib concentrations. Therefore, they conclude that tablets may be viable option if extra-label use can be justified,[5] but it is important to note that one cannot legally prescribe the

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formulation labeled for one species to another (in other words, the canine formulation cannot be legally prescribed for horses just because it is cheaper).[165]

Carprofen (Zenecarp, Rimadyl®)

Carprofen is a propionic acid NSAID approved in Europe for oral use in horses. Its mechanism of action in horses is still unclear; however, it is described as being a more effective analgesic than anti-inflammatory agent.[99] Carprofen in horses was well tolerated when given at twice the oral dose for 14 consecutive days.[99] The benefit of using this NSAID in horses compared with other available NSAIDs requires further investigation.

Vedaprofen (Quadrisol®)

Vedaprofen is an arylpropionic acid NSAID with anti-inflammatory, antipyretic, and analgesic properties. Vedaprofen is approved in Europe for oral use in horses. It is recommended for musculoskeletal disorders and soft tissue lesions. Side effects of this drug are those associated with the use of NSAIDs in general. The main toxic effect is ulcer formation in the GI tract, making it contraindicated for use in foals under 6 months of age.[146] Use of this NSAID in horses compared with other available NSAIDs requires further investigation.

Meloxicam (Metacam®)

Meloxicam is a relative COX-2-selective NSAID intended for the treatment of inflammatory orthopedic problems, including chronic musculoskeletal and soft tissue disorders.[10,105] It is available for horses in Europe. Meloxicam was shown to be more effective than PBZ in decreasing pain in an LPS synovitis model.[143] It was also shown to be effective in controlling swelling and lameness postoperatively after partial resections of fractured splint bones compared with placebo controls.[154]

Side effects seen with this drug are those routinely associated with the use of NSAIDs. Oral doses of 0.6 mg/kg were well tolerated when administered daily for 2 weeks[151] and 6 weeks,[109] but 1.8 and 3 mg/kg demonstrated decreased protein and albumin concentrations, GI and renal damage, or bone marrow dyscrasia.[109] Oral administration of three different doses (0.6, 1.8, and 3 mg/kg q24) for 14 days demonstrated less compromise to the gastric mucosa than oral administration of decreasing doses of PBZ over the same time frame; however, there was no difference in ulceration between the NSAIDs.[30] Oral doses of 0.6 mg/kg for 3 weeks were well tolerated in 6-week-old foals, and interestingly, foals demonstrated no adverse effects after 1.8 mg/kg was administered BID for 7 days.[116] Caution should be used and close monitoring performed when using this drug on horses treated concurrently with another NSAID, corticosteroid, or nephrotoxic medication.[148]

NUTRACEUTICALS

The nutraceutical term has undergone many different definitions over time but is basically any substance that is a food, or part of a food, that can be administered orally to provide or stimulate production of raw materials or biochemical pathways required for normal bodily functions. Although these substances are neither nutrients nor pharmaceuticals, they are generally used in an attempt to lower the dose of other drugs that are more problematic and provide medical benefits that prevent or treat disease. Included in this broad category of products are nutrients, dietary supplements, functional foods, and phytochemicals (including herbs).

The FDA does not recognize nutraceuticals as foods or drugs. However, based on the definition of nutraceuticals, the FDA perceives veterinary nutraceuticals as unapproved drugs even though they are not labeled or marketed as drugs. The FDA does not regulate these products unless they become unsafe or are associated with labels that claim a drug use. The Center for Veterinary Medicine (CVM) has allowed products to be marketed as nutraceuticals provided that they do not claim to treat, cure, or mitigate disease. Ultimately, there is no requirement to prove safety or efficacy of a nutraceutical for its intended use.[12]

The Dietary Supplement Health and Education Act (DSHEA) allows manufacturers to make claims with regard to health, structure or function, and nutrient content of a nutraceutical and requires that the manufacturer ensure that its product is safe before marketing.[142] In general, the manufacturer must identify each dietary ingredient contained in the product and adhere to state licensing requirements.[98] However, neither the DSHEA nor FDA have set up a system to monitor manufacturers. There is no requirement for manufacturers to guarantee high quality and batch-to-batch consistency, and product safety is completely up to each individual manufacturer.[142] In 2001, the National Animal Supplement Council (NASC), a nonprofit trade organization, was founded by animal supplement industry stakeholders to try to improve and standardize the industry.[145] As part of this goal, the NASC developed the concept of a "Quality Seal Program." The idea was that the presence of the seal on a product indicates to the consumer that the company is reputable and has undergone some form of quality control. To have a seal placed on their product, the company must demonstrate compliance in four main areas to NASC investigators: a quality control manual that demonstrates standard operating procedures that are in place, an adverse event reporting system to evaluate the ingredients within each product, proper labeling guidelines, and inclusion of any warning and caution statements recommended by the FDA's CVM for particular ingredients. A list of NASC companies that completed the quality audit can be found at www.nasc.cc/members/.

Nutraceuticals are often used by many owners or trainers independent of their veterinarians. Products have become widely available in many formulations including capsules, tablets, and powder, and they are incorporated within packaged feed. When using or recommending these products, documentation validating the contents, purity, and quality control procedures should be requested from the manufacturer to examine and understand the quality, efficacy, tolerance, and safety of each individual nutraceutical.[142,159]

Several of the products in the equine market have considerable variability in purity, formulation, and

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consistency between batches. Although these products are marketed with "guaranteed analysis," only a small number have consistently met the label claims.[1, 86,87,118,144] In fact, truth-in-labeling problems have been documented, in which ingredients listed on the package were not present at the claimed concentration or purity. The products that have the most inconsistency in concentrations compared with the label claims tend to be those that state that they are a "complex," "formula," or "blend" of ingredients with no weight of each component listed.[144] In general, products should be avoided if they do not have information on the quality, make exaggerated claims, or rely on testimonials.[142] The presence of lot numbers and expiration dates generally offers some evidence of accuracy in labeling, but does not guarantee it.[12]

Many manufacturers with similar products attempt to compete in the market with claims based on research conducted by another company, even though there is no proof of comparable efficacy. Efficacy of these products in the horse is hard to prove, even for those products in which research was conducted; a systematic review of five studies using nutraceuticals for treating OA in horses stated that the strength of evidence of efficacy to improve pain or gait abnormalities was poor.[152] Because these products are oral, it is also important to ensure that there is adequate evidence of absorption in the species of interest, or else the product is essentially useless.[142] As with many medications, each particular individual may respond in an adverse fashion to various ingredients within the product. This could be a reaction to the main ingredients, contaminants, or inert ingredients that are added to help disperse or dilute the product.[12] Unfortunately, to date, little to no information exists about these products, which is important to consider when discussing safety of the product with a horse owner or trainer. Most of the information is anecdotal and is based on the lack of any major adverse reactions after treatment.[142]

One should not, and cannot, assume that these products will not cause harm. Just because there is a lack of published adverse reactions to a nutraceutical does not mean that one should extrapolate that the product is safe. In fact, most manufacturers will not spend the money to prove safety, so the lack of side effects does not mean that a product is safe.[142] Unfortunately, even when safety data is present on a particular product, the variability in concentrations of the ingredients within and between batches can make this data inaccurate. It is also worth noting that these products have only been around for a few decades, so there is limited data regarding the effects of long-term administration.

The remainder of this section discusses the known scientific effects of the most common compounds contained in equine nutraceuticals that are currently available.

Glucosamine

Glucosamine (GLN) is an aminosaccharide essential for normal growth and repair of articular cartilage.[115,139] GLN compounds have been used as nutraceuticals in horses due to their possible role in stimulating chondrocyte metabolism and reducing inflammation in the articular cartilage.

Exogenous GLN can be produced synthetically or derived from marine exoskeletons or beef carcasses. There are three commercially available forms of exogenous GLN: hydrochloride (HCl), sulfate, and *N*-acetyl-D-glucosamine. Several reports have demonstrated differences among the available GLN forms. For instance, *in vitro* studies have demonstrated that GLN HCl and GLN sulfate appear to inhibit cartilage degeneration more consistently than *N*-acteyl-D-glucosamine.[45,46,129] The GLN sulfate form has been postulated to be more efficacious because serum and/or synovial fluid concentrations of sulfate increase after GLN sulfate administration.[59,65,102,130]

Oral bioavailability of GLN HCl in the horse has been reported to range from 2.5% to 6.1%, which has been attributed to a large volume of distribution, poor absorption from the GI tract, and extensive tissue uptake.[37,82] Radiolabeling studies have shown that there is good distribution of GLN to articular cartilage with levels exceeding those in the plasma.[128] However, GLN delivery to the articular cartilage does not necessarily mean that it becomes incorporated into articular cartilage. In fact, after oral dosing in the horse, it has been shown that synovial fluid levels of GLN are less than 10% of those in the serum at the same time.[82] This indicates that GLN does not diffuse readily into the synovial fluid from the plasma, suggesting that the effects of GLN may lie in its effect on nonarticular tissues.[82] The presence of synovial inflammation induced by LPS in carpal joints led to significantly higher synovial GLN concentrations compared with levels attained in healthy joints following oral administration of GLN HCl,[103] as well as the potential to mitigate production of inflammatory mediators and elicit reparative processes to articular cartilage.[83]

Exogenous GLN has been shown *in vitro* to have no detrimental effects on normal articular cartilage explants[32] or on chondrocyte metabolism after long-term exposure.[67] Many *in vitro* studies have generally found that GLN induces the production of new cartilage while protecting cartilage that is already present.[6,32–34,71] GLN stimulates synthesis of proteoglycans (PG) and collagen while inhibiting PG degradation. This may occur partly via downregulation of matrix metalloproteinases (MMP) and aggrecanases.[110,128] The anti-inflammatory effects are likely prostaglandin-independent and may be due to enhanced production of HA by synoviocytes.[44,66,82,115,131] In addition, GLN may protect against some of the negative effects of steroids on cartilage, as demonstrated in an *in vitro* equine chondrocyte pellet model in which GLN had a protective effect against the inhibition of proteoglycan production caused by methylprednisolone.[14]

Few equine *in vivo* studies have used only GLN. Oral administration of GLN in young horses in training showed no significant treatment effect on serum biomarker concentrations of bone and cartilage metabolism.[17,44] High levels of GLN used for *in vitro* experiments that were effective have never been achieved in experimental models in humans and animals.[153] Therefore, extrapolating the results from *in vitro* studies to the *in vivo* setting must be done with caution. The most consistent information that can be extrapolated from human studies is that it appears to take 4–8 weeks before GLN begins to act and that GLN would likely give the best results when used preventatively – when minor lesions are present prior to the advancement of the disease process.[108]

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The quality of products that contain GLN has been questioned. One study compared the measured amount of GLN in commercially available equine oral supplements to levels listed on each product's label.[110] The study concluded that of the 23 products considered, 9 (39.1%) contained less GLN than claimed by the manufacturer and 4 (17.4%) contained less than 30% of the listed amount. In another study examining five GLN equine products, the actual composition of GLN varied between 63.6% and 112.2% of the label claim.[118] A large variation in the average recommended daily maintenance dose of GLN was also found in the product labels, ranging from 1,800 to 12,000 mg GLN orally per day for an average sized mature horse. This variability should be considered when evaluating the efficacy of these products.[111]

Chondroitin Sulfate

Chondroitin sulfate (CS) is a long-chain polysaccharide that is a normal constituent of aggrecan, and it constitutes about 80% of all glycosaminoglycans in articular cartilage. Within the cartilage it plays an important role in creating osmotic pressure due to its polyanionic charge. CS is often derived from shark and bovine cartilage, and it is rather expensive to synthesize and extract. The species or tissue of origin of CS can determine differences in the concentrations, pharmacokinetics profile, molecular composition and weight, metabolic fate, and therapeutic results.[76,163] Therefore, it cannot be assumed that all formulations of CS have the same clinical effect.

CS is orally absorbed in horses; however, the molecular weight and source can have a direct influence on its permeability across the GI tract and its bioavailability.[37, 40] Studies in other species have shown that the GI mucosa contains a variety of glycosaminoglycan (GAG)-degrading enzymes, suggesting that CS is not absorbed as an intact molecule due to a potential enzymatic degradation.[88,89] It has been proposed that the liver can further modify the final CS molecule prior to reaching circulation.[54,88,89] Reports using radiolabeled CS have demonstrated that it achieves high concentrations in plasma, articular cartilage, and synovial fluid.[24]

Exogenous CS has been shown *in vitro* to have no detrimental effects on normal articular cartilage explants[32] and to increase the synthesis, or decrease the turnover, of proteoglycans/GAGs. *In vitro* studies have also demonstrated a profound anti-inflammatory effect on several tissues involved with joint metabolism. This may be due in part to exogenous CS-stimulating HA synthesis in synoviocytes along with improved composition (viscosity) of HA.[23,119,125] In addition, anti-inflammatory effects may stem from a downregulation of PGE_2 as well as from NO production.[19,113] CS has also been shown to inhibit MMP and aggrecanase expression/activity in a dose-dependent manner.[113,125,135]

In a synovitis model in horses, CS was found to be markedly less effective than polysulfated glycosaminoglycans (PSGAG) administered intramuscularly (Adequan®, Luitpold Pharmaceuticals, Inc., Shirley, NY) for relief of lameness, stride length, and carpal flexion.[160] In another study using the same model, there was evidence that CS had therapeutic value irrespective of the route of administration (oral or intramuscularly) by improving joint function and reducing lameness scores.[35] However, the time of onset of clinical improvement was slower with oral administration of CS. In a human study, response to treatment with oral CS appeared later (at 60 days) was greater at the end of the 3 months' treatment and lasted up to 3 months after the treatment,[104] suggesting that response not only may take time but also may last beyond the last administration. In a study examining five CS equine products, the actual composition of CS ranged from 22.5% to 155.7% of the label claim.[118]

Glucosamine and Chondroitin Sulfate

Many equine nutraceuticals contain a combination of GLN and CS. Synergistic effects have been suggested in a rabbit instability model in which the combination was more efficacious in delaying cartilage lesions than with either agent alone.[91] It has also been shown that the combination improves collagen synthesis in tenocytes and ligament cells, and it may be important for use in accessory joint structures.[90]

In vitro studies demonstrated no detrimental effects of GLN-CS on cartilage metabolism.[32] *In vitro* studies have also suggested that the combination inhibits proteolytic activity, potentially via retardation of the molecular and biochemical events associated with proinflammatory cytokines, including decreased PGE_2 and NO levels while also preventing GAG degradation.[19,32,113] These studies suggest that the GLN would be best for potentially inhibiting OA progression, whereas CS may be best for controlling symptomatic action of OA.

Oral administration of GLN-CS at doses greater than those recommended in horses is associated with a good safety profile, with no alterations in hematological or clotting profiles.[78] In an induced synovitis model in horses, a GLN-CS product showed no clinical detectable benefits.[158] However, there was a significant treatment effect in other synovitis studies, with one showing an anti-inflammatory effect (lower PGE_2 than controls).[35,150] In studies of clinical cases treated with this compound, the results are mixed but with more showing a generally favorable response. When an oral mixed supplement (GLN, CS, and methylsulfonylmethane [MSM]) was administered to veteran horses for 3 months, there was no evidence of any substantial improvement of locomotor performance in geriatric horses.[58] In other clinical studies, horses showed improved lameness, flexion, and stride length, while navicular horses showed significant improvement in soundness compared with placebo controls.[2,55,56] GLN-CS was also evaluated in horses with tarsal OA using gait analysis, demonstrating significant improvement in the left-to-right symmetry of peak vertical ground reaction forces and impulses, as well as tarsal range of motion and joint energy generation during stance.[21]

Hyaluronic Acid

Hyaluronic acid (HA) is a natural component of articular cartilage synthesized by synoviocytes, fibroblasts, and chondrocytes. It is responsible for the viscoelastic and lubricating properties of synovial fluid and plays an important physiologic role in nutrition of the articular cartilage.[64] Many oral formulations of HA have become available for horses. There are several anecdotal reports about the use of these products in horses, but scientific

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evidence of oral absorption, bioavailability, distribution, and controlled evaluation of the efficacy of oral HA products in the horse is lacking. One controlled double-blinded study using yearling Thoroughbreds that had arthroscopic surgery to remove osteochondritis dissecans (OCD) lesions from the tarsus demonstrated that the mean effusion score for the treated horses (100 mg orally once daily) was significantly lower than for the placebo group.[11]

Methylsulfonylmethane

MSM is a normal oxidative metabolite product of industrial-grade dimethyl sulfoxide (DMSO) that is an odorless and tasteless organic form of sulfur. It is naturally found in small amounts in fruit, alfalfa, and corn and is very soluble in water.[122,147] It can be found as a product by itself or in combination with GLN and/or CS.[58] It has been used as a nutraceutical because of its analgesic, anti-inflammatory, and antioxidant properties.[77,114,149] There is little known about the pharmacokinetics of MSM. Similar to DMSO, MSM has been suggested for the management of musculoskeletal pain as well as OA. It has been suggested that MSM may relieve muscle discomfort by decreasing the nerve impulses via cholinesterase inhibition and subsequently reducing muscle spasm.[42]

Very little is known about oral administration of MSM safety and toxicity. In a study in rats, MSM given for more than 90 days did not cause any adverse effects nor increase mortality.[61] Few studies have been published to support the use of MSM for managing OA in horses. Horses receiving oral MSM and vitamins demonstrated that MSM could exert some protective effect on oxidative and inflammatory exercise-induced injury related to jumping.[95] In another uncontrolled, non-peer-reviewed study, MSM was associated with improved performance in Standardbred racehorses in training.[123]

Avocado and Soybean Unsaponifiable Extracts

The unsaponifiable portions of avocado and soybean oils are extracted via hydrolysis to make up fractions of one-third avocado oil and two-thirds soybean oil. It appears that this mixture has synergistic properties; however, the active ingredient is still unknown.[93] *In vitro* studies have suggested that avocado and soybean unsaponifiable (ASU) extracts may have a positive effect on both the inflammatory cascade and structural components of the cartilage matrix. Studies have demonstrated that the extracts reduce MMPs, cytokines, nitric oxide, and PGE_2 while increasing growth factors and aggrecan production.[13,57,153] One controlled study that used horses with induced OA in the middle carpal joint failed to demonstrate any significant clinical effects.[72] However, the study demonstrated that ASU displays disease-modifying capabilities because of a significant decrease in intimal hyperplasia and histologic cartilage disease score along with an increase in articular cartilage GAG synthesis in joints with OA.

ASU has been combined with GLN-CS in some products, which may help to decrease some of the symptomatic signs. When combined with α-lipoic acid, an *in vitro* study demonstrated that ASU was better able to inhibit PGE_2 production from chondrocytes than either administered alone.[49]

Fatty Acids

Polyunsaturated fatty acids (PUFAs) are essential fatty acids that are found in fish and plants. Essential fatty acids are components of cell membranes, involved in lipid transport, and serve as precursors to the eicosanoid hormone family, which regulates inflammatory processes.[51] The two principle essential fatty acids are linoleic acid and α-linolenic acid. In the body they are desaturated and elongated to produce analogs of arachidonic acid (*N*-6 fatty acid) called eicosapentaenoic acid (EPA omega-3 fatty acids) and docosahexaenoic acid (DHA omega-3 fatty acids).

In horses, it was reported that the source of essential fatty acids affects the concentration of EPA and DHA in serum.[110] When horses received fish oil in the diet, there was an increase in the concentration of EPA and DHA in their serum, compared with horses that received corn oil. The anti-inflammatory effects of fatty acids have been attributed to stabilization of cell membranes, inhibition of the formation of inflammatory mediators, and protection against oxidation.[15,29,52] Studies have demonstrated that *n*–3 fatty acid supplementation can reduce or inhibit the inflammatory and matrix-degradative response elicited by chondrocytes during OA progression.[28,29] An *in vitro* study using an LPS challenge model in equine synovial explants demonstrated that pretreatment with α-linolenic acid significantly decreased PGE_2 production induced by LPS, reducing the inflammatory response.[107] An *in vivo* study of horses examined the effects of *n*–3 long-chain PUFA (LCPUFA) effects on LPS synovitis after 3 months of supplementation. Aggrecanase 1 concentrations were lower than controls, but there was no effect on PGE_2 or MMP activity in the synovial fluid compared with controls.[126] The clinical usefulness in the treatment of joint disease is still not entirely clear due to the uncertainty of which components of the oils might be effective.

Cetyl myristoleate (CM) is another fatty acid that is used in equine nutraceuticals. It is an ester, omega-5 fatty acid that may act by inhibiting the 5-lipooxygenase pathway,[66] which is responsible for the metabolism of leukotrienes (potent inflammatory mediators from the arachidonic acid cascade). One equine product demonstrated lower lameness scores compared with placebo horses in a double-blinded OA clinical trial.[73] However, there are no published reports on safety, absorption, and metabolism of CM in horses.

Collagen Hydrolysate

Collagen hydrolysate (CH) is a food ingredient that has been used in humans to improve joint comfort and function.[106] CH is derived from bovine or porcine skin and bones. Orally administered CH has been shown to be absorbed intestinally and to accumulate in cartilage.[164] In contrast with other nutraceuticals, no direct analgesic and anti-inflammatory effects have been found after using CH.[9] The theory behind CH is that it provides amino acids specific to the collagen network, playing an important role in the structure and function of cartilage by directly stimulating chondrocytes to synthesize collagenous matrix. Several studies in humans have indicated that the use of CH in people with OA is safe and provides improvement in some measures of joint

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pain and function.[7,9] A study using an experimental synovitis model in horses demonstrated anti-inflammatory properties (lower PGE_2 compared with controls) of collagen hydrolysate.[150] There are no published reports on the safety, absorption, metabolism, or clinical use of CH in the horse.

Another collagen product that has been used as a nutraceutical is glycosylated undenatured type-II collagen. A study using this product in horses diagnosed with moderate OA revealed a significant reduction in arthritic pain. This product was determined to be twice as effective compared with a glucosamine plus chondroitin formulation used in the same study. Results and conclusions of this study have to be considered with caution because the improvement was based on the response to flexion.[53]

Resveratrol

Resveratrol is a non-flavonoid, polyphenolic compound found in some fruits (such as grapes) and plants. It has been shown to decrease interleukin-1 synthesis via inhibition of nuclear factor-κB, downregulating COX-2 pathways, and scavenging of reactive oxygen species.[156] Cartilage does not have any blood vessels incorporated within it, and as such chondrocyte activity can be quite susceptible to low oxygen and pH levels. These levels can be further reduced in the face of OA due to mechanical stress, inflammatory mediators, and enzymatic activity causing chondrocytes to produce reactive oxygen species.[22] Resveratrol has been shown in experimental models of OA to protect cartilage due to balancing the oxidation–reduction (redox) reactions.[41,155] In an equine *in vitro* study, resveratrol partially restored mitochondrial membrane potential and reactive oxygen species levels resulting in prevention of GAG release and cell death of cultured chondrocytes.[22] Promise has been shown for use in lameness. Watts et al. performed a randomized, blinded, placebo-controlled clinical trial, where resveratrol was given to horses for 4 months after having their distal tarsal joints injected with corticosteroids.[156] A significantly greater percentage of riders reported that their horse's performance was better in the resveratrol group at 2 (95% versus 70%) and 4 (86% versus 50%) months compared with placebo. This was backed up by an objective inertial lameness exam, but not subjective lameness exam at 4 months.[156]

Vitamins, Minerals, Trace Elements, and Herbs

Many equine nutraceuticals contain vitamins, minerals, trace elements, and herbal ingredients in an attempt to supply elements that are necessary for the maintenance of normal cartilage metabolism as well as provide protection against reactive oxygen species.[142]

Vitamin C has been shown to stimulate collagen and aggrecan synthesis.[127] In addition, vitamin C is a free-radical scavenger that potentially provides anti-arthritic effects by protecting chondrocytes from damage by prooxidants.[92] α-Tocopherol (vitamin E) may have anti-inflammatory effects in the joint, in part by protecting chondrocytes from damage by reactive oxygen species.[92] In addition, vitamin E can enhance chondrocyte growth and increase the synthesis of proteoglycan.[50] Beta-carotene (provitamin A), selenium, zinc, manganese, niacinamide, and bioflavonoids all have potential antioxidant effects.[92] Many plants, parts of plants, or plant-derived substances have anecdotally been suggested to have antioxidant and/or anti-inflammatory properties,[161] with one *in vitro* study demonstrating that a polyherbal formulation was a significant source of antioxidants.[18] Some pharmaceuticals have "natural" plants as one or more of their components. Although some of these may have clinical merit, there are numerous products available, and none have been proven effective in the horse.[161]

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THERAPEUTIC TRIMMING AND SHOEING

Andrew H. Parks

INTRODUCTION

The terms "corrective shoeing" and "therapeutic shoeing" are frequently used interchangeably, and to make the differentiation yet more confusing, they mean different things to different people. Literally, corrective implies changing a situation to prevent something harmful, whereas therapeutic suggests a measure designed to heal a pathological process. For the purposes of this discussion, they will be considered synonymous, and the term therapeutic shoeing will be used.

Most horses requiring therapeutic shoeing present with either lameness, including poor performance, or undesirable foot/distal limb conformation/balance as the primary problem.

Ideally, therapeutic shoeing would follow a logical sequence of thought. A thorough understanding of foot function would determine one of two things: How foot conformation or imbalance has predisposed to or caused a disease process to occur in the foot, and/or how a disease process in the foot has altered foot function. From this it should be possible to deduce what principles need to be employed to counteract the causes or effects of the problem and devise a practical strategy to implement those principles.

There are obvious limitations at all these steps. While sometimes a diagnosis can be definitive, there are other occasions when the most specific diagnosis a clinician can make is heel pain. Although significantly improved over the last 10–20 years, our understanding of foot function is still quite incomplete, and therefore, the clinician must often pick the most rational explanation.

Most disease processes of the distal limb are traumatic in origin; more specifically, they are the result of repetitive stress resulting from normal athletic activity. The stresses on the distal limb vary with the phase of the stride.[26] The impact phase of the stride is separated into two overlapping collisions. The primary collision occurs as the foot contacts the ground and is associated with high accelerations but low mass. The secondary impact phase of the stride occurs as the mass of the body collides with the foot that is then firmly planted on the ground, but by this phase of the stride, the shockwaves are diminishing. During the support phase of the stride, the accelerations have ceased, but the overall load increases to a maximum at approximately mid-stride, after which the load diminishes.[26] The breakover phase of the stride is also associated with accelerations as the foot rotates. The distal limb has sophisticated mechanisms to dampen shockwaves and spread out the duration of loading and unloading, but extreme exercise, or changes to the foot or ground surface that enhance the accelerations within the distal limb or shorten the duration of loading, increase the likelihood of injury. Both shockwaves and large loads associated with weight-bearing are potentially injurious. In addition, attaching steel shoes with nails is known to increase the magnitude and frequency of impact shockwaves.[3,8] Therefore, once the principle abnormality is determined, the goals of therapeutic shoeing are almost always designed to strategically address the way structures in the distal limb are stressed to prevent, palliate, or heal injury to the structures that are at risk or diseased. Consequently, it is important to consider the way the structures of the distal limb are stressed during weight-bearing and locomotion.

The hoof capsule normally endures stress under tension or compression, depending on circumstances. These stresses may be longitudinal, radial, or circumferential and likely a complex combination of these. Ligaments and tendons are built primarily to resist tensile forces, cartilage best resists compression, and bones can withstand compression, tension, bending, or torsion to different degrees. The stresses associated with bending and torsional forces are much more dangerous to the bones of the distal limb than are those of compression, in which the bones are primarily loaded. Therefore, injury prevention is aimed at normalizing the stresses in the distal limb, and treatment is designed to protect a structure from stress as it heals.

The definition of ideal conformation is elusive. However, deviation from an optimal conformation is likely to lower the threshold at which injury related to the stresses of locomotion and weight-bearing is likely to occur. The hoof capsule, as part of the integument, differs from the musculoskeletal structures of the distal limb because it constantly grows and wears away and because it has remarkable viscoelastic properties. In response to prolonged application of abnormal stresses, the hoof capsule becomes distorted, and prolonged altered load bearing changes both wear and growth patterns.

EXAMINATION OF THE LIMB FOR THERAPEUTIC SHOEING

Examination of the horse and its limbs should identify both predisposing factors and disease. When the disease process is identified, a specific strategy may be formulated that includes a combination of therapeutic shoeing, medication, and/or surgery. Frequently, the clinician is expected to develop a strategy to treat a lameness originating from the digit that cannot be ascribed to a particular structure or process, compensate for a conformational abnormality, correct an apparent imbalance in the foot, or a combination of the above. This necessitates careful examination of the morphology of the foot, the relationship between the foot and the rest of the limb, and the way the foot interacts with the ground. Radiography is a useful adjunct to assess the relationship between the phalanges and the relationship between the phalanges and the hoof capsule.

Examination of the hoof capsule morphology indicates where the wall is unduly stressed, though care must be taken not to overemphasize any one characteristic,

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but rather correlate all findings. The hoof capsule should be examined from all sides with the foot on the ground, and from the solar perspective with the foot off the ground. With the foot on the ground, the following findings suggest too much stress in the wall in one portion of the foot: compression of growth ring (Figure 8.1), flares at the quarter or toe, underrunning of the heels, and proximal and/or abaxial displacement of the coronary band. With the foot held up, the ground surface should be examined for approximate mediolateral symmetry, including position of the heels and irregularities in the outline of the capsule. Additionally, the ground surface should be evaluated for appropriate width in relation to length and position of the widest point of the foot in relation to the center of the ground surface (see Chapter 11).

The phases of the stride are best observed at a walk. The most common pattern of landing at a walk or trot is either lateral heel/quarter first or flat, i.e. both sides simultaneously. Toe-first landing and medial-first landing are both suggestive of an abnormality in gait when a horse is moving in a straight line and should also prompt investigation.

Lateromedial and dorsopalmar radiographs are required to assess conformation and balance of the distal limb. For these purposes, the center of the X-ray beam should be centered approximately 1.5 cm above the ground surface of the foot. For the lateromedial radiographs, the X-ray beam should be centered midway between the toe and heels, and for the dorsopalmar radiographs, the beam should be centered in the middle of the foot. In a foot with good conformation and balance, the dorsal wall is approximately parallel to the dorsal surface of the distal phalanx, the sole depth is appropriate for the size of horse (variable from 10 to 16 mm though optimally greater than 15),[11,16,19] the center of rotation of the distal interphalangeal joint is slightly palmar to the center of the weight-bearing surface of the foot, and the average angle that the solar margin of the distal phalanx makes with the ground is 6°.[9,22]

Dorsopalmar radiographs must be taken with the metacarpus vertical and aligned with the median plane of the foot, which may not be the same as the median plane of the more proximal limb or the trunk. Both feet should be weight-bearing because elevating the contralateral limb induces collateromotion and rotation within the digit.[7] There should be no forced rotation in the limb. The most important observations are the angle of the articular surface of the distal phalanx to the ground and the symmetry of the articulations. The height of the coronet should be correlated with the position of the distal phalanx and any flares identified.

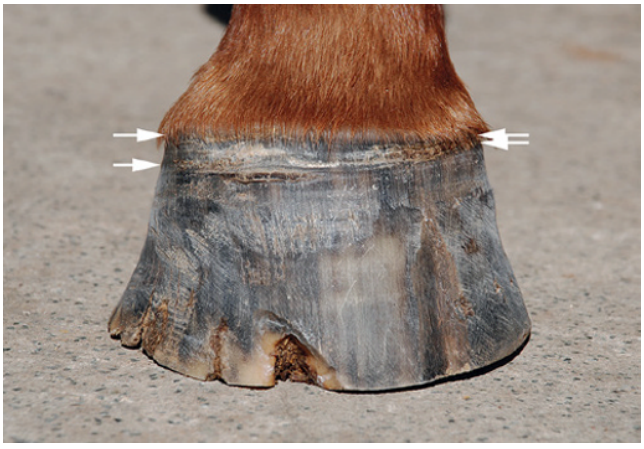


Figure 8.1. Photograph of a horse with growth rings that diverge markedly from medial to lateral (arrows). This horse has medial distal displacement of the distal phalanx.

THE TRIM

The foundation for any corrective shoeing is the trim. If the trim is not appropriate, whatever shoe is used will be less than optimal. The ideal shape of the foot and basic technique of trimming is well described in Chapter 11. In short, there are just three basic variables that can be altered: depth of sole, angle of the dorsal wall, and mediolateral symmetry. The length of the wall at the toe is predicated on the depth of the sole; the wall is either level or slightly longer than the adjacent sole. The length of the heels is then predicated on the length of toe and the angle of the foot-pastern axis. Distortions in the hoof capsule may necessitate additional measures. Likewise, application of techniques described below may require variation from the basic pattern.

SHOEING

There are only so many basic principles that can be implemented to improve the function of the equine foot, and these include increasing shock absorption, moving the center of pressure, easing movement about the distal joints in the limb, altering the distribution of force, changing traction on the ground surface of the shoe, and altering the motion of the limb during the flight phase of the stride (Table 8.4). However, there is a wide range of tools that can be applied to achieve these principles, and the combinations thereof are almost infinite. Some of these modifications to the shoe are covered in Chapter 11. Unfortunately, all too often the focus is on the method rather than underlying principles.

Shock Absorption

High-frequency, high-energy shockwaves are generated during the impact phase of the stride and to a lesser extent during the breakover phase. They occur in all planes, and the magnitude and frequency of these waves are affected by the structure of the distal limb, the presence of shoes and pads, and the nature of the ground surface.[2,3,18,21] Numerous manipulations of the foot influence – potentially influence – the magnitude of shockwaves, including the application of a shoe, the method of shoe attachment, the material of the shoe, pads interposed between the shoe and hoof, traction devices on the shoe, and the surface of the ground.

Usually, it appears that most interventions that involve shoeing increase the shockwaves associated with impact. Therefore, the natural damping mechanisms within the digit almost always function better in a healthy horse if it is barefooted. However, conditions of the working environment frequently necessitate shoeing. Most research has compared attachment of a

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Table 8.4. Principles for therapeutic trimming and shoeing.

Modification	Goal of modification	Examples of how to achieve goal
Move the center of pressure	Move the ground reaction force in relation to the center of the foot/center of rotation of the distal interphalangeal joint to move the load of weight-bearing from one side of the foot to the other	Wedges, extensions; alter width of web unevenly, either medial vs. lateral or dorsal vs. palmar
Shock absorption	Dampen vibrations associated with deceleration and acceleration	Aluminum shoes instead of steel, synthetic polymer shoes, viscoelastic pads
Ease movement about the distal interphalangeal joint	Shorten arm of extensor moment about the distal interphalangeal joint at breakover	Roll the toe, rocker the toe, square the toe, set the shoe back
Change distribution of force	Increase ground contact of part or all of sole, bars, and frog to reduce load on wall	Heart-bar shoes, custom pad (e.g. heart-bar pad), pour-in pads
Motion of limb during flight	Prevent interference or improve esthetics of gait	Forging: Speed up breakover of forelimb and retard breakover of hind limb Brushing in hind limbs: Widen gait by squaring toe, adding traction or trailer to lateral branch, ± lowering medial wall
Traction	Increase traction to reduce slippage; decrease traction to reduce horizontal deceleration	High traction shoes: rim shoes, calks, and grabs Low traction shoes: sliding plates, half-rounds, flat shoes

steel shoe with nails to the barefoot condition and the horse exercised on a firm, flat surface. Under these circumstances, the steel shoe reduces the natural damping mechanisms and reduces – but does not abolish – foot expansion.[6,8] Research also shows that aluminum and synthetic shoes may impede the natural damping mechanisms less than steel.[1,3]

The effect of rim pads inserted between the shoe and ground surface of the foot vary, depending on the material they are made from. Rigid pads and those that become compressed with use are unlikely to be of much benefit, but pads that undergo viscoelastic compression and relaxation with the phases of the stride may offer significant benefit. Unfortunately, there is virtually no information about commercially available pads to know which are likely to provide the greatest benefit. Similarly, there is a wide range of synthetic shoes available, some of which dampen impact vibrations, whereas others do not show a detectable improvement.[1,3]

The nature of the ground surface is also important in the interaction between the shoe and the ground in both damping impact shockwaves and loading and unloading the osseous and tendinous structures of the limb.[21,26] Usually, the surface is dictated by the nature of the work. Numerous devices such as calks, toe grabs, and modifications to the shoe profile (such as rims) are used to increase traction. Wide-web flat shoes or half-round shoes may decrease traction. Too much traction increases the horizontal impact vibrations, and too little predisposes to slipping and potential ligamentous injury. There are few data to assist a clinician in the choice of these devices to optimize traction, except to implicate the use of toe grabs in causing musculoskeletal injuries in racehorses.[14] Therefore, their application is largely based on individual experience. It is wise to err on the side of caution and use creases or low rims if possible, and if calks or grabs are needed, to use the lowest, broadest devices applicable.

Weight-Bearing and the Center of Pressure

The center of pressure determines the relative load bearing by the medial and lateral and dorsal and palmar halves of the foot, which in turn determines the loads and subsequent compressive and tensile stresses on the different structures of the foot. At rest, the center of pressure is at or near the center of the ground surface of the foot. The center of pressure changes during the course of the stride; it can be most simply described as moving rapidly from the point of contact to the point at or near the middle of the ground surface of the foot for the majority of the stride, until breakover, when it moves toward the toe.

The center of pressure can be moved in numerous ways. The simplest that works on all surfaces is to increase the relative length of one side of the foot to the other, which moves the center of pressure to the elevated side.[28] Increasing the length of the heel or toe moves the center of pressure toward or away from the center of rotation of the distal interphalangeal joint (Figure 8.2). In doing so, it shortens or lengthens the arm of the extensor moment about the distal interphalangeal joint.

Other manipulations to move the center of pressure without altering the relative lengths of one side of the foot are based on increasing or decreasing the resistance of the foot to descend into the surface of a yielding substrate. Extension of the shoe outside of the normal perimeter of the foot increases the length of the lever arm on that side. This increases the leverage that the substrate can exert about the center of rotation; for example, an egg-bar shoe moves the center of pressure away from the toe.[20] A similar effect can be achieved by altering the width of the shoe at any point about the perimeter in relation to the rest of the shoe. A decrease in the width of the web increases the descent of the shoe into a yielding substrate at that point; an increase in web width decreases it. Moving the center of pressure in this

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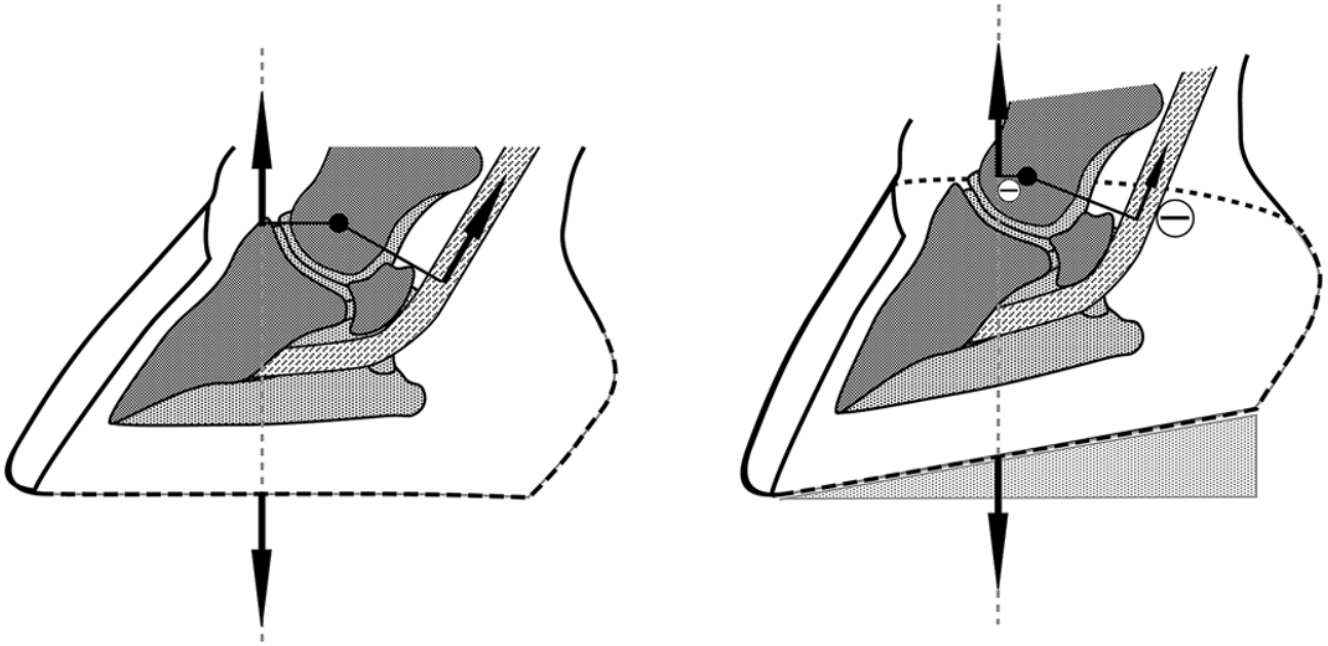


Figure 8.2. Schematic illustration demonstrating effect of heel wedge on moments about the distal interphalangeal joint. Elevating the heel shortens the moment arm of the extensor moment. As the flexor moment arm remains approximately the same, the force in the deep digital flexor tendon is decreased. The center of pressure remains approximately in the center of the foot. (Vectors represented by arrows are for illustrative purposes and do not represent real values.)

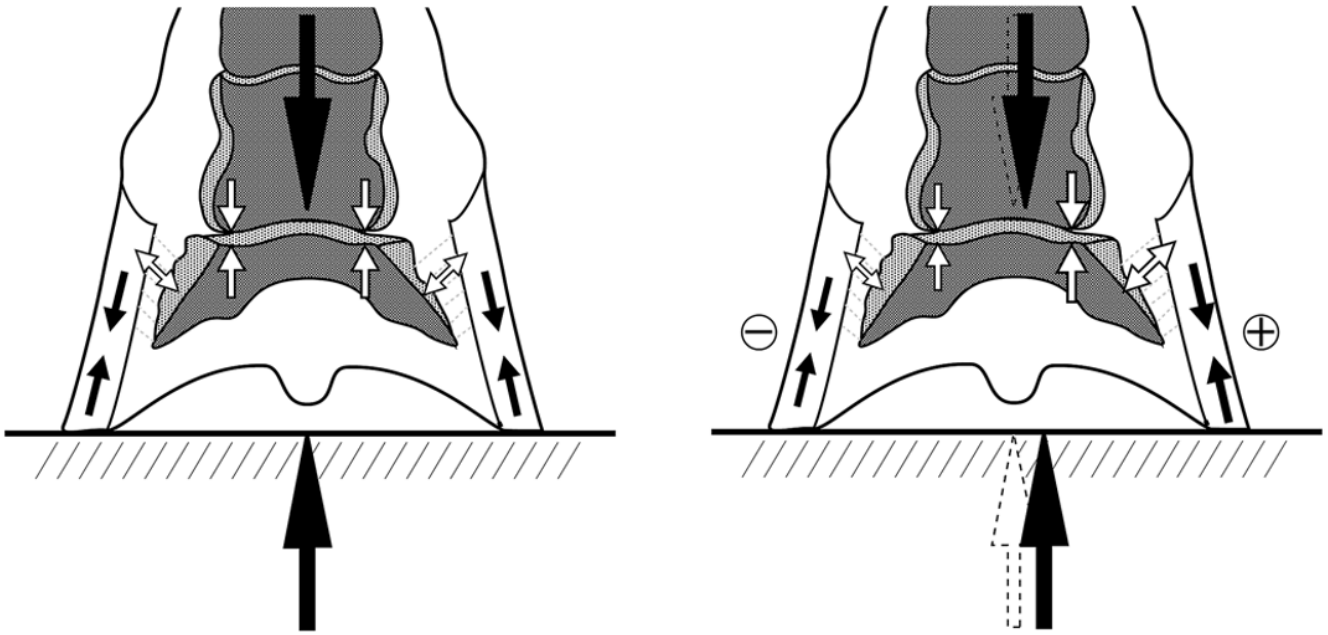


Figure 8.3. Schematic illustration demonstrating the effects of moving the center of pressure in the frontal plane on the structures in the foot. As more weight is borne by one side of the foot, the bones and joints become more compressed, the tension in the collateral ligament is reduced, and the tension in the lamellae is increased. These changes are reversed in the opposite side of the foot. (Vectors represented by arrows are for illustrative purposes and do not represent real values.)

manner to one side of the foot decreases the stress in the ipsilateral collateral ligament and the contralateral lamellae and articular surfaces (Figure 8.3). Moving the center of pressure toward the heels with a wedge or any shoe that functions as a wedge reduces tensile stress in the deep digital flexor tendon, compressive stress on the navicular bone, and is thought to decrease the tensile stress in the dorsal lamellae (Figure 8.4).

Each of these manipulations has a concurrent side effect. The manner in which horses place their foot on the ground suggests that proprioceptive feedback, rather than the ground surface of the hoof, controls the position

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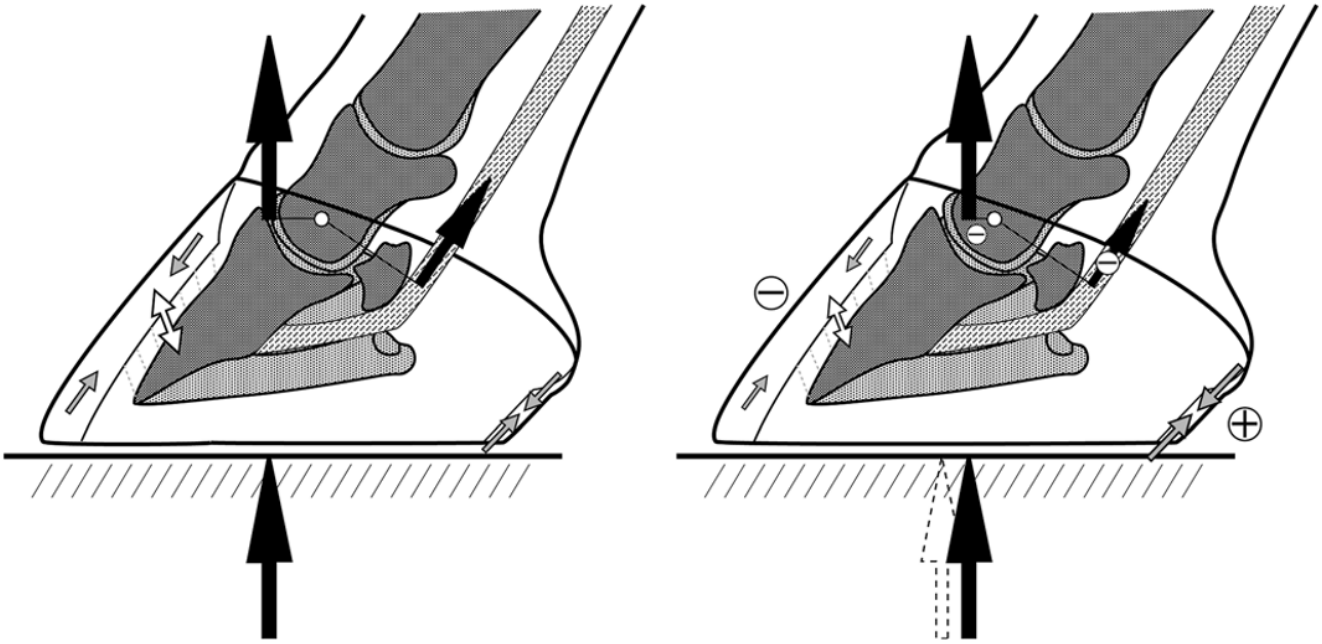


Figure 8.4. Schematic illustration demonstrating the effect of moving the center of pressure in a palmar direction. The arm of the extensor moment becomes shorter, and because the arm of the flexor moment remains approximately the same, the force in the deep digital flexor tendon is decreased. (Vectors represented by arrows are for illustrative purposes and do not represent real values.)

of the distal phalanx.[5] Therefore, extending the effective border of the hoof distally at any point around its circumference increases the tendency that the foot will land on the lengthened wall first. Extensions on the medial side of the foot of a horse should be used with caution because of the risk of interference. Similarly, toe extensions should be used with caution because they increase the propensity of a horse to trip. Lateral extensions may be used without such obvious side effects but are likely to alter flight of the foot as the limb is protracted due to increased air resistance, the magnitude, and significance of which have not been determined. Changing the width of the web of one branch of the shoe causes the foot to become asymmetrically weighted, changing the inertia that must be overcome as the foot accelerates away from the ground at breakover and decelerates at impact. The significance of this effect has not been investigated.

Ease of Movement

Clinicians have long thought that making the breakover point more palmar improves the function of the foot and relieves mild lameness, particularly that associated with palmar heel pain. However, several studies have examined the effect of changing breakover in this manner and have been unable to demonstrate a decrease in the duration of the breakover or a change in the flight of the foot,[10,17] though it may cause the foot to breakover slightly earlier. Additionally, it does not change the peak moment about the distal interphalangeal joint or the peak force on the navicular bone, even though it decreases the length of the moment arm of the ground reaction force about that the distal interphalangeal joint at breakover (Figure 8.5).[10]

The most likely explanation for the improvement seen by clinicians is that the foot breaks over (unrolls) more smoothly, thereby decreasing the abruptness in changes in stress in the underlying tissues.[13] To a lesser extent the medial and lateral abaxial branches of the shoe can be beveled to improve the ease of motion as a horse is turning. While this has not been explicitly studied, it is known that a turning horse loads the side of the hoof on the inside of the turn.[25] Therefore, it is rational to anticipate that rolling or beveling the abaxial margins of the shoe smoothes out the changes in stress in the underlying tissues on the ipsilateral side of the foot.

Distribution of Force

The main rationale for distributing part of the load of weight-bearing over more of the sole and frog is to reduce load bearing by the lamellae and wall, which assumes that most of the load is normally born by the wall. This is frequently done by recruiting the entire ground surface in horses with laminitis. In those with underrun heels, reducing weight-bearing by the heels is attempted by increasing weight-bearing by the frog, bars, and angles of the sole.

However successful these manipulations appear clinically, the mechanical effectiveness of this manipulation is uncertain because the role of the sole and wall in weight-bearing is poorly understood. It has been clearly demonstrated that when a horse that has been at pasture barefoot stands on a firm surface, it bears weight in a "4-point" pattern[15], that is, on its heels and at the toe-quarter junctions. If the same horse is housed on concrete, the wear of the ground surface of its foot causes it to bear weight around more of the perimeter of its foot and frog. If the same horse is made to stand on sand, the weight is distributed over a much greater area, which is no longer distributed around the periphery of the foot but over the sole in a band running from quarter to

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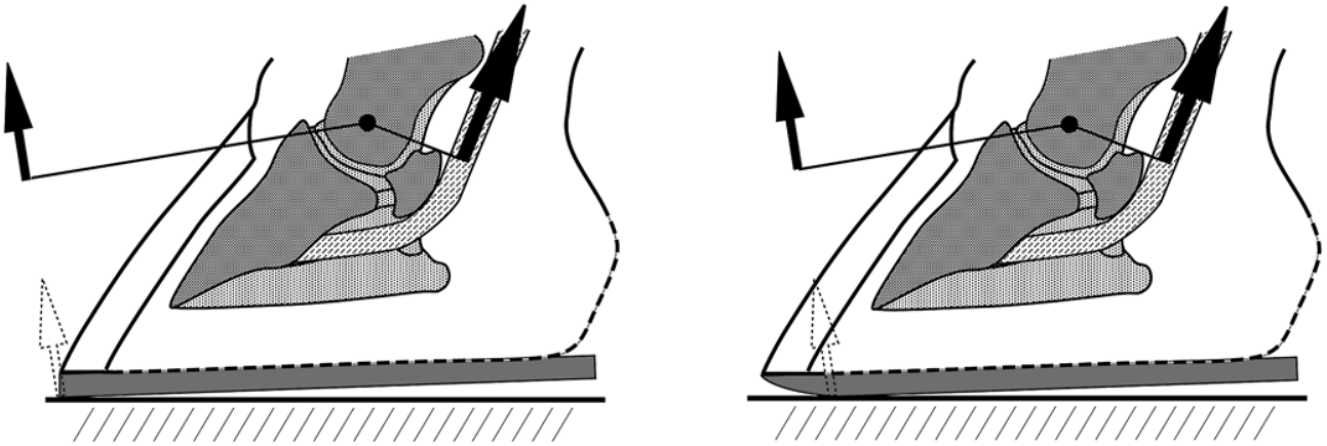


Figure 8.5. Schematic illustration demonstrating the effect of moving the point of breakover in a palmar direction. At the moment the heel leaves the ground, the arm of the extensor moment is shorter in shoes with rolled toes compared with shoes without rolled toes. (Vectors represented by arrows are for illustrative purposes and do not represent real values.)

quarter.[15] It has also been demonstrated that filling the space between the sole and the branches of the shoe with polyurethane (a pour-in pad) distributes the forces associated with weight-bearing broadly across the bottom of the foot when standing on sand though not when standing on a firm rubber surface.[12]

What is much less clear is how the weight is transmitted from the hoof capsule to the distal phalanx. Intuitively, it would seem that when a horse is bearing weight on a flat, firm surface and only the wall is in contact with the ground, all of the load is transferred through the wall to the distal phalanx through the lamellae and that the sole has little role in weight-bearing.

What then is the manner of load transmission when the horse is standing on a deformable surface such as sand or turf? Intuition might suggest that now weight is directly transmitted from the sole to the distal phalanx. However, evidence from finite element analysis suggests that this is not the case.[24] Rather, evidence suggests that the load is transmitted from the sole to the wall and presumably then to the lamellae. This model also suggests that expansion of the foot is caused by the wall at the quarters spreading, which causes the sole to spread and flatten. In doing so, the sole descends more than the distal phalanx. The implication behind the latter observation is that completely filling the space between the branches of the shoe, as with pour-in pads, may decrease the sole from descending, limiting expansion of the wall, and hence decreasing one of the hoof's mechanisms for dissipating shockwaves.

The distribution of force on the ground surface of the foot can be changed by increasing the area of contact with the ground during the stride, so that the force on the ground surface of the foot is more broadly distributed (Figure 8.6) or vice versa. This may be achieved by increasing the ground surface area of the web of the shoe so that it has greater contact with the ground and the foot. Alternatively, a variety of materials, such as polyurethane or silicone putty, may be placed inside the contour of the shoe to either partially or completely fill the space between the branches of the shoe, the ground surface of the foot, and the ground. A similar result may be achieved by using a heel plate in conjunction with a composite (Figure 8.7). A symmetrical increase in distribution of load about the center of pressure across the ground surface of the foot is unlikely to alter the center of pressure, but any asymmetrical redistribution of load bearing is likely to cause the center of pressure to shift. Additionally, there is a risk that making the ground surface of the foot completely flat reduces traction excessively, depending on the surface, and hence predisposing to ligamentous injuries related to slippage.

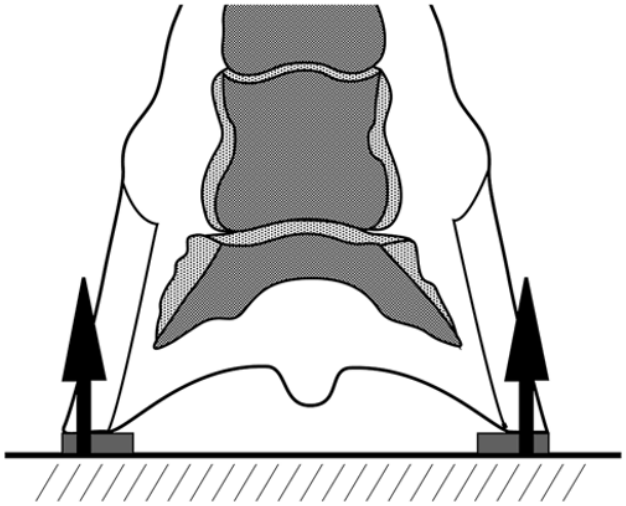
Clinical experience indicates that the lameness of some horses can be dramatically improved by distribution of weight across the sole, while in others it is made worse, and in yet others it seems to make little difference. The failures only serve to emphasize that our knowledge is insufficient to accurately apply scientific principles to the use of redistributing weight-bearing across the ground surface in a predicable manner. That said, the successes encourage further exploration to determine when and why it is successful.

Motion of Limb During Flight

The flight phase is the least investigated of the phases of the stride with respect to the effects of shoeing. Therefore, most modifications to change it are based on reasoned arguments, with all their flaws, and experience. The way the foot lands influences the position of the foot during the stance phase, and the position of the foot during the stance phase influences the way the foot leaves the ground. Thus, the patterns of the stance and flight phases are at least in part intertwined. Redirection of the path of flight of the foot not only is primarily directed at preventing interference injuries but also might be directed at improving the appearance of flight path for esthetic reasons. Any manipulation of the foot to change to flight path that causes the structures to move outside of their normal plane of motion or increases their range of motion potentially predisposes these structures to injury.

Interference between two limbs is a potential cause of injury. It may occur between two forelimbs, two hind limbs, and two ipsilateral limbs and in pacers between diagonal limbs. Interference is more common in shod

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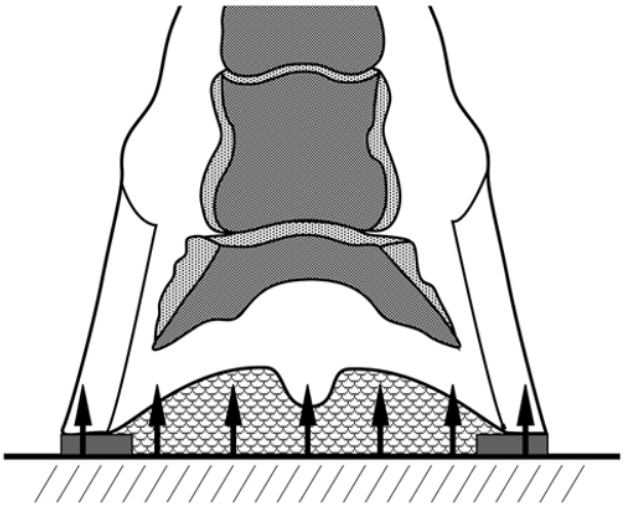


Figure 8.6. Schematic illustration demonstrating the effect of distribution of pressure. It is unknown how the redistribution of force on the ground surface of the foot affects the transmission of load to the distal phalanx. (Vectors represented by arrows are for illustrative purposes and do not represent real values.)



Figure 8.7. Shoe with a heel plate welded between the palmar aspects of the branches. *Source*: Courtesy of James Gilchrist.

horses, and it is thought that conformation, imbalance, and fatigue are the main precipitating factors.

Given the likely etiology of interference, the best starting point is to ensure the foot is balanced and use the lightest, appropriately sized shoe compatible with the level of work. Although the general strategy to avoid interference that occurs between ipsilateral limbs is to speed up breakover of the forelimb and slow down breakover of the hind limb, this practice is not well supported by scientific evidence. Changing the point of breakover on a forelimb has limited impact on the kinematics of breakover. Furthermore, while lengthening the toe and therefore moving the point of breakover dorsally in the hind limb does prolong breakover, the limb compensates during protraction to impact the ground at the same time as it would with a shorter toe.[4,17]

The general principle in treating interference between contralateral limbs is to widen the gait. This is generally achieved by a combination of squaring the toe, increasing traction laterally, and using a lateral trailer on the hind foot, with or without lowering the medial side of the foot. Again, evidence to substantiate these modifications of the shoe to achieve these goals is lacking, but they are long-standing traditional approaches. When these approaches do not work and interference persists, then the limb that is interfered with is protected with boots, and the shoe on the interfering limb may be safed.

PROTECTION AND SUPPORT

Protection and support are goals of therapeutic shoeing, and they deserve separate consideration since there is some overlap in their implementation. The term protection in relation to shoeing is usually used to indicate reduction or prevention of pressure on part of the ground surface of the foot. Therefore, protection is usually provided by eliminating pressure in a given area by redistributing the force associated with weight-bearing on the distal surface of the foot or placing a shielding device superficial to the area. This may involve using a wider web shoe, a strategically placed bar, or a treatment plate across the entire ground surface of the foot.

The use of the term support is more ambiguous. Support can be used in relation to maintaining an anatomical relationship; for example, after the deep digital flexor tendon has been severed, a heel extension may be used to maintain the foot flat on the ground. Alternatively, a structure can be considered supported when the stress in it is reduced; for example, in the case of a strained deep digital flexor tendon, elevating or extending the heels decreases the stress in the tendon. In these examples, one action meets both definitions of support; however, this is not always the case. It is common to hear that a modification to the shoe supports the heels, usually by extending or elevating them. In reality, extending or elevating the heels maintains the alignment of the foot-pastern axis and supports the deep digital flexor tendon but increases the load on the heels, thereby increasing the likelihood that they will deform and decrease their growth. Most actions that provide support involve movement of the center of pressure.

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CLINICAL APPLICATIONS OF PRINCIPLES

The specific diseases of the equine digit are covered in detail elsewhere in the text, but it is useful to briefly examine how the above principles might be applied to several common conditions. Because navicular syndrome is generally considered to be progressive, prolonged rest for a working horse is not usually an option. Therefore, therapeutic measures are primarily directed at palliation, and therapeutic shoeing is pivotal.

Moving the point of breakover in a palmar direction by aggressively rolling the toe smoothes out breakover. Increasing the ground contact in the palmar half of the foot, such as with a straight bar, heel plate (Figure 8.7), or egg-bar shoe, decreases the descent of the heel into a yielding substrate and thereby acts as a mild wedge. Therefore, these maneuvers move the center of pressure in a palmar direction to decrease the tension in the deep digital flexor tendon at rest and in the support phase of the stride.

To move the center of pressure in a palmar direction more aggressively, the heels may be raised by one of several means including a graduated shoe or a wedge pad. The compromise made in moving the center of pressure in a palmar direction is that the palmar hoof capsule will bear more load and will be more likely to become deformed. Therefore, the center of pressure should be moved as little as possible to produce the desired result.

There are several shoe designs in which the palmar half of the shoe is flat, but the design of the shoe incorporates a breakover point at or just dorsal to the midpoint of the foot, for example, the Tennessee navicular shoe (Figure 8.8). Presumably, this shoe acts as a flat shoe for the landing and support phases of the stride but as a wedge during breakover. It appears to mitigate some of the disadvantages of heel elevation.

Finally, impact vibrations can be potentially dampened by switching from a steel shoe to an aluminum or synthetic shoe, or by incorporating a viscoelastic pad.

Strains and rupture of the deep digital flexor tendon share features with navicular disease in that they are likely caused by repetitive excessive strain; therefore, similar measures should benefit both. However, the goal of treatment of a deep digital flexor tendon injury is to promote repair. The horse is rested, and reducing tension on the tendon at rest or in the support phase of the stride becomes the primary goal of shoeing. Consequently, the center of pressure is moved in a palmar direction more aggressively, usually by extending the shoe in a palmar direction and by elevating the heels, often using a custom forged patten shoe (Figure 8.9). Prolonged used of this type of shoe causes marked distortion of the hoof capsule by decreasing heel growth, causing the heels to underrun and contract, increasing growth at the toe, and causing the dorsal wall to assume a marked convex shape. Therefore, the degree of elevation must be reduced as rapidly as is compatible with healing of the tendon.

Laminitis is a complex disease that has many clinical manifestations. The goals of shoeing are to minimize additional injury and control pain in the early chronic stages of the disease and in later stages of the disease to encourage realignment of the hoof wall with the distal phalanx and normal hoof wall growth. Because the lamellae are stressed under tension, which contributes to the pain, the center of pressure should be moved to the opposite side of the foot. In a horse with rotation, this is commonly accomplished with heel extension or elevation to decrease stress in the dorsal lamellae (see Chapter 4). Additionally, because it is preferable to reduce weight on all the lamellae as much as possible, part of or the entire ground surface of the foot between the branches of the shoe may be recruited to bear weight (this is apparently used to good effect when standing an acutely laminitic horse on sand). However, caution must be taken to ensure that the shoe or packing material/pad does not put pressure on the sole where the solar margin of the distal phalanx has displaced distally. If the horse has sunk unilaterally, then the center of pressure should be moved to the contralateral side of the foot. This has been attempted by setting a shoe, either metal or wood, wide to act as an extension on the contralateral side of the foot. In horses that have complete loss of lamellar integrity and have distal displacement of the distal phalanx, there is little advantage to moving the center of pressure. In addition, spreading the load across the sole



Figure 8.8. KB navicular shoe, a variant of the Tennessee navicular shoe. *Source*: Manufactured by G.E. Forge and Tool, Inc., 959 Highland Way Grover Beach, CA 93433.



Figure 8.9. Patten shoe used to elevate and extend the heels in horses with severe deep digital flexor tendon injury.

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is problematic because in unloading the wall it loads the sole where the distal phalanx has displaced. Generally, once the distal phalanx has become more stable and the sole thickened, horses with distal displacement can be shod. Then a seated out wide-web shoe is used to protect the sole adjacent to the wall, and a composite such as silicone putty is used to recruit weight-bearing by the sole, bars, and frog. While this outline suggests logical approaches to protect the affected lamellae as they heal, more documented experience is needed.

Pedal osteitis is a syndrome associated with a flattened sole, bruising of the solar dermis, and osteopenia of the solar margin of the distal phalanx. Therefore, the principal goal of treatment is to protect the affected areas by limiting their contact with the ground. Wide-web shoes that have been seated out protect that area immediately adjacent to the wall, the principally affected area, and therefore are the most common shoe modification used. Pads that elevate the ground surface of the foot may also decrease the likelihood of ground contact. However, pads that cover the ground surface of the foot are of mixed benefit because while they may dissipate the energy of contact, they may actually increase the likelihood of contact; this particularly applies to pads of the pour-in variety. Therefore, the use of a pad must be decided on a case-by-case basis and careful choice of material.

Osteoarthritis of the interphalangeal joints is progressive, and pain can be exacerbated by concussion and extreme motion within the joint. Therefore, the goals of treatment should be to decrease concussion associated with impact and loading and decrease the abruptness in changes of stress associated with locomotion. This can be accomplished by using aluminum or synthetic polymer shoes instead of steel shoes, using viscoelastic rim pads, and ensuring that the shoe has limited traction to decrease the jarring associated with deceleration. Moving the breakover in a palmar direction and beveling the abaxial margins of the shoe minimize strain on the joint capsule and collateral ligaments. These may be achieved with a roller motion shoe (Figure 8.10), but other shoes, such as half-round shoes, can be modified to provide similar all-around benefits.

Ideal distal limb conformation is poorly defined and depends on the nature of athletic activity a horse performs. Nevertheless, some generalizations can be made about the various forms it takes. Most are related to mediolateral angulation within limb, rotation within the limb, and changes in relative length and angle of the pastern. If one of these is present, without clinical lameness, hoof wall distortion, or changes in foot flight, it is usually unadvisable to perform anything other than routine trimming and shoeing because almost all measures will have an unintended consequence. For example, it has been proposed that if the foot is lateral to the median plane of the metacarpus, a medial extension should be placed on the foot to bring the center of pressure under the metacarpus. The potential benefit is that the metacarpophalangeal joint is more evenly loaded.[27] Unfortunately, this also loads the medial structures of the foot that are under compression and the lateral structures that are under tension. At a minimum, the compression of the medial hoof capsule causes decreased hoof wall growth and flaring of the capsule at the quarters.

Similarly, it has been proposed that when the foot is further dorsal in relation to the metacarpus than "normal," the shoe is extended farther in a palmar direction to bring the center of pressure closer to the axis of the metacarpus to decrease the strain on the tendons.[23] However, the tendon that receives the greatest benefit is the deep digital flexor tendon, whereas the tendons most affected by the length/low angle of the pastern are the superficial flexor tendon and the suspensory apparatus. This strategy also places greater load on the heels, increasing the likelihood that they will deform. Nevertheless, it can be envisaged that there are occasions when these compromises are necessary. Indeed, in foals, both strategies are used. Abaxial extensions are used to treat angular and rotational deformities and palmar extensions to treat flexor tendon laxity. In part, these techniques are successful in foals because they are only needed temporarily and the growth plates have not fused.

In brief, the treatment of imbalance restores the shape of the hoof capsule, its relationship to the skeletal structures of the limb, and the pattern of landing. Because imbalance takes so many forms, there is no universal recipe, but there are some general guidelines. Imbalance of the hoof capsule is best treated by trimming rather than shoeing if possible. Imbalance may be a more general problem, i.e. when it affects the symmetry of the whole foot, either medial versus lateral or dorsal versus palmar. Alternatively, it may be a local phenomenon, such as occurs when there is proximal displacement of the coronary band immediately proximal to the ground surface of the heel buttress associated with a quarter crack.

The trimming is best guided by examining the hoof capsule for evidence of areas with increased stress, such as flares and compression of growth rings (Figure 8.1). If possible, the length of the wall should be trimmed in these areas. If there is not enough wall below the preferred plane of the sole to trim to correct it – for example, when the wall has moved proximally in relation to the distal phalanx or correction of a local imbalance leaves an uneven ground surface – then it is best to leave the hoof unshod. This encourages migration of the wall into a more normal position.



Figure 8.10. ACR Rolling Sport Shoe, a form of a roller motion shoe.

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Graduated shoes, pads, and attachment of shoes with composites can also be used to correct imbalance. However, placement of a shoe may also limit or prevent movement of the hoof capsule that contributes to restoring the desired shape of the foot. There are occasions when indicators of imbalance suggest conflicting actions; then it is advisable to go with the preponderance of the evidence and be prepared to use trial and error.

SUMMARY

The science of farriery is far from fully developed. It is important that clinicians understand the limited amount of experimental evidence that is available to support different trimming and shoeing techniques. For experiments to have value, they must be performed under very specific constraints. Extrapolation beyond these constraints increases the variability of expected results, and therefore, an apparently rational approach is not always successful. Consequently, a willingness to use trial and error greatly increases a clinician's chances of success; it also builds the clinician's body of knowledge. It is natural for any veterinarian or farrier to prefer a given technique based on experience because practice improves success, and it is difficult, if not impossible, to be adept at all techniques available. The goal should always be to find the simplest solution that meets the objectives. Finally, it is important to remember that whenever a change is made to a shoe, there is an unintended, though potentially anticipated, consequence.

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ACUPUNCTURE TREATMENT OF LIMB LAMENESS AND BACK PAIN

Kevin K. Haussler

INTRODUCTION

Acupuncture is the stimulation of specific anatomical landmarks with the goal of providing pain relief or normalizing physiologic function. The exact location of equine acupuncture points has been debated, and clear consensus on anatomic locations and naming systems is lacking.[14] Numerous studies have proposed unique anatomical or physical characteristics of acupuncture points, such as sites of palpable indentation or tenderness, decreased electrical resistance, increased free nerve endings, motor points, or neurovascular bundles. A Western perspective of acupuncture points is that most sites correspond to underlying nerves that produce specific local, autonomic, and central neurophysiologic effects.[19] Acupuncture has many reported effects via neurophysiologic, humoral, and bioelectric mechanisms. Needle insertion into the skin produces local tissue effects and afferent sensory stimulation of central neural pathways.

The most commonly reported mechanisms of action for acupuncture-induced analgesia include stimulation of endogenous opioids and peripheral electrical stimulation of large sensory afferent nerves that modulate nociceptive input in the dorsal horn of the spinal cord (i.e. gate control theory of pain) via release of neurotransmitters and neuromodulators.[18,25] However, there is no consistent relationship between the level of analgesia and plasma or cerebrospinal fluid concentrations of β-endorphins or cortisol. Neurologic connections between acupuncture points on the trunk and sympathetic pathways in the thoracolumbar spinal cord also provide possibilities of influencing internal organ function by stimulating paraspinal acupuncture points.[14] Recent neuroimaging studies using functional MRI to measure brain activity have revealed that acupuncture stimulation modulates the human central nervous system, including cerebral limbic/paralimbic and somatosensory cortex.[27]

De qi is a characteristic needling sensation as perceived by the patient and the sensation of needle grasp as perceived by the acupuncturist. During needle grasp, the acupuncturist feels pulling and increased resistance to further movement of the inserted needle, which is considered to be due to mechanical coupling between the needle and connective tissue.[6] A large number of acupuncture points have been reported to occur along intermuscular or intramuscular connective tissue planes. Evaluation of connective tissue responses to acupuncture stimulation has revealed quantitative differences between acupuncture and nonacupuncture points, which may provide a valid anatomical and physiological explanation for acupuncture meridians. Mechanical coupling between the acupuncture needle and connective tissue produces mechanical, neurologic, and chemical extracellular signaling, which may explain some of the local and remote and long-term effects of acupuncture.[7] This may provide one explanation why inserting a needle anywhere in the body can produce measurable physiological responses.[13] Further research is needed to fully explain the physiologic effects of acupuncture.

TECHNIQUES OF STIMULATION

Numerous forms of stimulation have been applied to acupuncture points. Different pain-controlling mechanism can be evoked by changing the type or intensity of acupuncture stimulation.[19] The most common methods of acupuncture stimulation include the insertion of solid needles (i.e. dry needling), electroacupuncture, and aquapuncture. Dry needle acupuncture uses disposable, 25- to 34-gauge, filiform, solid needles of varying lengths. Dry needle insertion causes immediate changes in neural or segmental interactions and local inflammatory responses.[19] Prolonged stimulation can induce humoral responses and activation of the opiate system, which creates generalized analgesia.

Electroacupuncture is the application of electrical stimulation to inserted needles, with the goal of providing more intense or focused neurologic stimulation and muscle relaxation (Figure 8.11). Electroacupuncture and dry needle acupuncture both provide cutaneous analgesia in horses, compared with controls.[18] However, electroacupuncture is more effective than needle acupuncture for activating the release of β-endorphins into the cerebrospinal fluid. Electroacupuncture produces effects according to the polarity, frequency, intensity, and duration of electrical stimulation, as well as the number and pairing of needles stimulated.[25] Low-frequency

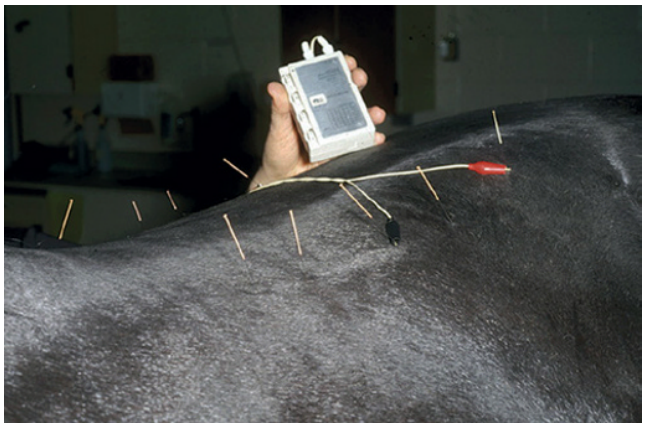


Figure 8.11. Electroacupuncture of the longissimus muscle near the thoracolumbar junction in a horse with back pain and muscle hypertonicity. *Source*: Courtesy of Dr. Gary Baxter.

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(less than 20 Hz), high-amplitude stimulation causes visible muscle contractions and produces generalized analgesia with slow, prolonged, and cumulative effects that are mediated by endorphins.[19] High-frequency (80–100 Hz), low-amplitude stimulation activates low-threshold skin and muscle receptors and produces non-opiate-derived analgesia that has a rapid onset and decline, is localized segmentally, and is not cumulative.

In a randomized control trial, four different electroacupuncture protocols were evaluated for antinociceptive effects on thermal stimulation of the pastern region.[25] Local acupuncture points and high-frequency (80–120 Hz) stimulation were more effective in blocking limb pain than the use of distant acupuncture points and low-frequency (20 Hz) stimulation.

Aquapuncture uses a 20- to 25-gauge, hypodermic needle to mechanically stimulate acupuncture points, followed by injection of various solutions in an effort to produce longer-lasting or combined pharmacological effects. Some of the more commonly injected substances include saline, vitamin B_{12}, and 2% iodine in peanut oil. Other substances include corticosteroids, Sarapin, Traumeel (a homeopathic remedy), vitamin B_1, 25% magnesium sulfate, 2% procaine, and 15% dextrose.[23] Injected volumes at each site vary from 0.5 to 5 mL (and up to 30 mL)/site, depending on the injected substance and desired response. Some practitioners believe that treatment effects last longer with injected substances, compared with dry needling. Other practitioners prefer dry needling with solid acupuncture needles due to the increased risk of breaking off hypodermic needles under the skin due to skin or fascial movement.

Additional forms of acupuncture stimulation include acupressure (i.e. digital pressure), implantation of gold beads or surgical staples for long-term stimulation, and laser stimulation, which uses low-level laser generation to stimulate superficial acupuncture points. Laser acupuncture provides the benefit of having no physical puncturing of the skin, but the initial cost of equipment is expensive. Anecdotal evidence experienced by the author suggests that low-level laser stimulation is effective for producing a temporary reduction in acute or severe back pain when affected horses do not tolerate acupuncture needle placement or forms of manual therapy. Dry needling, laser stimulation, and injection of saline into acupuncture points appear to be equally effective for treating horses with chronic back pain.[8]

GENERAL INDICATIONS FOR TREATMENT

Acupuncture case selection is often based on thorough physical examination and elimination of conditions that are judged to respond more effectively to conventional therapies.[11] Because of the lack of proven effective medical or surgical approaches for the treatment of chronic back pain in horses, acupuncture is often considered as a first treatment of choice.[11] If clinical signs of back pain persist after a few acupuncture treatments, then additional diagnostics and re-examination are indicated in an attempt to better localize and identify the source of pain and to prescribe a targeted treatment or a combination of therapies. Acupuncture is generally not sufficient by itself to manage overt lameness, unless the lameness is judged to be caused by muscle hypertonicity.[9] Acupuncture may be used in cases that have an incomplete or lack of response to traditional medical and surgical approaches or compensatory lameness associated with primary joint or limb pain.[11] The use of acupuncture or the combination of acupuncture with conventional care is believed to produce beneficial and lasting effects and provides an opportunity to expand a veterinarian's diagnostic and therapeutic armamentarium. Acupuncture combined with diagnostic joint blocks and intra-articular therapy is thought to provide a more comprehensive diagnostic and treatment approach to chronic lameness.[9,10]

Contraindications for acupuncture include acute injuries, fractures, severe cardiac disease, extremely nervous or exhausted patients, acute infections, and neoplasia. Patients with organic disease or overt musculoskeletal pathology need a complete diagnostic workup and consideration of appropriate Western treatments with documented efficacy and safety for the condition of interest.

LAMENESS

Acupuncture is reportedly useful for treating myofascial pain, osteoarthritis, stifle or hock pain, tendinitis, laminitis, and navicular disease, but there is a lack of controlled studies to validate efficacy.[21] Most evidence is in the form of clinical experience, case reports, and case series.[17] Numerous equine acupuncture case series on lameness are reported in the Chinese literature; however, poor scientific quality and lack of controls make the evidence difficult to interpret.[22] A review of several case series on nonspecific shoulder lameness, fetlock injuries, and laminitis report overall success rates ranging from 87% to 94% after 1–5 treatments, applied every 1–4 weeks.[22] However, the lack of details about case selection, diagnostic findings, objective outcome parameters, and nonuniform point selection and type of stimulation reduces the strength of the studies. A large number of horses across several disciplines were used to investigate the reported usefulness of acupuncture in the diagnosis of stifle disease in athletic horses.[12]

Only a few randomized, controlled trials have evaluated the effectiveness of acupuncture for limb pain or lameness. Cutaneous nociceptive thresholds to mechanical and heat stimuli during control and electroacupuncture trials in 23 horses showed significant cutaneous analgesia within 5 areas of the trunk, but not in the head or limbs.[1] Electroacupuncture has demonstrated variable effectiveness for reducing pain associated with either experimentally induced or naturally occurring lameness.[20,25] In a randomized controlled trial, electroacupuncture was used to treat horses with chronic laminitis ($n = 10$) and navicular disease ($n = 10$).[20] The degree of lameness was assessed by changes in subjective lameness scores, stride lengths, and ground reaction forces. Although 7 out of 10 horses with chronic laminitis improved clinically during the trial, there were no statistically significant differences between treatment and control groups. Six out of 10 horses with navicular disease improved, but there were no significant differences between treatment and control groups. Reasons for failing to demonstrate efficacy of acupuncture treatment

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include small sample size and inappropriate point selection, frequency, or intensity of stimulation. An additional randomized, blinded study using dry needle and electroacupuncture to treat palmar heel pain reported no significant differences between treatment and control groups with regard to lameness grade or hoof compression thresholds.[15] The authors reported limitations of a small sample size and the need for MR imaging to improve diagnostics for inclusion criteria. A case series of 12 horses with chronic laminitis showed positive results to two acupuncture treatments 1 week apart, based on inertial sensor-based lameness evaluation and subjective lameness scores.[3] Another prospective, single-blinded, crossover study to evaluate the effects of acupuncture treatment on gait parameters demonstrated that objectively assessed pelvic limb parameters could detect changes in gait that might be difficult to appreciate by visual assessment.[2] Overall, treated horses moved in a more symmetrical manner, which suggested a lesser degree of discomfort.

Electroacupuncture has also been evaluated in an experimentally induced lameness model using an instrumented shoe to apply transient localized sole pressure.[24] Electroacupuncture was able to partially reduce lameness scores, compared with bupivacaine or saline controls. Inconsistent changes in stride kinematics were noted, but there was no change in overall stride lengths. In another study using instrumented shoes to induce lameness in 6 horses, there was inconsistent evidence to suggest that pain, as measured by increased heart rates, was reduced after needle puncture to induce bleeding (hemoacupuncture) around the coronary band and dry needle placement between the heel bulbs.[4] For empirical treatment of laminitis, acupuncture points around the coronary band (*Ting* points) reportedly reduce local pain, and proximal thoracic limb points are used to address the sustained laminitic posture and subsequent muscle hypertonicity and pain associated with chronic laminitis (Figure 8.12).



Figure 8.12. Horse with bilateral chronic laminitis treated with acupuncture needles placed around the coronary band at *Ting* acupuncture points. *Source*: Courtesy of Dr. Tim Holt.

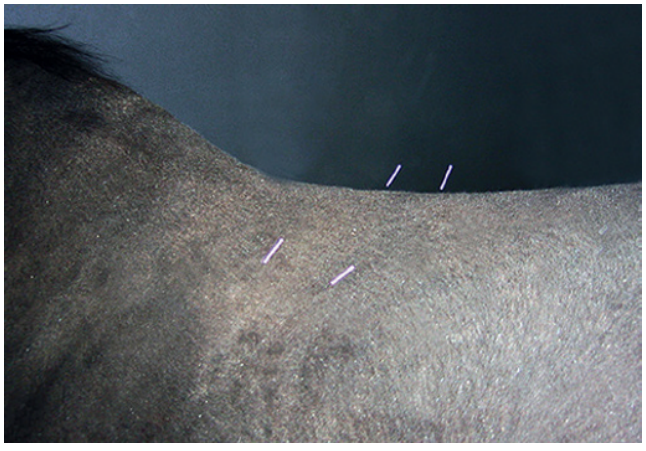


Figure 8.13. Horse presenting with pain localized to the saddle region treated with acupuncture needles placed along the dorsal midline adjacent to the spinous processes and within the epaxial musculature. *Source*: Courtesy of Dr. Tim Holt.

CHRONIC BACK PAIN

The majority of the equine acupuncture literature addressing musculoskeletal issues is focused on treatment of chronic back pain (Figure 8.13). In one review of acupuncture treatment for back pain, 11 different types of acupuncture stimulation have been described; however, some methods are quite unconventional and not used in Western acupuncture.[23] In acupuncture case series for treating back pain, success rates range from 66% to 100%; however, many studies have small sample sizes, and all studies lack objective outcome parameters.[22,23] In a case series of 200 horses treated with acupuncture, 73% had primary back problems or back problems combined with pelvic limb lameness.[11] Of the horses that received treatment for primary back pain, 58% (84 of 146) received acupuncture treatment alone. Another case series of 25 horses with back pain and poor performance reported that all horses returned to their previous level of competition following a combination of acupuncture treatment, changing saddle pads, and addressing poor saddle fit.[5] In a large case series (n = 350) reported over several successive studies, horses presenting with a chief compliant of poor performance, presumably due to primary back pain, were treated with different forms of acupuncture stimulation, and pain alleviation varied from 53% to 87%.[8] Out of the 350 horses, 75% were able to perform at an acceptable level after acupuncture treatment. Unfortunately, there were no control groups in these studies, so the natural course of back pain resolution or true effectiveness of acupuncture treatment could not be determined.[8]

In a randomized controlled study, both dry needle acupuncture and electroacupuncture of the trunk significantly increased cutaneous analgesia measured by thermal stimulation, compared with sham needle placement.[18] Another randomized, controlled study reported that electroacupuncture produced significant decreases in thoracolumbar pain scores after three treatments, which remained reduced 14 days after the last treatment, compared with oral phenylbutazone and control (i.e. oral

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saline) groups.[26] A third randomized, double-blind, controlled trial evaluated the effectiveness of electroacupuncture on chronic back pain using pressure algometry to assess mechanical nociceptive thresholds.[16] A series of electroacupuncture treatments over 15 days combined with rest was significantly more effective for increasing mechanical nociceptive thresholds (i.e. reducing pain) than sham electroacupuncture and rest.

ADVERSE EFFECTS

Adverse effects associated with acupuncture use in horses are largely unreported. Bleeding at the site of needle insertion is common and may actually be used as a treatment technique (i.e. hemoacupuncture) for certain conditions. In one equine study, approximately 20% of horses experience increased signs of back pain the day after saline aquapuncture treatment, which was alleviated by prescribed exercise.[8] Bent or retained needles are most commonly due to the use of excessive force to insert needles through thick skin, muscle spasms, or patient movement. Close patient monitoring is indicated, and repeat acupuncture treatment sessions may need to be revised by changing point selection or the method or intensity of stimulation. Equine acupuncture is considered a safe treatment modality for practitioners experienced in addressing pain associated with lameness or back problems.

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MANUAL THERAPY TECHNIQUES

Kevin K. Haussler

INTRODUCTION

Manual therapy is the application of the hands to the body with a therapeutic intent.[11] Chiropractic, osteopathy, physical, massage, and touch therapies are all considered forms of manual therapy techniques that have been developed for treatment of musculoskeletal disorders in humans and transferred for use in horses. Each technique has unique origins and different proposed biomechanical or physiological effects; however, all forms of manual therapy are characterized by applying variable gradations of manual force and degrees of soft tissue or articular displacement. The goal of all manual therapies is to influence reparative or healing processes within the neuromusculoskeletal system.

Therapeutic effects may be generalized to the entire body by inducing relaxation or altering behavior, regional effects may include alterations in pain perception or neuromuscular control, or effects may be localized to specific tissues and cellular responses.[15] The challenge is in selecting the most appropriate and effective form of manual therapy to produce the desired physiological effect within an individual patient, such as increasing joint range of motion, reducing pain, or promoting general body relaxation. Anecdotally, all forms of manual therapy have reported levels of effectiveness in humans and horses. Unfortunately, most claims are not supported by high levels of evidence such as randomized controlled trials or systematic reviews of the literature.

THERAPEUTIC TOUCH

The physical act of touching someone can induce physiologic responses and is often considered therapeutic.[5] In horses, touch therapies have been primarily developed and promoted by Linda Tellington-Jones in a collection of techniques named the Tellington Touch Equine Awareness Method (TTEAM) or Tellington TTouch.[25] Anecdotally, therapeutic touch is thought to improve behavior, performance, and well-being of horses and enhance the relationship between horse and rider, but no controlled studies support these claims. Similar touch therapy techniques have been used in foals at birth to assess the effects of touch or imprint training on behavioral reactions during selected handling procedures.[23] Conditioned foals are reported to be significantly less resistant to touching the front and hind limbs and picking up the hind feet at 3 months of age. More controlled studies are needed to determine the effectiveness of touch theories in managing behavioral or musculoskeletal issues in horses.

MASSAGE THERAPY

Massage therapy is the manipulation of the skin and underlying soft tissues either manually (e.g. by rubbing, kneading, or tapping) or with an instrument or machine (e.g. mechanical vibration). Massage techniques include many named methods such as Swedish massage, sports massage, trigger point therapy, cross-fiber friction massage, lymphatic drainage, and acupressure. Clinically, massage and soft tissue mobilization are thought to increase blood flow, promote relaxation, relax muscles, increase tissue extensibility, reduce pain, and speed return to normal function.[2] In horses, massage therapy has been shown to be effective at reducing stress-related behavior and pain thresholds within the thoracolumbar spine.[16,24] A noncontrolled, clinical trial using 8 horses measured increased stride lengths at the walk and trot compared pre- and postmassage, but changes were not significant due to a small sample size.[28] More high-quality, scientific evidence is needed to support the use of massage therapy in horses.[20]

PASSIVE STRETCHING EXERCISES

Passive stretching consists of applying forces to a limb or body segment to lengthen muscles or connective tissues beyond their normal resting lengths, with the intent of increasing joint range of motion and flexibility (Figure 8.14).[1] The amplitude of motion and length of time that an individual stretch is held is gradually increased over time, according to patient tolerance and ability. Stretching exercises are thought to increase joint range of motion, enhance flexibility, improve coordination and motor control, increase blood flow to muscles, and help to prevent injuries. In horses, passive stretching



Figure 8.14. Passive thoracic limb protraction stretch. The thoracic limb is fully extended and held at the end range of motion to promote elongation of soft tissues and increase joint range of motion.

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of the limbs and axial skeleton have anecdotal effects of increasing stride length and joint range of motion and improving overall comfort.[7] In a noncontrolled study, passive thoracic limb stretching lowered wither height due to possible relaxation of the fibromuscular thoracic girdle.[8] However, a randomized controlled trial in riding school horses evaluating the effect of two different 8-week passive stretching programs reported no significant changes in stride length at the trot but did measure decreased joint range of motion within the shoulder, stifle, and hock articulations, instead of the expected increased joint range of motion.[21] The authors concluded that daily stretching may be too intensive in normal horses and may actually cause negative biomechanical effects.

MOBILIZATION

Mobilization is manually induced movement of articulations or soft tissues for therapeutic purposes. Soft tissue mobilization focuses on restoring movement to the skin, connective tissue, ligaments, tendons, and muscles with the goal of modulating pain, reducing inflammation, improving tissue repair, increasing extensibility, and improving function.[2] Joint mobilization is characterized as nonimpulsive, repetitive joint movements induced within the passive range of joint motion with the goals of restoring normal and symmetric joint range of motion, stretching connective tissues, and restoring normal joint end feel.[22] Manipulation is a manual procedure that involves a directed impulse that moves a joint or vertebral segment beyond its physiological range of motion, but does not exceed the anatomical limit of the articulation. Therefore, the primary biomechanical difference between joint mobilization and manipulation is the presence of a high-speed thrust or impulse. Spinal manipulation involves the application of controlled impulses to articular structures within the axial skeleton with the intent of reducing pain and muscle hypertonicity and increasing joint range of motion (Figure 8.15).

Manual techniques used by physical therapists consist primarily of soft tissue and joint mobilization to assess the quality and quantity of joint range of motion and as a primary means of treating musculoskeletal disorders. Subjective assessment of the ease of joint motion, joint stability, and joint end feel provide insights into the biomechanical and neurologic features of an articulation. Few formal studies support the use of limb or spinal mobilization techniques in horses.[14] Most mobilization studies in horses involve a period of induced joint immobilization by a fixture or cast followed by allowing the horse to spontaneously weight bear and ambulate on the affected limb, without evaluation of specific soft tissue or joint mobilization techniques.[10]

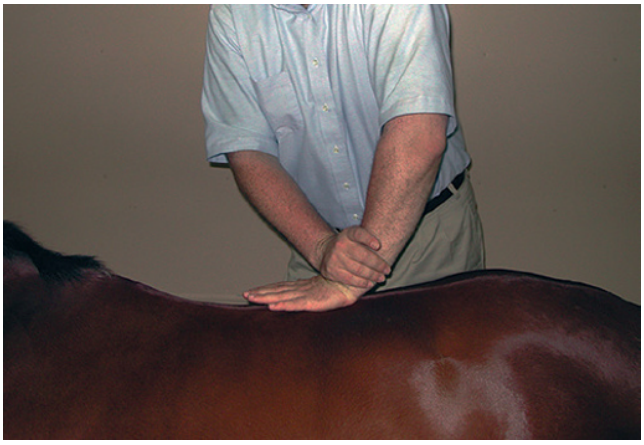


Figure 8.15. Thoracolumbar spinal extension mobilization. A gentle rhythmic force is applied over the dorsal spinous processes of the thoracolumbar junction in an effort to assess regional spinal flexibility and local pain or muscle hypertonicity.

JOINT MOBILIZATION AND MANIPULATION

The chiropractic and osteopathic professions have many overlapping philosophies, techniques, and potential mechanisms of action related to joint mobilization and manipulation. Manual osteopathic techniques use a combination of mobilization and manipulation methods to address impaired or altered function of the musculoskeletal system (i.e. somatic dysfunction).[3] From an osteopathic perspective, somatic dysfunction relates to impaired or altered function of skeletal, articular, myofascial, and related vascular, lymphatic, and neural elements.[17,19] Human osteopathic techniques also include highly controversial methods associated with mobilizing cranial bones and abdominal viscera, which have questionable application to horses.[26] Equine osteopathic evaluation and treatment procedures have been described in textbooks and case reports, but no formal hypothesis-driven research exists.[18,26] A case series of 51 horses with chronic lameness or gait abnormalities that were poorly localized were treated with osteopathic techniques under sedation and had reported positive results in the majority of case from 6 to 12 months after treatment.[4].

Chiropractic treatment is characterized primarily as the application of high-velocity, low-amplitude (HVLA) thrusts to induce therapeutic effects within articular structures, muscle function, and neurological reflexes. From a chiropractic perspective, the basic elements of joint or spinal dysfunction include altered articular neurophysiology, biochemical alterations, pathological changes within the joint capsule, and articular degeneration. Recent equine chiropractic research has focused on assessing the clinical effects of spinal manipulation on pain relief, improving flexibility, reducing muscle hypertonicity, and restoring spinal motion symmetry. Obvious criticism has been directed at the physical ability to even induce movement in the horse's back. Pilot work has demonstrated that manually applied forces associated with chiropractic techniques are able to produce substantial segmental spinal motion within the thoracolumbar region of horses.[13]

Two randomized controlled clinical trials using pressure algometry to assess mechanical nociceptive thresholds (MNTs) in the thoracolumbar region of horses have demonstrated that both manual and instrument-assisted spinal manipulation can reduce spinal nociception (or increase MNT values).[12,24] Additional studies have assessed the effects of equine chiropractic techniques on increasing passive spinal mobility (i.e. flexibility) and

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Table 8.5. Types of manual therapy, proposed mechanisms of action, and possible clinical applications.

Manual therapies	Mechanisms of action	Clinical applications
Touch therapies	Psychological: calming Behavioral deconditioning Novel cutaneous stimulation Mechanoreceptive stimulation	Behavioral issues Anxiety or nervousness Poor proprioception or body awareness Pain
Therapeutic massage	Psychological: relaxation Mechanical: soft tissues Neurophysiological: pain	Anxiety or nervousness Soft tissue restriction Muscle hypertonicity Pain
Passive stretching exercises	Mechanical: articular and soft tissues Neuromotor learning	Soft tissue restriction Muscle hypertonicity Joint stiffness Pain
Mobilization		
Soft tissue	Mechanical: soft tissues	Soft tissue restriction Muscle hypertonicity Pain
Articular	Mechanical: articular and soft tissues	Restricted joint motion Joint effusion Pain
Manipulation	Mechanical: articular and soft tissues Neurophysiological: Pain Mechanoreceptive stimulation	Restricted joint motion Joint motion asymmetry Muscle hypertonicity Pain

reducing longissimus muscle tone.[14,27] The effect of manipulation on asymmetrical spinal movement patterns in horses with documented back pain suggest that chiropractic treatment elicits minor but significant changes in thoracolumbar and pelvic kinematics and that some of these changes are likely to be beneficial.[6,9]

Manual therapy is believed to produce physiological effects within local tissues, on sensory and motor components of the nervous system, and at a psychological or behavioral level.[15] It is likely that specific manual therapy techniques are inherently more effective than others in addressing each of these local, regional, or systemic components (Table 8.5). The question is choosing the most appropriate form of manual therapy or combination of techniques that will be efficacious for an individual patient with specific musculoskeletal disabilities.

CONTRAINDICATIONS

Contraindications for mobilization and manipulation are often based on clinical judgment and are related to the technique applied and skill or experience of the practitioner.[22] Few absolute contraindications exist for touch therapies, massage, and joint mobilization, if the techniques are applied appropriately. Manual therapy is generally contraindicated in the presence of fractures, acute inflammatory or infectious joint disease, osteomyelitis, joint ankylosis, bleeding disorders, progressive neurological signs, and primary or metastatic tumors.[22] Acute episodes of osteoarthritis, impinged dorsal spinous processes, and severe articular instability, such as joint subluxation or luxation, are also often contraindications for manipulation. All horses with neurologic diseases should be evaluated fully to assess the potential risks or benefits of joint mobilization or manipulation. Serious diseases requiring immediate medical or surgical care must be ruled out and treated by conventional veterinary medicine before any routine manual therapy is initiated.

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REHABILITATION/PHYSICAL THERAPY

Melissa King, Katherine Ellis, and Narelle C. Stubbs

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INTRODUCTION

Rehabilitation and physical therapy (PT) play an important role in performance enhancement, injury prevention, and restoration of full function during recovery from injury. The principles of rehabilitation and PT are based on a combination of evidence-based medicine, clinical reports, and comparative human sports sciences. Veterinary medicine continues to follow the principles of human sports medicine and rehabilitation to encompass many professional fields, including PT. Rehabilitation encompasses broad-based concepts with a focus on tissue healing, biomechanics, and neuromotor control. These concepts relate to morphology, conformation, training, environmental conditions, type of competition, age, performance limitations, exercise effects on the neuromuscular and skeletal systems, and interaction between horse and rider. The sciences underlying rehabilitation/PT include biomechanics, motor control, and exercise physiology.[11,28,76,127]

The word rehabilitation comes from the Latin "rehabilitare," meaning to make fit again. Rehabilitation/PT techniques are individually tailored to each patient rather than prescriptive for a given lesion or pathoanatomical diagnosis and consider the whole horse, short- and long-term goals, discipline, and overall prognoses. Each individual patient protocol is designed to complement medical management and facilitate the process of recovery by promoting tissue healing, assisting the normal physiological processes, and restoring the patient to its former capacity following an illness or injury. This includes manual therapies, electrotherapy, functional retraining, proprioceptive exercises, application of various modalities, and therapeutic exercise-based treatments, along with education and ongoing owner-managed procedures. These are the founding principles of the physiotherapy or PT profession as described by the World Confederation for Physical Therapy (WCPT).[196] PT in all species is concerned with identifying and maximizing quality of movement potential within the spheres of promotion, prevention, treatment/intervention, rehabilitation, and more recently sports medicine and performance enhancement.

Rehabilitation in veterinary medicine involves the veterinarian as the primary pathoanatomical diagnostician, a thorough objective functional assessment of the patient, and consultation with other health professionals.[127] Using knowledge and skills unique to these professions, the movement potential of the patient is assessed, with all information and confounding factors incorporated to establish an accurate functional diagnosis, problem list, management plan, and goals. Clinically, it is imperative that during the rehabilitation process, valid and reliable objective measures are taken to determine accurate outcome measures. It is the author's opinion and one that is advocated in all modern sports medicine literature that rehabilitation should begin as soon as possible after the injury occurs, alongside therapeutic pharmacological measures such as anti-inflammatory and other pain-modulating agents.[55] This might also begin before or immediately after surgery; therefore, the rehabilitative process is managed by a multidisciplinary team.

There is still a great need for further research on the effects of PT in horses to complement the considerable research in pathology and the high prevalence of injuries in athletic horses.[50,179,206] Although peer-reviewed research is limited in equine rehabilitation,[29,31,72,106,107,161,193,204,211] many animal models have been used for human rehabilitation research, with many findings able to be related to current concepts in equine rehabilitation. The *Animal Physiotherapy: Assessment,* Treatment *and Rehabilitation of Animals* textbook laid the foundations of animal physiotherapy; it is a good reference with respect to evidence-based rehabilitation.[128] The profile of rehabilitation and performance enhancement has been heightened by the official use of PT and other allied health professionals during international level equestrian competitions, highlighting how equestrian sports are rapidly catching up to other international competitive sports.

Readers are directed toward the many evidence-based human therapeutic texts to extend the scope of evidence-based techniques that may be enlisted to rehabilitate joint and soft tissue disorders and lesions. These include manual therapies, soft tissue mobilization/massage,[18] myofascial pain and dysfunction,[179] electrotherapy and physical agents,[131,209] and integrative therapeutic approaches relating to mechanical passive constraints of locomotion and neuromotor control, including facilitation and strengthening techniques.[116,126,135]

It is beyond the scope of this section to cover all aspects of equine rehabilitation. The aim is to present some fundamental evidence-based concepts of PT/rehabilitation focusing on manual therapies, electrotherapy, therapeutic and proprioceptive exercise-based treatments, and commonly applied modalities.

CLINICAL AND CLIENT DEMAND FOR REHABILITATION/PT

The clinical need for rehabilitation interventions is verified by the amount of waste and loss of performance in equine sports due to musculoskeletal injuries.[14,50,146,200] Statistics indicate that diseases of the musculoskeletal system are the predominant cause of loss of use and death in sport horses.[26,45,74,147,206] For example, it is well established that flexor tendon and suspensory ligament injuries are the most frequently reported soft tissue injuries in sport horses, resulting from chronic repetitive overuse injuries.[32,92,125,133,162,165,167] Similarly, in a population of western performance horses, soft tissue injuries of the forelimbs (deep digital flexor tendinopathy within

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the foot) and hind limb proximal metatarsal desmopathy were most notably the highest identified sources of lameness.[96] Furthermore, conservative management of hind limb suspensory desmopathy carries a poor prognosis with only 14% of horses returning to work for at least 1 year.[44] However, recent retrospective studies are demonstrating that professional rehabilitation alongside other employed therapeutics improved results, with 56% of horses suffering from hind limb proximal suspensory disease returning to their previous level of work.[97]

Back pain is considered one of the most common and least understood clinical problems in horses. It is a common cause of decreased performance, behavioral issues, and loss of work in equine athletes. Equine back pain can be of primary origin or can be secondary to referred pain from the appendicular skeleton.[77] The prevalence of back problems has been reported in 27%–100% of the ridden horse population.[118] It has been reported that 32% of horses with lameness also have concurrent back pain.[112] Biomechanical studies demonstrating three-dimensional motion of the thoracolumbar and pelvic regions have provided valuable information into the adaptive and maladaptive compensatory movement patterns associated with fore and hind limb lameness and back pain.[8] With our increased understanding of the underlying mechanisms causing various types of back pain comes a dire need to develop multimodal therapeutic and rehabilitative protocols.

Common treatments of back pain in horses include local injection with corticosteroids, systemic anti-inflammatories, extracorporeal shockwave therapy, and acupuncture.[164] Unfortunately, these traditional therapeutic pursuits often result in recurrent pain, associations with concurrent lameness and behavioral issues.[21,37] In people with low back pain, the early institution of therapeutic modalities targeting pain, muscle spasms, and restricted range of motion leads to significant improvements in function and substantial reductions in pain.[25,103,203] In addition, institution of core stability exercises results in reduced back pain and longer remission of low back pain.[2,102,218] In horses, improving core fitness results in improved lameness.[164] However, before a core fitness program can be instituted effectively, a reduction in the horse's pain must occur. Whether human or equine, the specific rehabilitation goals to augment spinal and pelvic dysfunction include decreasing pain, improving flexibility, and restoring neuromotor control and function. Treatment of back pain can be extremely challenging and often requires a multimodal approach including targeted rehabilitation protocols designed to maximize back pain treatment efficacy.

MANUAL THERAPY

Manual therapy techniques are suitable for many conditions following a veterinary pathoanatomical diagnosis, when possible. These techniques encompass the application of very specific passive and/or active assisted movements by the therapist to the horse to manage and/or alter pain and dysfunction of the articular, neural, and muscle systems. Manual therapy techniques are based on a wide range of methods and theories related to the intervertebral and peripheral joint complexes, myofascial, and the neuromuscular system.

Passive accessory and physiological joint mobilization techniques are specific to PT and osteopathy and have been clinically reported to be successfully adapted to treat the horse. Some technique examples used in human manual therapy include Maitland, Mulligan, Kaltenborn, McKenzie, and muscle energy techniques. Numerous myofascial and neuromuscular mobilization techniques also can be used, some of which include massage (friction, effleurage, petrissage, tapôtement, vibrations, and shaking), trigger point therapy, direct and indirect myofascial release, positional release, reflex inhibition techniques, craniosacral therapy, adverse neural tension techniques, and stretching.

To effectively establish a treatment protocol and apply these techniques, an accurate functional assessment of the musculoskeletal and neuromuscular systems is vital, identifying the primary and secondary condition(s) that may be responsible for and/or attributed to pain and loss of function. The assessment procedure, where applicable, includes a full static and dynamic examination, followed by palpatory evaluation including specific manual testing and provocation tests. Examination procedures determine the regions of altered range of motion, quality of movement, muscle function, and tissue irritability that may be related to pain, pathology, altered motor control, and/or dysfunction.[61,62,151]

Joint Mobilization

Degenerative joint disease or osteoarthritis is an example of a common dysfunction and cause of lameness and loss of performance in the horse in which manual therapy can be successfully applied, as is the case in humans. Many manual therapy techniques such as passive mobilizations can be applied to the articular system, including the vertebral column and peripheral joints, with complementary soft tissue techniques applied to the associated neuromuscular and fascial tissues in this condition. These repeated movement techniques have reported effects on the intra-articular, periarticular (joint capsule and ligament), and extra-articular structures (muscle, fascia, and neural tissue) and thus affect the passive and active constraints of the joint complex and primarily assist in pain modulation.[60,62,70,151]

There is a vast amount of scientific evidence in the human literature to support manual therapy techniques and the possible biomechanical and neurophysiological mechanisms behind the effects, with many studies using animal models.[10,90,183,184] The neurophysiological mechanisms that explain the pain-relieving effects are now supported by scientific evidence, both in the vertebral column and in peripheral joints. Evidence has shown that manual therapy produces an initial treatment-specific hypoalgesia and sympathetic excitatory effect locally in the treatment area beyond placebo or control conditions and then a nonopioid-mediated hypoalgesia systemically.[148,183,201,202,204,213]

Human clinical studies assessing passive range of motion exercises in patients following knee arthroplasty showed a significant improvement in range of motion that was still apparent after 1 year.[170,171] The improvement in range of motion can be supported from results in the rabbit models that demonstrate as healing occurs within the joint capsule collagen fibers align randomly.

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This random, cross-linking pattern causes increased fibrosis and thickening of the capsule. Passive range of motion exercises are proposed to work at a cellular level by decreasing the random alignment of the collagen fibers through stimulation of physiologic motion enhancing parallel fiber orientation.[52] Two very effective mobilization techniques often used to restore joint motion and reduce pain in the horse are described by Goff:[60] (1) passive physiological mobilizations, whereby the forces being directed reproduce passive physiological or coupled joint motion around the 3 axes of motion of the affected joint complex, as occurs with voluntary motion, and (2) passive accessory movements, in which the motion produced is a translation that accompanies rotations, which cannot be voluntarily performed by the horse. Figure 8.16 is an example of the application of a combination of these techniques applied to the carpus.

These techniques are highly applicable in mid and end of range of motion positions (as seen in Figure 8.16), which is commonly the practice in human PT, rather than treatment taking place only in the neutral standing posture. Clinically this is particularly effective in relation to intersegmental motion in the spine. The practitioner is able to use combined mobilization with movement techniques as described below, or with an assistant to maintain the desired postures. Direct dorsoventral and lateral passive accessory glides can then be performed manually over the dorsal spinous processes, ribs, vertebral bodies (cervical spine), and transverse processes (lumbar spine), in the end-of-range functional positions.

Soft Tissue Mobilization

The aim of many manual therapy techniques directed at the myofascia, tendons, and ligaments is to normalize tissue irritability, muscle tone, extensibility, length, contractility, strength, and coordination and ultimately improve motor control. Addressing these impairments in an attempt to regain function and optimize performance is a key feature of the rehabilitation process.

Muscular dysfunctions are often seen occurring secondarily to underlying bone pathology in horses with limb and/or back pain; however, dysfunction and/or atrophy may also be due to pathology of actual muscles such as a generalized muscle disorder, or muscle damage attributed to a poorly fitting saddle.[58,69,159,199] Valentine[200] investigated the pathological findings in equine muscle (excluding polysaccharide storage myopathy) in 229 equids. Sixty-five percent (n = 129) suffered muscle lesions, (13.1%, n = 30) generalized muscle atrophy, and (6.1%, n = 14) denervation atrophy. Clinical signs of myopathies have been shown to improve with dietary management along with a regulated exercise routine as part of rehabilitation.[86]





Figure 8.16. Manual therapy mobilization technique. A combination of passive physiological and passive accessory mobilization techniques is applied to the carpus to increase the available range of motion into flexion. The carpus is flexed, the radius is stabilized, and medial (A) and lateral motions (B) are applied to the end of range via the third metacarpus.

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Following soft tissue trauma or pathology, dynamic stretching regimes can be implemented to regain and maintain full range of motion during the healing process. As a preventative measure, this also applies to any horse that is on a restricted exercise regime or confined to the stable for other reasons to help avoid the detrimental effects of immobilization.[144] Passive stretching techniques are much more difficult in the horse because the horse is never entirely passive with respect to muscle tonicity, because it is usually standing during treatment. Therefore, the stretching procedure is dynamic and never completely passive, even when stretching limbs. However, an osteopathic technique that is reported in the clinical literature suggests that a form of gentle stretching ("positional release") in the treatment of chronic myofascial restrictions can be performed under sedation or anesthesia. This technique has been reported to have some success.[157] The technique is often used after all other avenues have been exhausted due to the anesthetic risk. Therefore, tissue mobilization with movement may be a more apt title for many forms of stretching techniques in the horse. These techniques are performed rhythmically or in a sustained manner depending on the desired response and effect on the tissues. Examples of these techniques are described below in the section on mobilization with movement exercises.

If there is a primary lesion of a soft tissue structure such as muscle, ligament, or tendon, stretches or active mobilizations with movement exercises in conjunction with other therapies may be appropriate to avoid excessive scar formation and disorientation of the fiber alignment in the subacute and chronic phases of healing. With respect to rehabilitation of distal tendon and ligament lesions, it has been widely reported in the human literature that dynamic motor control-based exercises, including eccentric loading exercises, accelerate healing and tensile strength, especially in relation to Achilles tendon lesions.[108] This type of rehabilitation strategy is beginning to be more commonly implemented where appropriate in horses. In these cases, the horse undergoes "active rest" and begins a walking program along with other forms of therapy, once the lesion is determined to be stabilized. It is the author's suggestion to also include unmounted exercises to increase the dynamic core muscle strength as described below.[190] Fibrotic myopathy is an example of a chronic condition in which repeated sustained positioning of a limb may be warranted in combination with other forms of therapy. In this case, the affected limb is manually moved to the available range of motion to stress the desired soft tissue structures.

Mobilization with Movement Techniques/Exercises

Indirect mobilization with movement techniques/exercises in conjunction with direct manual therapy techniques is also very effective clinically, especially in relation to functional motor control. Altered motor control (neuromuscular function) may be a result of the underlying lesion in the spine and/or peripheral joint disease, with pain and inflammation causing reflex inhibition of motor neurons, which in turn causes weakness and atrophy of associated muscles.[219] This is widely reported in the human literature, indicating that motor control is a vital component of the management of musculoskeletal conditions, especially related to spinal disorders and dysfunction.[75,102] McGowan et al.[128] reported that there is a relationship between muscle function and pathology via ultrasonography and necropsy analysis, whereby significant atrophy of the multifidus muscles was evident at the level and side of the thoracolumbar lesion in 22 racehorses. This has been widely reported in the human literature.[75] This is also an area of veterinary medicine in which rehabilitation/PT techniques may be of great use.

Many of these mobilization techniques/exercises use neuromuscular reflexive responses along with muscular facilitation and inhibition. They use similar theoretical principles to those used in human manual therapy, including muscle energy techniques to gain motion that is limited by restrictions of neuromuscular structures, as well as restore or normalize motor control. In people, the patient is asked to contract and relax specific muscles in a given range of motion, and often resistance is applied to the body part. These techniques promote muscle relaxation by activating the Golgi tendon reflex.[38] The techniques implement the principles of reciprocal inhibition and post-isometric relaxation. Reciprocal inhibition uses the body's antagonist-inhibition reflex to induce relaxation of a muscle.

A simplified example in people: If there is a loss of elbow flexion range of motion due to tricep muscle hypertonicity, the elbow is placed into end range of flexion, and biceps (agonist) are then contracted, which induces the triceps (antagonist) into relaxation. The joint is then moved farther into maximal flexion, and the technique is repeated, with incremental restoration of range of motion. Conversely, other techniques use the antagonist-inhibition reflex to incrementally restore range of motion, whereby a muscle is stretched immediately following its isometric contraction due to the neuromuscular apparatus becoming briefly refractory, or its inability to respond to further excitation.

It is the author's opinion that many of these applied techniques/exercises are very useful in the majority of rehabilitation cases to maintain and improve mobility, strength, and dynamic stability. This is especially true in those cases involving regions where the affected joint complexes are inaccessible due to the horse's morphology.

The horse alters its posture reflexively in response to digital pressure on various regions of the axial skeleton, both into dorsoventral flexion–extension and into coupled lateral bending/rotation and dorsoventral motion. For example, this can be seen when there is a reduction of dorsoventral motion into flexion and/or lateral bending of the cervicothoracic and thoracolumbar–lumbopelvic regions due to pain, muscle spasm, and/or joint pathology or dysfunction. The intention is not only to increase the joint mobility but also to activate and strengthen the muscles required to move the horse's body to the desired position.

A simple example of these exercises is seen in Figure 8.17, where the therapist applies a slow, constant pressure to the ventral midline and dorsal sacral region simultaneously, facilitating end of range of motion via a dorsoventral flexion response of the cervicothoracic and thoracolumbar spine and the pelvis. In this example, activation of the horse's core muscles, serratus ventralis and

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Figure 8.17. Manual therapy mobilization with movement technique. A combined rounding response into end-of-dorsoventral flexion range of motion of the thoracolumbar spine and pelvis. This is performed by manual finger pressure to the ventral midline with one hand and dorsally directly over the midline of the sacrum with the other hand. Depending on the size of the horse, this may need to be performed by two people.

hypaxial muscles (pectoral, abdominal, and iliopsoas complexes) produces the desired posture in combination with relaxation and lengthening of the opposing epaxial muscles. This posture is maintained for 3–5 seconds and repeated multiple times as reported by Stubbs and Clayton to incrementally restore range of motion and strengthen the core muscles.[190] Indirect techniques can also be used to mobilize other inaccessible regions such as the cervicothoracic junction, whereby a muscle reflex/response occurs on deep palpation (constant index and thumb pressure) of the distal third of the brachiocephalicus muscle, producing flexion in this region. The brachiocephalicus response is shown in Figure 8.18. This can also be coupled with ventral pressure to the sternum to mobilize the joint complexes into an increasing range of upper thoracic flexion.

Many combinations of mobilization, core strengthening, and stability/balancing exercises have been described in the literature.[60,144,190] These often use a food incentive or bait (such as a carrot) to encourage the horse to move into the desired posture. Baited exercises encourage the horse to move into end-of-range postures of the cervicothoracic and lumbopelvic regions, for example, into flexion with movement of the chin to chest, between carpi and fetlocks, or coupled lateral/rotation bending and flexion, with movement of the chin to girth, flank, and hock (Figure 8.19). The aim of these exercises is not only to mobilize both the axial and distal skeletons but also to facilitate core muscle activity (thoracic sling, hypaxial/epaxial, and pelvic musculature), improve neuromuscular control, and strengthen over time.[190] This is a key concept in human rehabilitation and sports medicine. Pursuant to the equine patient, several core strengthening exercises and their role in activating deep epaxial musculature to subsequently improve postural motor



Figure 8.18. Mobilization with movement technique. The brachiocephalic response results in flexion of the mid-lower cervical spine, including the cervicothoracic region. Gradual digital pressure is applied by the forefinger and thumb (pincer) to the distal third of the brachiocephalicus muscle. Pressure is maintained and gradually increased until the desired posture is attained, then held for 3–5 seconds.

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Figure 8.19. Baited mobilization with movement exercises; lateral bending coupled with flexion and rotation. The horse's functional end-of-range posture is attained by the horse's chin following the carrot toward its hock/fetlock. The position is held for 3–5 seconds to increase the activity of the core muscles of the thoracic sling, trunk, and pelvic sling, as seen in the photograph.

control and alter thoracolumbar kinematics have been investigated.[30,100] Both baited and passive exercises offer opportunities to facilitate stretching during dynamic phases and strengthening during static phases of the exercise. Institution of dynamic mobilization exercises over a 3-month time period has been shown to increase both size and symmetry of the multifidus muscle as assessed through longitudinal ultrasonographic evaluation.[191,192]

Multiple human studies have demonstrated that individuals with spinal pathology and back pain have reduced muscle CSA leading to loss of function and impairments in postural control and proprioceptive acuity. Human athletes that incorporate core, balance exercises into their rehabilitation programs are significantly less likely to suffer re-injury during a 12-month period following injury, compared with those individuals with similar injuries that did not emphasize core strength (7% re-injury rate in the balance training group versus 29% re-injury rate in the control group).[80] Strengthening and improving proprioception and balance control following injury remain a central focus of human physical therapy programs, and while standardized investigations have yet to focus on equine applications, there are several mechanisms through which neuromotor control can be recruited. Physical therapy aides such as balance pads (Figure 8.20), ground poles, tactile stimulators, and incorporation of surface changes offer clinicians passive means of engaging neuromotor control during activities of daily rehabilitation or training. Ground poles when arranged at various distances, heights, and configurations can encourage increase in lateral thoracolumbar excursion. Hill work and incorporating backing exercises into hill work can also be used to simultaneously improve muscular strength and challenge proprioceptive acuity.

PROPRIOCEPTIVE FACILITATION/NEUROMOTOR CONTROL TECHNIQUES

Recent human research has expanded our understanding of neuromuscular responses to joint pain.[194] Mechanoreceptors are characterized as sensory receptors within periarticular tissues that respond to changes in joint position and acceleration of movement, as well as playing an important role in regulating and maintaining neuromuscular control of joint stability. Pain, inflammation, and joint effusion associated with OA alter the normal sensory input from articular mechanoreceptors, which may result in decreased motor neuron excitability and reduced muscle activation.[93] A common outcome following experimentally induced knee effusion is the presence of significant quadriceps muscle inhibition.[81,82,89,143] A linear relationship exists between increased intra-articular pressure and the level of quadriceps inhibition.[81] Electromyography studies conducted on the quadriceps muscle of patients with knee osteoarthritis often indicate a decrease in muscle strength and altered muscle timing.[39] Typically there is a delay in the onset of quadriceps muscle activation, which results in the inability of the quadriceps to stabilize the joint and attenuate

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Figure 8.20. Horse standing with all four limbs on firm textured balance pads. Balance pads can be used during various stages of rehabilitation to engage the core and improve core strength and postural stability.

the loading forces properly during locomotion.[82] The increased joint instability further alters the distribution of weight-bearing forces across joint surfaces and increases the recruitment of adjacent muscles to aid in joint stability.[177] The resulting imbalances in paired agonist–antagonist muscle groups contribute to increased joint instability and altered limb biomechanics, which leads to progressive OA and chronic maladaptive compensatory mechanisms.[214]

Similar results would be expected in horses: the presence of pain may lead to loss of function, diminished neuromotor control, decreased joint range of motion, and muscle atrophy. The application of various proprioceptive techniques is frequently utilized to increase joint range of motion, reestablish appropriate neuromuscular firing patterns, and improve the strength of targeted muscles that function to move and stabilize the joints.[31] Sensory integration techniques during exercises involve stimulating the afferent proprioceptive/mechanoreceptive input that modulates and coordinates motor function. These techniques are potentially very effective due to the horse's heightened skin-mechanoreceptive system in conjunction with the underlying cutaneous trunci myofascial attachments to the skin. For example, the horse has the ability to feel the smallest tactile stimulus such as a fly and respond by altering its muscle activity (i.e. fasciculation of cutaneous trunci). Tactile stimulants (Figure 8.21) or cues can be applied to the skin over targeted regions, including the limbs or specific muscles,



Figure 8.21. Sensory integration technique. A lightweight (55-g) tactile stimulation device is placed around the coronary band of a horse and worn during exercise to increase the peak height of the swing phase of gait.

to alter mechanoreceptive/proprioception feedback and thus potentially alter motor control. The use of tactile stimulators applied around the equine hind limb pastern demonstrated a significantly higher hoof flight arc, with increased flexion of the fetlock, hock, and stifle joints.[31] The use of this proprioceptive technique supports the facilitation of enhanced afferent input to indirectly produce and modulate a targeted efferent response for the purpose of reestablishing motor control and improving joint range of motion.

A multitude of other proprioceptive techniques are utilized from resistive bands and kinesio tape, to ground

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poles and changes in ground surfaces. Resistance band training is successfully used in human physical therapy programs to improve core strength and stability.[101,122] Commonly referred to as a Theraband,[11] the two-piece equine elastic band system is thought to stimulate core abdominal muscles with the abdominal band and engage hind limb musculature with the hindquarter band. Its use in horses at a trot was recently investigated and found to reduce mediolateral and rotational movement throughout the thoracolumbar region.[152] Further studies investigating more long-term use and potential mechanistic pathways will help refine its use in the rehabilitation setting. Also pertinent to rehabilitation is the use of training lines. Pessoa training aids were demonstrated to increase both lumbosacral angles and thoracolumbar dorsoventral excursion when used in horses being lunged at a jog.[205] While further studies are needed to establish specific recommendations regarding use, initial biomechanical affects for targeted rehabilitation are encouraging.

Additional proprioceptive facilitation involves the use of elastic therapeutic tape. Elastic therapeutic tape is an adhesive product designed for use in treating sports injuries and a variety of other musculoskeletal disorders in humans. Manufacturers claim that elastic therapeutic tape supports injured muscles and joints and helps to relieve pain and edema by lifting the skin and allowing improved blood and lymph flow.[212] A review of the current human literature suggests that elastic therapeutic tape may have a small beneficial role in improving cutaneous proprioception, strength, and joint range of motion, but further studies are needed to confirm these findings.[212] Studies have shown effects on underlying muscle activity, but it is unclear whether these changes are beneficial or harmful in the management and prevention of specific human sports injuries. In the veterinary literature, Ramon et al.[201] reported that rigid mechanical athletic taping of the fetlock does not alter the kinematics of the forelimb during stance, but it does limit flexion of the fetlock during the swing phase.[183] However, there was a decrease in the peak vertical force, which may be due to an increased proprioceptive effect. The authors concluded that the reduced peak vertical forces may be helpful in preventing or reducing injury and potentially could be applied for tendinous or ligamentous rehabilitation in equine patients. These effects also may differ if using a nonmechanical but functional taping technique, as is commonly used in human athletes and has been widely researched. Theoretically, tape is stretched and placed along the length of a muscle that the practitioner wishes to facilitate. This is achieved through tension that increases the kinesthetic awareness across the fibers of the muscle where an inhibitory effect is desired. The horse can perform specific exercises with the tape attached, including individual sport-specific activities under saddle, which is an advantage with respect to alteration of neuromotor function over time. The author has successfully used taping techniques in the regions of the core dynamic stability musculature, specifically the biceps femoris and the abdominal complex (Figure 8.22). Equine taping certification courses have been initiated; however, the current level of evidence for use in treating musculoskeletal disorders is largely anecdotal, but promising results have been appreciated with preliminary investigations incorporating the use of pre- and post-application algometry pressure scores (King, unpublished data). Elastic therapeutic taping is used clinically most often to promote changes in motion and limb function.

These techniques may be combined with many forms of in-hand and ridden exercises that are widely described in the clinical literature.[60,63] The exercise protocol should be individually tailored by the therapist, with a skilled handler/trainer required to perform these exercises on a daily basis. The exercise protocol should follow the principles of conditioning,[27] with constant reassessment of the horse's motion to ensure that compensatory strategies are minimal and the desired effects evident. The program should be gradually increased with consideration given to exercise time, gaits, transitions, direction, surfaces, and gradients/slopes. The horse should be constantly monitored for excessive fatigue.

PHYSICAL MODALITIES

It is beyond the scope of this section to define and describe all forms of electromodalities and biophysical agents that alter and/or accelerate tissue healing and minimize the effects of disuse atrophy, immobilization, and denervation. The reader is advised to consult texts that are specifically aimed at evidence-based practices in the field of PT, electromodalities, and physical agents.[15,132,166,209]

Thermal Therapy

Thermal therapy consisting of cryotherapy, heat therapy, or a combination thereof (contrast therapy) remains a fundamental cornerstone of many physical therapy programs. Generally speaking, cryotherapy is indicated in acute injuries to reduce pain, swelling, and inflammation, while heat therapy is utilized in more chronic conditions to encourage soft tissue extensibility, decrease muscle spasming, and increase local blood flow.[11] Methods to apply thermal therapy have been widely developed for use in the equine distal limb, but the equine back and pelvic regions remain challenging due to limitations in anatomic depth of penetration and difficulty in secure application mechanisms.

Cryotherapy

Mechanism of Action

The therapeutic effects of cold therapy are generated through reducing tissue temperatures to 10–15 °C. (petrov03)[152] Tissue cooling produces peripheral vasoconstriction and decreased soft tissue perfusion, which can reduce edema formation and swelling at the site of tissue injury. Cold therapy also mitigates tissue metabolism and apoptosis, inhibits the effect of inflammatory mediators, and abates local enzymatic activity.[6] The application of cold also serves as a form of pain modulation by decreasing nerve conduction velocities in local sensory neurons and by activating descending inhibitory pathways.[182] Cold therapies can penetrate up to 1–4 cm in depth, which is dependent on local circulation and adipose tissue thickness.[19] Human studies have documented

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Figure 8.22. Sensory integration. In this example of a taping technique of the abdominal complex, Kinesio Tape™ is applied to stretch from the transverse processes of the lumbar vertebrae, following the line of the transverse abdominus, internal oblique, and rectus abdominus muscles. The tape finishes on the ventral aspect of the sternum and pectoral muscle complex.

the analgesic benefits of cryotherapy with a 15–20 minute application providing pain relief for 1–2 hours.[65,172]

Evidence in Horses

Ice water immersion for 30 minutes reduced the superficial and subcutaneous tissues in the distal limb to within optimal therapeutic range compared with cold pack application.[100] Ice water immersion remains the superior form of cryotherapy for the horse; however application of a compression boot with continuous circulating coolant applied to the distal forelimb of horses for 1 hour significantly reduced the superficial digital flexor tendon (SDFT) core temperature to 10 °C.[150] Similarly, a dry sleeve perfused cuff with continuous circulating coolant that included the hoof and distal limb was as effective as ice water immersion in reducing hoof wall surface temperatures to <10 °C for over an 8-hour period.[51] In both humans and dogs, circulating cryotherapy and intermittent compression provide a significant reduction in pain, swelling, and lameness and an increase in joint range of motion after orthopedic surgery.[16,42] The recent results of tissue cooling being as effective with circulating cryotherapy units compared with ice water immersion enhances efficacy and safety in the clinical application of cryotherapy. Further *in vitro* research has demonstrated that the viability of tenocytes exposed to cold treatment (10 °C) for 1 hour did not significantly differ from those cells maintained at 37 °C.[51] Cryotherapy research in horses has focused primarily on applications within the distal limbs and on inflammatory responses associated with laminitis.[155] The exact effect of cryotherapy on various equine musculoskeletal injuries has not been fully elucidated. Additional studies are still needed to create evidence-based guidelines on the use of cryotherapy. These must address effective duration, frequency, temperature, and safety of application that will optimize outcomes after injury.

Heat Therapy

Mechanism of Action

The topical application of heat produces increases in local circulation, induces muscle relaxation (and therefore, reduction of muscle spasms and associated pain), and increases tissue extensibility.[73,132] Increased local blood flow mobilizes tissue metabolites and increases tissue oxygenation and the metabolic rate of cells and enzyme systems. In general, metabolic rate increases

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2–3 times for a tissue temperature increase of 10 °C.[161] Soft tissues may be stretched more effectively when they are warm. Heat decreases tissue viscosity and increases tissue elasticity. Low-load, prolonged stretching of tissues heated between 40 and 45 °C results in increased extensibility of tendons, joint capsules, and muscles.[73,132]

There are two forms of thermotherapy: superficial (heating tissues at a depth of 1 cm) and deep. Deep thermotherapy is the application of modalities that cause a rise in tissue temperatures beyond the level of the skin. Mechanisms to heat the deeper tissues include use of continuous wave therapeutic ultrasound and radiofrequency diathermy. Both modalities have been reported in nonequine species to therapeutically heat tissues and thus modulate pain at a depth of 3–5 cm. Methods of superficial heat application include heating blankets and packs, or other materials that can be heated in a microwave and affixed to the patient. Although clinical effectiveness for superficial heating has yet to be demonstrated, it is often recruited in both the training and rehabilitation settings prior to exercise as mounted heating lamps (solarium).

Therapeutic Ultrasound

Mechanism of Action

Therapeutic ultrasound converts mechanical energy into sound waves through the piezoelectric effect – passing an electric current through a crystal. The sonic waves produced are transmitted by propagation through molecular collision and vibration, which results in generation of heat if a continuous pulse wave is applied.[142,188] The thermal effects of therapeutic ultrasound are dependent on the amount of tissue heating that occurs. Increasing tissue temperature by 1 °C from baseline tissue temperatures will increase the regional tissue metabolic rate, a 2–4 °C rise in tissue temperature reduces pain and inflammation and increases blood flow, and a tissue elevation >3 °C is necessary to improve tissue elasticity.[36,78,114] Nonthermal effects also occur, which include induction of cavitation and acoustic microstreaming. These effects alter cell membrane structure and function, resulting in stimulation of tissue repair, fibrinolysis, collagen synthesis, tissue regeneration, and bone healing. Other nonthermal effects demonstrated *in vitro* include earlier resolution of inflammation, heightened fibroblast recruitment, accelerated angiogenesis, and increased tissue tensile strength.[24,188] Nonthermal effects occur during both application of a continuous and pulsed duty cycle application. Penetration and absorption of the ultrasound waves are dependent on the intensity applied and the frequency used. Use of 1 MHz results in absorption at a depth of 3–5 cm, while use of 3 MHz results in absorption at 1–2 cm. Low absorption of ultrasound waves occurs in tissues of high water content, such as fat, while high absorption occurs in tissues rich in protein, such as skeletal muscle.[188] Therapeutic ultrasound can also be used to delivery low molecular weight drugs transdermally, a process known as phonophoresis.[141,188]

Evidence in Humans

Therapeutic ultrasound has been shown to be effective at reducing pain (average reduction of 47%) and increasing functional scores in patients with knee osteoarthritis, when compared with placebo.[142] It has also been shown to increase the effectiveness of isokinetic exercise for functional improvement in knee osteoarthritis (range of motion, walking speed), particularly when a pulsed duty cycle was used.[84] However, therapeutic ultrasound showed no significant reduction in pain when used for conditions affecting the lower limb.[175] Thermal application of therapeutic ultrasound was shown to increase tissue temperature by 5 °C in the triceps at 3 MHz. This temperature increase, however, was short-lived, lasting 3.3 minutes.[41] Therapeutic ultrasound has been used with mixed success to stimulate bone growth and fracture healing in both animal models and human clinical trials. In humans, low-intensity pulsed therapeutic ultrasound has been approved by the FDA to treat acute and nonunion fractures. Low-intensity pulsed therapeutic ultrasound targeting fracture healing has established an optimal intensity of 30 mW/cm^2 at 1.5 MHz for 20 minutes in both human and nonhuman models. Success rates of low-intensity pulsed ultrasound for the treatment of human delayed unions and nonunions range from 67% to 90%.[94,207]

Evidence in Horses

A study performed in 2013 evaluated the ability of therapeutic ultrasound to heat the SDFT, DDFT, and epaxial musculature to therapeutic levels. The mean temperature rise was 3.5 °C in the SDFT and 2.5 °C in the DDFT after 10 minutes of application using 1.0 W/cm^2 at 3.3 MHz with a continuous wave. No significant increase in heat production occurred when the intensity was increased to 1.5 W/cm^2. Cooling of the SDFT and DDFT occurred at 2.5 and 1.5 minutes, respectively.[134] Similar results were found in a canine study, which showed that cooling of the caudal thigh muscle after therapeutic ultrasound occurred in less than 10 minutes.[120] Therefore, if stretching is to be performed following heating of the tissues, it should be performed immediately after application of therapeutic ultrasound.

A 20-minute treatment was applied to the epaxial musculature of horses using an intensity of 1.5 W/cm^2 at 3.3 MHz. Tissue temperature was measured at three different depths – significant differences in mean temperature rise were seen when comparing 1-cm versus 4-cm depths and 1-cm versus 8-cm depths. No difference was seen between 4-cm and 8-cm depths.[134] While therapeutic ultrasound was ineffective at heating the deeper epaxial muscle, the use of a frequency of 3.3 versus 1 MHz likely resulted in a lack of penetration of the ultrasound waves, as acknowledged by the authors. Therefore, further studies are needed to determine if therapeutic ultrasound can heat deeper tissues. Additionally, although a statistically significant difference was seen between 1-cm and 4-cm depths, the rise in tissue temperature at 1 cm was only 1.6°F, and therefore unlikely to be clinically significant.[134]

Radiofrequency Diathermy

Mechanism of Action

Microwave and shortwave radiofrequency radiation is used to heat tissues situated deep in the body while minimizing a rise in skin temperature. Microwave

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diathermy units operate at a frequency higher than that used for shortwave diathermy. Heating occurs by production of electromagnetic radiation that is absorbed in the tissues, creating vibrational energy that results in heat production. Tissues with high water content absorb microwaves strongly with muscle and joint effusion heated preferentially.[59] The tissue effects are determined by the degree of tissue heating that occurs, similar to that described for therapeutic ultrasound above.

Evidence in Humans

Radiofrequency diathermy has been shown to significantly improve shoulder pain, range of motion, and better performance in activities of daily living in people with frozen shoulder.[119] In addition, radiofrequency diathermy had a small but significant effect on pain and muscle performance in patients with knee osteoarthritis.[114] However, another study demonstrated no additional benefit of radiofrequency diathermy for knee osteoarthritis when isokinetic exercises were performed.[3] This modality must be used with caution, as negative effects, such as burns/scalding at the application site and teratogenic effects in pregnant animal species have been described. Radiofrequency diathermy is contraindicated in humans with metal implants (excessive heating can occur) and those with a cardiac pacemaker.[35]

Evidence in Horses

Currently, no studies are available in the horse.

Laser Therapy

Mechanism of Action

Light amplification by stimulated emission of radiation (laser) is created when energy from a power source excites a lasing medium. Energy is then released as photons. The laser medium typically creates light with one wavelength (monochromatic). Biostimulating lasers are those used in our rehabilitation patients and are categorized into classes 1–4.[79,166] Class 1 and 2 lasers have no adverse effects on eyes and skin (laser pointers, laser used for laser disc). Class 3 lasers are those used for low-level laser therapy (LLLT) that operate at a power level <500 mW and include mid-power lasers that are safe on the skin but carry a risk for eye injury (cold lasers used for therapeutic purposes).[17] Class 3 lasers are further subcategorized into Class 3a and 3b. Class 3a lasers consist of lasers from 1 to 5 mW in power and pose moderate ocular hazard. Class 3b lasers consist of lasers from 5 to 500 mW power output and pose greater ocular hazard than Class 3a.[181] Class 4 lasers are used for high-intensity laser therapy (HILT) and carry a risk to both eyes and skin (used for therapeutic purposes).[17,79] The effect that occurs in the tissue depends on the wavelength and power that is used, as well as the absorption potential of the tissue itself.[17]

Evidence in Humans and Laboratory Animals

Both LLLT and HILT have been shown to reduce musculoskeletal pain, increase range of motion, and reduce neuropathic pain.[1,48,186] Laser therapy has been shown to accelerate tissue healing by improving fibroblast function, enhancing local microcirculation and oxygen supply, aiding in the removal of waste products,[1] increasing neovascularization, and promoting angiogenesis.[33] In addition, laser therapy significantly reduces inflammation by decreasing the production of prostaglandins, TNF-alpha, IL-1beta, plasminogen activator, and cyclooxygenase.[124] Laser therapy has also been demonstrated to improve motor and sensory nerve conduction by enhancing myelin production,[123,195] as well as improving collagen fiber alignment within injured tendons.[87]

There is also evidence that laser therapy may improve muscle strength and recovery in human athletes. LLLT applied to people before an eccentric training session improved muscular strength gain and the hypertrophic muscle response.[13] LLLT performed in elderly women resulted in increased number of knee flexion–extension repetitions compared with those who received sham treatment.[197] In a study of university athletes with a variety of musculoskeletal injuries, those who received LLLT had a significantly shorter return-to-play duration than those who did not receive laser therapy.[54]

HILT may be more effective than LLLT for certain conditions. HILT is able to reach and stimulate large and/or deep structures that are difficult to reach with LLLT. In a study comparing HILT versus LLLT for patients with knee osteoarthritis, HILT was more effective than LLLT at reducing pain after 6 weeks of treatment.[104] HILT was also shown to significantly increase range of motion, decrease pain, and improve functional disability in patients with cervical and shoulder pain.[5,153] For patients with chronic low back pain, HILT combined with exercise was more effective at reducing pain and improving function than exercise and HILT alone.[4]

Evidence in Horses

Laser therapy in horses has not been as extensively researched. However, studies show that equine patient preparation is essential to having adequate laser energy penetration. Clipping and cleaning of the limb results in significantly greater energy penetration than in unclipped limbs.[43,168] In horses with unclipped hair, only white or cream-colored hair allowed laser energy to penetrate through equine skin. In horses with gray, brown, chestnut, and black hair, >99% of laser energy is blocked if unclipped. Better penetration occurs in all horses after clipping; however, energy penetration is still greatest in nonpigmented skin and is greater for medium-colored than dark-colored skin. While a longer wavelength is meant to result in greater energy penetration, skin color has been shown to have a greater effect. In horses with moderate/dark pigmented skin, laser energy penetration was greater at a wavelength of 980 nm compared with 800 nm. However, in horses with nonpigmented skin, energy penetration was greater at 800 nm than at 980 nm.[43] Therefore, adjusting laser setting based on skin pigmentation is critical if targeting deeper tissues.

One retrospective equine study is available showing the benefit of laser therapy in horses with tendinopathy/desmopathy. In a study of 150 sport horses with tendinopathy/desmopathy of the SDFT, deep digital flexor tendon (DDFT), and suspensory ligament (SL), lameness, and ultrasound scores were significantly improved

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after 2 weeks of HILT.[154] A single study of chronic back pain in horses that applied laser therapy (904 nm) over 9 acupuncture points weekly demonstrated that clinical signs of back pain were alleviated in 10 of 14 horses.[71]

In addition, laser therapy may enhance stem cell applications. In an *in vitro* study using equine mesenchymal stem cells combined with the addition of growth factors, LLLT influenced the differentiation of stem cells toward the tenogenic fate.[64] An additional *in vitro* study evaluated the safety of using HILT with stem cells. This study showed no negative effects of HILT on the stem cells *in vitro*, and stem cells showed increased expression of IL-10, which has anti-inflammatory action, and VEGF, which positively regulates angiogenesis.[145] However, caution should be taken in combining laser therapy with stem cells until *in vivo* studies have been performed.

Transcutaneous Electrical Nerve Stimulation

Mechanism of Action

Transcutaneous electrical nerve stimulation (TENS) uses electrical current applied via surface electrodes to preferentially stimulate peripheral nerves and is used primarily for pain modulation.[34,95] There are two proposed mechanisms for the antinociceptive effects of TENS:

1. Gate control theory: TENS stimulates large, myelinated A-beta fibers that are stimulated during movement. These fibers synapse within the spinal cord at the same location as small, unmyelinated C-fibers that transmit pain. Because the A-beta fibers are large and myelinated, their signal transmits faster than the signal from C-fibers, thereby blocking pain.
2. Opiate-mediated pain control: TENS stimulates release of endorphins and their precursors into the cerebrospinal fluid. Three applications of TENS have been described: (1) conventional TENS, which consists of application of high-frequency, low-intensity current; (2) acupuncture-like TENS, which consists of application of low-frequency, high-intensity current; and (3) intense TENS, which consists of application of a high-frequency, high-intensity current and is typically painful in its application.[95]

Evidence in Humans

TENS is commonly recommended for use in patients with chronic pain in addition to medication. Benefits have been reported in 60% of patients during the first few months of use. However, this effect is noted to diminish over time.[34] Conflicting results are present when evaluating the ability of TENS to diminish pain. In some studies, TENS has been shown to relieve chronic musculoskeletal pain compared with placebo. TENS has also been shown to alleviate procedural pain during dental procedures (minor tooth extractions, cavity preparations, intraoral injections) and to reduce postoperative pain, analgesic consumption, nausea/vomiting, and speed of recovery.[95] TENS was shown to be effective for reducing acute postoperative pain if electrodes were placed close to the incision and a strong, definite, subnoxious (maximal tolerable) sensation was achieved.[34] However, in a randomized study evaluating the use of TENS for chronic lower back pain, there was lack of support for its use even when patients were educated of how to correctly use the TENS unit.[56] In addition, conflicting results are present when evaluating pain modulation for rotator cuff tears, sciatica, and fibromyalgia.[34] Use of TENS is contraindicated for patients with cardiac conditions, pregnancy, and epilepsy.[34,95]

Evidence in Horses

While there is no evidence of the effectiveness of TENS use in horses, there may be some overlap in the mechanisms of action, clinical indications, and similar effects reported for electroacupuncture.[176] If used on the horse, it is recommended to clip the hair and apply a small amount of ultrasound gel to the application pads in order to maximize contact.

Neuromuscular Electrical Stimulation

Mechanism of Action

Neuromuscular electrical stimulation (NMES) uses a low-level electrical current that through stimulation of the alpha-motor neurons allows recruitment and muscle contraction after orthopedic or neurological injury. NMES therapy has been successfully used by human physiotherapists to increase muscle strength, maintain muscle mass during prolonged periods of immobilization, and control edema after injury. This therapy assists neuromuscular function by enhancing the force capacity, or the ability of the muscle to contract, as compared with a true strengthening of the muscle. It is unclear if the role of electrical stimulation in improving muscle function is actually related to increased muscle strength, improved voluntary contractions, restoring motor control, or possibly due to proprioceptive activation within injured or atrophied myofascial tissues.[140,174]

Evidence in Humans

The combination of electrical stimulation and exercise has been reported to be effective in alleviating pain and improving voluntary activation in human osteoarthritis patients, but in one study did not enhance muscle strength or functional performance. However, other studies showed significantly improved muscle strength and functional performance following NMES(langeard 17,son14,salazar18).[113,169,185] The use of NMES can also aid in the reduction of edema and swelling as the direct current drives the charged plasma protein ions within the interstitial spaces to move in the direction of the oppositely charged electrode, facilitating movement into the lymphatic channels.[20]

Evidence in Horses

The difficulty in applying this modality to the horse is the lack of standardized protocols and no validated treatment programs due to large variations in methodology that include treatment parameters, the frequency and duration of treatment, the disease stage and severity, and patient selection. One recent equine study used functional electrical stimulation in horses with induced

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injury to the recurrent laryngeal nerve and cricoarytenoid muscle. Horses that received electrical stimulation showed improved muscle strength and geometry and had an increased type I:II fiber proportion than controls.[23] NMES is typically conducted 3–5 times a week, inducing 8–15 contractions within a treatment session. The electrodes are placed in a bipolar fashion with the muscle at resting length or slightly lengthened at the time of electrode placement. The rest cycle should be 5–6 times as long as the contraction, typically in a 1:6 ratio. The hair coat may need to be clipped depending on length and should be cleaned of dirt and debris with water and coupling gel applied.

Pulsed Electromagnetic Field Therapy

Mechanism of Action

Pulsed electromagnetic field (PEMF) therapy is created when an electrical current is driven through a coiled wire that generates a magnetic field. This magnetic field creates small currents inside the tissue. PEMF utilizes a low-frequency and short-phase duration, which results in a current generated within the tissues without heat production.[83,166] PEMF is most commonly used to aide in bone healing. When bone deforms during weight-bearing, it becomes electrically polarized and generates a very low electrical current. This explains Wolff's law of functional adaptation within bone, in which this electrical signal regulates the osseous elements and maintains bone mass.[174] PEMF also generates a weak electric current in the tissue similar to those generated by bone in response to mechanical deformation, thus stimulating osteogenesis.[109,220] PEMF has also been shown to help reduce pain and inflammation, mainly through its stimulation of nitric oxide, which results in vasodilation and enhanced circulation.[180]

Evidence in Humans and Laboratory Animals

PEMF is approved by the FDA for treatment in humans with long-bone fracture nonunions and as an adjunct treatment following lumbar/cervical spine fusion surgery.[198] PEMF has been shown to accelerate fracture healing in both skeletally mature normal rats and in rats with induced osteoporosis.[9] PEMF significantly improved the success rate in people with delayed union long-bone fractures with 77.4% of the PEMF-treated fractures healing compared with 48.1% in the sham group at 4 months.[180] PEMF has shown to increase bone volume fraction, trabecular thickness, trabecular number, and suppress trabecular separation in a rat model of induced knee osteoarthritis. In this same study, PEMF was shown to promote WNT gene expression in the subchondral bone. Activation of WNT promotes osteoblast and osteocyte activity while indirectly suppressing bone resorption and osteoclast differentiation.[217] In a rat model of rotator cuff tearing and repair, PEMF improved early tendon healing and bone quality.[198] PEMF has also been shown to reduce pain in patients with low back pain;[49,187] however caution must be taken in interpreting these findings, as in some studies patients were not blinded to treatment or no control group was present.[88,173] One study showed PEMF was not effective in reducing pain, stiffness, or improving function in women with fibromyalgia.[136] However, more information on device settings for specific conditions is needed. One human study showed that a small change in the dose of PEMF energy applied had a significant effect on patient outcome.[187]

Evidence in Horses

Limited equine studies have been performed. One study from 1987 looked at the effect of PEMF for incorporation of cancellous bone grafts in ponies and found a small but statistically significant positive effect.[109] A more recent canine study showed significantly faster recovery of load bearing, significantly increased new-bone formation, and higher mechanical strength of healing in dogs treated with PEMF following mid-diaphyseal tibial osteotomy.[91] An equine study assessing tendon healing conducted in 1985 evaluated use of PEMF in surgically created defects in the SDFT. This study showed no significant differences in PEMF vs control limbs for tendon enlargement or peritendinous fibrous reaction. Interestingly, the PEMF-treated tendons showed delayed transformation of collagen from immature to mature fibers, increased vascularity, and more poorly organized and differentiated cells compared with controls. This study suggests that PEMF may actually delay tendon healing.[208] However, significantly more equine research is needed to determine the efficacy of PEMF for a variety of conditions. Given the wide variability in available device parameters,[57] specific information regarding device settings is necessary.

Underwater Treadmill

Mechanism of Action

Underwater treadmill (UWT) training has been utilized in humans and horses for rehabilitation of acute and chronic injuries.[99,106,107] Underwater treadmill training is used because of effects from multiple water characteristics, including buoyancy, hydrostatic pressure, viscosity, drag force, and temperature.[99,106] The effect of buoyancy decreases the vertical ground reaction force[137] and allows partial weight-bearing, which alleviates stress and pain on the lower extremity joints. It also allows patients to train to improve gait when they may be deficient in muscle strength, postural control, and motor coordination.[99] Hydrostatic pressure and water viscosity provide postural support to patients with impaired balance or motor control[99,178] while the drag force (resistance) of the water allows functional strength training.[130]

Underwater resistance has been shown to be 12 times greater than over ground resistance.[117] Water temperature can be utilized to provide one of two effects – warm water can help improve the level of comfort during training,[67] while use of cold water provides cryotherapy effects, including reduced pain and swelling.[105] Decreased pain has also been suggested to occur due to stimulation of sensory receptors by the water.[129]

Evidence in Humans

Underwater treadmill training has been shown to reduce body weight by 50% with the water level that is

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at the waist, 70%–75% with the water level at the chest, and 90% with the water level at the neck.[111] The effect of water depth has also been shown to affect stride length, stride cadence, and range of motion. Increased stride length and decreased stride cadence were seen with water at the level of the neck compared with water at the level of the waist and chest. An increase in ankle range of motion and a decrease in hip range of motion were seen as the water level was progressively increased.[99] Underwater treadmill training has also been shown to increase muscle strength[117,189] and improve balance.[115,189]

Evidence in Horses

Underwater treadmill training has been shown to have significant effects in horses with experimentally induced carpal osteoarthritis. In the horses undergoing UWT training compared with those walked on an overground treadmill, a significant decrease in synovial membrane inflammation was seen. In addition, the UWT group showed significant improvements in symmetric limb loading, muscle activation, and joint range of motion[107] indicating a benefit of UWT training in horses with carpal osteoarthritis. Underwater treadmill training was also shown to significantly improve postural sway characteristics compared with horses exercised on an overground treadmill with carpal osteoarthritis.[106] However, UWT training was shown to have no significant effect in adaptation of the superficial digital flexor (SDF) and gluteal muscles or muscle metabolic response compared with horses undergoing overground treadmill training.[53]

As in humans, water depth has also been shown to have a significant effect on joint range of motion as well as limb protraction/retraction. The greatest range of motion of the carpus has been shown to occur with the water at the level of the tarsus. The greatest range of motion of the tarsus occurs with the water at the level of the stifle, while the greatest range of motion of the metacarpo/tarsophalangeal joints occurs with the water at the level of the metatarsophalangeal joint and tarsus.[129] Overall forelimb range of motion has been shown to be the lowest with the water at the level of the stifle. While the greatest overall hind limb range of motion occurs at the level of the stifle. Walking in the UWT resulted in lower forelimb protraction with the water at the level of the tarsus and stifle compared with dry treadmill. Hind limb retraction was significantly greater in the UWT compared with dry treadmill, and retraction was greater with water at the tarsus and stifle compared with the fetlock in the UWT. A slower walking speed on the UWT resulted in decreased forelimb and increased hind limb protraction–retraction compared with a normal walking speed.[138] Increased overall flexion–extension of the back and lumbar flexion occurred as the water depth is increased to the level of the stifle. Horses showed increased cranial thoracic extension and increased thoracolumbar flexion in higher water depths.[139] Because of this, horses with back and sacroiliac joint pain should be monitored closely for worsening discomfort if undergoing UWT training at high water depths. In addition, UWT training should be used with caution in horses with respiratory and cardiovascular disease. The increased hydrostatic pressure has effects on lung volume, which can result in inadequate ventilation.[101] A higher water depth also results in increased VO_2, tidal volume, and heart rate.[149] Underwater treadmill training should also be avoided in horses with surgical incisions, open wounds, fever, myositis, and in animals that are fearful or panicky.[105]

Whole Body Vibration Therapy

Mechanism of Action

Whole body vibration (WBV) therapy is performed with the goal of improving muscle and core strength. In addition, vibration stimuli provide mechanical loading that is necessary to facilitate mechanotransduction in bone and thus osteogenesis. The transmission of mechanical oscillations initiates rapidly and repeating eccentric and concentric muscular action through activation of alpha-motor neurons.[12,22,163] These oscillations lead to physiological changes by stimulation of skin receptors, muscle spindle cells, and joint mechanoreceptors.[163]

Evidence in Humans and Laboratory Animals

WBV therapy has been shown to improve muscle strength,[12,47,98,156] as well as improve postural stability [40,66,160] and power.[46,98,216] WBV therapy has also been shown to improve bone strength in humans[47,66,160] and improve fracture healing in a sheep model.[121] Peripheral blood flow and tissue oxygenation in people have been shown to be positively influenced by WBV. The mechanical oscillations transferred from the vibration plate to elderly individuals have improved postural control and thus have reduced the risk of falling within the elderly population. Furthermore, there is modest evidence that supports the use of vibration therapy to reduce pain perception in osteoarthritic individuals. Lastly, there is an emerging profile for application of vibration therapy as an exercise modality for people. Vibration training for human athletes appears to have a rapid and energy sparring warm-up effect and has demonstrated increases in jump height and muscle power.[53,115,138,189] However, other studies demonstrate no change in bone density or microstructure after patients used a low-magnitude vibration plate for 6 months.[215] In addition, the timing of application of WBV therapy has been shown to be important. In one study, increased upper body power was seen 10 minutes after cessation of WBV therapy; however, decreased power was seen 1 minute after cessation of WBV therapy.[98] Therefore, exercising immediately after WBV therapy may be detrimental.

Additional detrimental effects of WBV therapy may be seen in patients with osteoarthritis. In a rat model of knee osteoarthritis, rats treated with WBV therapy showed accelerated cartilage degeneration and further functional deterioration than control.[158] Changes in machine parameters (i.e. frequency and oscillation type) have been shown to have significant effects;[7] therefore, exact parameters need to be determined for specific conditions. For example, both closed and open fracture repair models in rodents demonstrate that vertical vibration at a frequency of 45 Hz negatively impairs fracture healing, while vibration frequencies of 35 and 50 Hz enhance fracture healing. In contrast, horizontal oscillatory vibration therapy regardless of frequency demonstrates no positive or negative effects on fracture healing in rodent models.[110,210] A similar study found

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no significant difference between application of horizontal versus vertical vibration on jumping force.[12]

Evidence in Horses

A translational comparison within the human literature to the horse becomes very difficult as a number of different WBV platforms are commercially available. Anecdotally, WBV has been applied to horses with various claims of effectiveness. Acute hematologic and clinical effects of horses undergoing alternating horizontal and vertical vibration therapy have been recently described, noting no adverse effects following vibration sessions exposed to a frequency of 15–21 Hz.[22,68] Within the rehabilitative setting, there has been recent interest in the effects of prolonged vibration therapy on the cross-sectional area and symmetry of the multifidus muscle. A significant increase in multifidus muscle cross-sectional size and symmetry was found following 60 days of twice daily, 30 minute, 40 Hz vertical WBV sessions. Known for its role in spinal stabilization and postural muscle acuity, development of the multifidus muscle is thought to have potential as an osteoarthritis deterrent.[85] Only one study has been conducted in the horse evaluating the effects of vibration therapy on bone density in stalled patients. Twelve horses were confined to stalls for 60 days with half the group exercised daily on a mechanical walker, and the others underwent vertical WBV therapy at 50 Hz for 45 minutes, 5 days a week. Whole body vibration therapy in stalled horses maintained the same bone mineral content to that of horses that received daily light exercise and therefore should be considered for horses restricted to stall rest only.[85] However, to date no direct comparison studies on vibration direction, frequency, duration, or magnitude in the horse have been conducted.

CONCLUSION

Although there is currently relatively limited scientific evidence regarding rehabilitation strategies and PT in equine veterinary medicine, their use is rapidly expanding. Research and documented clinical experiences are still needed to complement the adaptation of the scientific-based principles presented in this section. Nevertheless, it is the author's opinion and clinical experience that rehabilitation/PT strategies can be successfully incorporated in conjunction with traditional veterinary medicine. A team approach with the necessary clinical reasoning skills is vital in choosing the appropriate rehabilitation strategy and PT technique and monitoring the patient's progress. Constant reassessment and, where possible, objective measures to validate the outcome of the interventions are indicated for successful treatment strategies involving PT and rehabilitative techniques.

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9

CHAPTER

Occupational-Related Lameness Conditions

THE THOROUGHBRED RACEHORSE

Ryan Carpenter

The incidence and impact of lameness in Thoroughbred racehorses is significant. Essentially all horses participating in training and racing are vulnerable to injury associated with varying degrees of lameness. In one review, 37 of 40 juvenile horses became lame during training.[48] Failure to train due to lameness has a significantly detrimental economic impact. Monetary considerations, coupled with the recent increased public awareness focused on the welfare of horses in general, have resulted in a new era of philosophy in lameness management in the Thoroughbred racehorse. As a result, there is marked emphasis on injury prevention, early recognition of lameness, and early proactive intervention prior to the onset of serious lameness. It is important to be able to differentiate potentially life-threatening disorders from those that are transient or pose minimal threat to the horse's well-being while continuing to compete.

As with any sport, occupational-related injuries from racing and training are prevalent, and several etiologic factors have been incriminated in these types of injuries. Variables include genetics, racetrack surface, number of starts, age of the horse, preexisting pathology, conformation, and trauma. In the Thoroughbred racehorse, stress remodeling and stress fractures are the most common cause of lameness. Only occasionally, these injuries result from a single event that stresses the bone or tissue beyond its ultimate strength. More commonly these injuries are a result of repetitive overuse that results in cumulative fatigue failure of the bone or tissue. Therefore it is important to recognize when the balance may be tipped toward ultimate tissue failure. Balanced stress remodeling is critical in the longevity of the horse's athletic career. Ideally every stress-related lameness would be identified prior to tissue failure allowing an alteration in the horse's training program. Therefore, the focus of this discussion will be to identify the most common causes of lameness in the Thoroughbred racehorse, with an emphasis on how to manage each case with an appropriate plan to help minimize the number of days lost to training and to prevent the progression to a more serious injury or catastrophic failure.

RISK FACTORS FOR INJURY

Emphasis has been given to the identification of risk factors for development of musculoskeletal injury, especially those that are fatal.[11,15,38,43,44,57] As noted, the occurrence of lameness is extremely high. Fortunately, the incidence of catastrophic breakdown in racehorses is very low; approximately 1–2 catastrophic events occur per every 1,000 starts.[23] The 2016 Jockey Club reported a declining number of catastrophic events to the lowest level since 2009. The total number of catastrophic events in 2016 was 1.54 per every 1,000 starts. Dirt, synthetic, and turf surfaces accounted for 1.70, 1.14, and 1.09 catastrophic events, respectively.

Several postmortem studies have categorized injuries that result in destruction of the horse. These studies have elucidated not only training factors that appear to predispose racehorses to specific injuries but also apparent geographic differences. For example, the incidence of soft tissue injuries incurred during racing differs among various racetracks in the United States. One Australian study

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of fatal injuries in Thoroughbred racehorses found that most were euthanized after a catastrophic injury to a forelimb; however, there was no statistically significant difference between forelimb affected.[6] Conversely, there was a predisposition for right forelimb injury in the United Kingdom.[44] In the United States, horses were more likely to sustain fracture of the left forelimb during racing and right forelimb during training.[29] Fractures of the proximal sesamoid bones were the most common injury in the United States, but Australian studies showed that fracture of the front or rear cannon bone was most common during racing, followed by accompanying fractures of the proximal sesamoid bones and proximal phalanx.[6,7]

Training and racing schedules have been scrutinized.[2,11–13,17–20,26] The risk of catastrophic injury and forced lay-up in fit California Thoroughbred racehorses was significantly increased following 2 months of high-speed exercise with distance accumulation.[17] Two-year-olds exceeding 0.76 furlongs/day, 3-year-olds exceeding 0.85 furlongs/day, and 4-year-olds exceeding 0.95 furlongs/day were at higher risk of sustaining injury than those working shorter distances. In another study, the racing injury rates were inversely proportional to the success of the individual trainer.[59] The incidence of tibial and pelvic stress fractures rises with increasing distances cantered during a 30-day training period but not over a 60-day period.[40,58]

Track conditions also correlate with injury.[10,46] Fracture rates in Japan increased as dirt tracks became muddier and decreased as turf surfaces became softer.[41] The differences between synthetic surfaces, dirt, and turf courses are still under investigation. One study showed a greater proportion of stress fractures detected on scintigraphic examinations from horses training on a synthetic surface (31.7%) compared with horses training on a dirt surface (23.0%) at an earlier point in time. Additionally, there were a greater proportion of pelvic and tibial stress fractures diagnosed in horses from a synthetic surface-trained group than from the dirt-trained group.[34]

Individual conformation has been recognized clinically to affect locomotion and long-term soundness.[36,37] Some traits have been shown to influence the onset of clinical abnormalities. For example, horses with offset knees have an increased incidence of stress-related disorders of the knees and an increased risk of fetlock problems.[36] Other flaws that likely predispose a horse to injury include back-in-the-knee conformation and carpal or fetlock varus deviations.

DIAGNOSIS OF LAMENESS

History and physical evaluation, economics, and personal experiences help determine the appropriate diagnostic steps for every horse. Knowledge of the exercise and racing schedule, including when fast work has taken place, intensity level of training, and past performance is important. It is also important to be aware of recent lay-up, convalescence, and previous surgery.

The physical evaluation is straightforward and basic, but it must be thorough. It is especially helpful if the veterinarian is familiar with the patient. The general health of the horse must be considered, including appetite, hair coat, general disposition, and the presence of any current medical conditions as well as treatment thereof. Recognition of previous lameness in the specific patient coupled with results from current diagnostic procedures may raise the level of suspicion for a recurrent injury.

Racetrack practitioners who are familiar with their patients have a number of advantages, including the ability to perform multiple examinations either in hand or on the racetrack in the horse's routine work environment. A disadvantage of known history is the potential for bias in diagnoses. Another disadvantage for the racetrack practitioner is obstacles in the working environment that may preclude a thorough locomotor examination. Special scheduling may be required to accomplish a complete examination. The behavior of a fit racehorse likewise limits the time allowed for the evaluation, in addition to presenting an inherent safety hazard. Many racehorses become rank and difficult to handle after only a few jogging sessions and may hide a subtle lameness due to excitement. Diagnosis of a new injury may be challenging if there are preexisting healed injuries that are not clinically active.

Lameness may be displayed in a broad spectrum of clinical signs, ranging from behavioral changes, reluctance to train, and poor performance to mild or overt gait alterations. It is common for horses to display changes in temperament, such as aggressive or passive behavior, prior to displaying actual lameness. Horses may become track sour or display changes during training such as lugging in or out or running off. Other manifestations include reduced appetite, poor hair coat, soft palate displacement, or tying up.

Lameness associated with pain is the manifestation of an avoidance response. The character of lameness is largely determined by the location or source of an injury, although it is difficult to differentiate exact foci of pain or specific disorders based on gait alone. Generalities of gait combined with the rest of the clinical picture often aid in lesion localization, but there are no absolutes based solely on gait.

Gait alterations associated with mechanical limitations may be challenging to distinguish from lameness associated with pain. An example of characteristic gait patterns is a delay in contact of the foot with the ground (drift) caused by a foot bruise or developing abscess in a front foot; a similar problem in a hindfoot produces a stringhalt appearance. Abduction of a limb prior to ground contact is often observed with a fracture of the lateral wing of the coffin bone, incomplete lateral condylar fracture, and fracture of the lateral aspect of the carpus. Changes in advancement of a limb and stride length occur with many lameness conditions and vary with location and severity. Horses with a humeral stress fracture, high suspensory desmitis, or other proximal limb injury do not advance the limb fully in the cranial phase of the stride. Tibial stress fractures often display a limited contact or support phase of the stride, which results in a jerking lift-off of the limb in addition to a limited cranial phase of the stride.

Diagnosis of lameness should be confirmed by isolating the source of lameness. Ancillary diagnostic anesthesia may be employed to localize the pain if necessary and feasible. Diagnostic nerve blocks may be difficult to perform for two reasons: the fractious behavior of some

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horses and potential regulatory hurdles if the horse is scheduled to compete. Caution is imperative in the racehorse if the possibility of anesthetizing a potential fracture exists. The results of local anesthesia are sometimes open to interpretation, due to the ineffectiveness of the block and the time frame required for anesthesia to develop.

For example, a horse with proximal suspensory desmitis will occasionally improve to diagnostic anesthesia of the middle carpal joint. Results may also be confused because excitement of the horse can obscure the lameness independent of the regional anesthesia.

Further ancillary diagnostic techniques include imaging modalities such as radiographs, ultrasound, nuclear scintigraphy, MRI, and computed tomography. It is routine for racetrack practitioners to be equipped with portable digital radiographic and ultrasonographic equipment.[5] Instant stall side diagnosis of fracture or other injury has become standard.

Nuclear scintigraphy has had a tremendous impact on understanding bone disease in the racehorse and the management of many of these conditions. Because it provides a reflection of the physiologic status of tissue, pathology may be detected and therefore addressed earlier than with plain radiography. For example, detection of stress-related bone injuries may be observed before catastrophic failure occurs.

COMMON CONDITIONS

An almost endless number of clinical problems resulting in lameness regularly confront the racetrack practitioner. Certain problems occur routinely and become standard and expected, while other conditions are uncommon and may be overlooked because of their relative infrequence. As more information is gained with advancement of studies, our knowledge base and future management of these conditions may be subject to change.

Foot

The foot of the racehorse is a common region affected by lameness. It is standard belief that the hoof of the North American Thoroughbred is inferior to that of the European Thoroughbred due to less mass. The front feet of the average racehorse undergo conformational changes as it progresses through its training and racing career. It is common to develop a long toe and an underslung heel with the axis of the foot becoming reoriented in a broken back fashion. Improper shoeing is often implicated; however, these changes are also the result of the mechanical forces on the foot associated with training. These conformational changes combined with the trauma to which the foot is subjected contribute to many lameness conditions in the foot.

The most frequent problem is bruising in the subsolar region, heels, or frog. Treatment for bruising is to rest and protect the region, remove the offending load responsible for the bruising, and prevent abscessation. Generally little training is missed as a result of a foot bruise because most are self-limiting problems; however, abscessation may occur if sepsis occurs. Abscessation is treated with plain hot water soaks and a softening hoof pack (Animalintex) until the horn is pliant enough to allow eruption of the abscess. Most resolve uneventfully and carry a good prognosis if managed with proper hygiene and rest.

Quarter cracks occur less commonly but often enough to be a particular nuisance. They almost always occur in horses that have shearing of the heels, thin walls, an unbalanced foot, and often underslung heels, and males seem to be more affected than females. The side with the crack is usually taller than the unaffected side. An integral component of management involves balancing the foot in addition to stabilizing the wall. Without balancing the foot, repair of the crack is futile and it will recur. A support shoe such as a Z-bar shoe or a three-quarter shoe with a Z bar is helpful at lessening the shearing forces of the involved wall while healing occurs. Direct repair of the crack by lacing and/or patching is often necessary to stabilize the hoof wall. The crack must be dry and free of sepsis, and if there is sensitive tissue involved, a drain must be placed beneath the crack. Many horses are able to continue training while undergoing treatment for quarter cracks, while others must be convalesced to allow hoof regrowth and conditioning. The farther palmar the crack is located on the foot, the more difficult it is to manage.

Six types of coffin bone fractures are described in the literature and occur in racehorses, but the most common are fractures of the lateral wing of the left forelimb and medial wing of the right forelimb (Figure 9.1).[27,49] These fractures may be recognized with nuclear scintigraphy prior to the appearance of radiographic changes, suggesting a stress-related bone injury as the underlying



Figure 9.1. Forty-five-degree dorsoproximal palmarodistal radiograph illustrating a wing fracture of the third phalanx.

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cause.[49] Fractures may not be apparent on radiographs until 7–10 days after they occur. Conventional management includes rest with a bar shoe with or without clips for 8–12 weeks. If lameness is still evident it is common to perform a palmar digital neurectomy; however, some racing jurisdictions do not allow the horse to race following this procedure. There is usually a good prognosis for return to racing, even though there is commonly evidence of osteoarthritis (OA) or a step defect once healing is complete. Unfortunately, horses with type III coffin bone fractures with significant displacement or fractures of the navicular bone have a poor prognosis to return to racing.

Pastern

Lameness associated with the pastern region is relatively uncommon, but fractures of the proximal phalanx (P1) occur frequently. The most commonly reported fracture configuration is parasagittal, extending distally from the sagittal groove of the proximal articular surface of the bone. As the fracture courses distally, it most commonly diverges laterally before either exiting the lateral cortex of the bone or spiraling into an oblique (dorsolateral/palmar/plantaromedial) plane approaching the distal epiphysis and proximal interphalangeal joint. Long (>30 mm) incomplete parasagittal fractures are the most frequent fracture configuration and commonly affect the forelimbs more than the hindlimbs.[53] Typically lameness is very apparent; however, soft tissue swelling is often minimal, and varying degrees of fetlock joint effusion may be present. Therefore, if a pastern fracture is suspected, radiographic evaluation is recommended prior to diagnostic anesthesia as it is possible to block a pastern fracture with palmar digital anesthesia. The fracture often extends more distally in the weeks' post-injury suggesting that these fractures are more extensive than initially appreciated at the time of injury.[53] Incomplete parasagittal fractures heal very well with internal fixation and have an excellent prognosis for return to athletic use. However, complete biarticular parasagittal fractures typically have a much lower prognosis. Although the parasagittal configuration is by far the most common fracture configuration seen, almost any fracture configuration can occasionally be seen.

Fetlock

The fetlock is the single most common region affected by lameness in the TB racehorse. The fetlock joints of both the forelimbs and hindlimbs are plagued with multiple problems that contribute to lameness, and they are the most commonly affected articular structure in the racehorse. One report indicates that up to 56% of the total days lost in training as 2-year-olds are attributed to fetlock pathology.[4] Most are associated with some form of cumulative stress-related disease, which may involve the bone, cartilage, or soft tissue.

Synovitis and capsulitis of 2-year-old training horses are common and often self-limiting, as long as training is reduced and the tissue is permitted to heal.[35] The predominant clinical sign is joint effusion with or without heat. Lameness is mild if present, but there is usually resentment to flexion of the joint. Radiographs are unremarkable, whereas ultrasonography confirms increased joint fluid and mild thickening or proliferation of the synovium. Treatment includes local therapy with ice, hydrotherapy, poultice, and bandaging. Joint injection with chondroprotective agents and corticosteroids or IRAP is common. Systemic nonsteroidal anti-inflammatory drugs (NSAIDs) are also frequently administered.

Common areas of cartilage and osteochondral trauma involve the dorsal rim of P1, usually medially or less commonly medially and laterally. This is believed to occur from the repetitive trauma and wear of the rim against the distal metacarpus and is the most common area for development of chip fractures in the fetlock.[54] Usually there are joint effusion, warmth, and varying degrees of pain on flexion, along with focally sensitive palpation on the area of the rim that is involved. Lameness is usually only transient if present at all, unless advanced changes have occurred in the joint. Often there is a reduction in performance rather than an overt lameness. The prognosis is good with arthroscopic removal of dorsal P1 chip fractures in racehorses as long as appropriate convalescence is allowed.[14] Recuperation time averages 2 months or longer. Reports indicate that there is no difference in prognosis with different sizes of fragments, but larger fragments usually induce more cartilage damage and wear on the distal metacarpus, especially if they are longstanding.

Dorsal frontal fractures of P1 occur relatively frequently. These fractures commonly affect the hindlimbs although these can occur in forelimbs. There is no specific gait characteristic associated with this injury, but affected horses are often described as having a proximal limb lameness. Frequently this injury is confused with tibial stress fractures. Diagnosis is often made with nuclear scintigraphy and confirmed by radiographs. Unless extreme displacement occurs, which is infrequent, the treatment is rest for 60–90 days. Extracorporeal shockwave therapy (ESWT) can help promote healing, although these fractures heal consistently without treatment. There is a good prognosis for return to racing.

The proximal sesamoid bones and other components of the suspensory apparatus are subject to tremendous tensile forces and fatigue, especially when under full load at high velocity. Several fracture types occur in the proximal sesamoid bones, but apical fractures predominate. Other fracture types include midbody, basilar, abaxial, and comminuted fractures, but all occur less frequently than fractures of the apex (Figure 9.2).

Apical fractures are articular; therefore, joint effusion is usually observed if the fracture is displaced. There is pain on flexion and on focal pressure of the involved sesamoid. Diagnosis is confirmed radiographically, and there is a good prognosis for return to racing with forelimb involvement (67%) with an even better prognosis for return to racing for hindlimbs (83%).[51] The medial sesamoid of the front fetlock carries the worst prognosis; only 47% return to race.[51] A major determinant in the prognosis is the integrity and degree of damage of the suspensory ligament; therefore, ultrasound evaluation is critical.

Abaxial, transverse midbody, and basilar fractures occur less commonly and carry a guarded prognosis for future racing, with or without surgery. Surgical removal

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Figure 9.2. (A) Lateromedial radiographic view of the metacarpophalangeal joint, showing a displaced apical sesamoid fracture. (B) Lateromedial radiographic view of the metatarsophalangeal joint, showing a wedge-shaped basilar fracture fragment of a proximal sesamoid bone. (C) Dorsolateral palmaromedial oblique radiographic view of the metacarpophalangeal joint illustrating a suspensory ligament breakdown injury where comminuted fractures of both proximal sesamoid bones have occurred.

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of abaxial fragments is routinely performed. Generally, if marked displacement has occurred, there are usually excessive suspensory damage and a poor prognosis for return to racing. However, if there is minimal suspensory damage, surgical removal can have a favorable outcome to return to race, but only a fair prognosis for returning at the same level. In general, horses with fractures of the lateral sesamoid bone do better than those with fractures involving the medial sesamoid bone, and hindlimbs do better than forelimbs. Midbody fractures may be managed conservatively or with surgical treatment with lag screw fixation or circumferential wiring.[25] Contrary to literature reports, there is a guarded prognosis for future racing. One report that supports this demonstrated a 44% return to racing when lag screw fixation was elected and 0% when circumferential wiring was elected.[8] Radiographs often indicated that the fractures have completely healed; however, once back in training, lameness often occurred when the horse worked 5/8th of a mile and re-injury was common. Degenerative joint changes often accompanied these injuries and worsened the prognosis. With conservative management, horses usually become sound and are useful for breeding or light athletic use.

Stress-related bone disease of the distal metacarpus or metatarsus is an extremely common cause of lameness in juvenile horses and is also seen in older horses. The most common clinical manifestation is a variable severity of lameness, usually seen when cooling after a workout or on the following day. Nuclear scintigraphy often demonstrates intense focal uptake of radioisotope of the involved condyle. Radiographs generally show some degree of resorption of subchondral bone.[31] At this stage, conservative therapy yields a good prognosis. If continued training is allowed, resorption will progress to a fracture line. Lameness is usually evident at this point, and surgical intervention is necessary if there is any distraction at the fracture site (Figure 9.3). If initial radiographs only show subchondral resorption, it is imperative to perform follow-up radiographs in 3–4 weeks to assess development of a fracture line once resorption is complete. The metacarpi develop fractures of the lateral condyle more frequently than medial, while the metatarsi sustain medial and lateral fractures in approximately equally. There is a tendency for lateral condylar fractures to remain in a relatively simple configuration and exit the metacarpus laterally, whereas the medial fractures tend to spiral proximally and may exit medially or laterally. Prognosis is directly correlated with the extent of articular injury and if the fracture is displaced with concurrent palmar fragmentation or avulsion of the intersesamoidean ligament, resulting in an axial sesamoid bone fracture.



Figure 9.3. Dorsopalmar radiograph of a right front fetlock showing a complete displaced comminuted lateral condylar fracture of the third metacarpus.

Metacarpal Region

The third metacarpal bone is commonly subject to stress-related bone injury. The most common manifestation is dorsal cortical disease that ranges from mild bucked shins to dorsal cortical fracture.[39] Numerous configurations of bone reaction and resorption are recognized, including periosteal reaction, dorsal cortical thickening, vertical resorption lines in the dorsal cortex, and saucer fractures (Figure 9.4).

Diagnosis is based on clinical findings such as palpable sensitivity of the dorsal cortex while holding the limb in a non-weight-bearing frame. Typically the horse will jog very sore when wearing front bandages. Local anesthesia is occasionally necessary to isolate the lameness. Radiographs eventually detect periosteal new bone proliferation or bone resorption, but these changes may not be evident for several weeks. Treatment is based on the severity of the disease and degree of lameness, but the most common and effective treatment is reducing the intensity of training or rest. Ancillary treatments include shockwave therapy, needle periosteal scraping, osteostixis, and cortical screw placement.[9] In general, there is a good prognosis for future racing.

High suspensory lameness in the forelimb presents in a variety of different ways and can be a diagnostic challenge. Clinically these horses demonstrate a shortened cranial phase of the stride. Clinical exam can reveal heat, sensitivity to palpation, thickening of the suspensory ligament, or no abnormalities. Often mild effusion of the middle carpal joint will be noted without heat or sensitivity to palpation of the carpus, and this is believed to be due to referred inflammation. Diagnostic anesthesia of this region is critical followed by several diagnostic modalities to further clarify the disease. An ultrasound exam is important to rule out a primary desmitis or core lesion. If the ultrasound exam is normal and radiographs do not reveal any obvious bony changes, nuclear scintigraphy

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Figure 9.4. Lateromedial radiograph showing a dorsal cortical fracture of the third metacarpal bone.



Figure 9.5. Dorsolateral palmaromedial oblique radiographic view of the carpus showing a chip fracture of the distal radial carpal bone.

is indicated to determine the degree of bone involvement. When nuclear scintigraphy shows a mild IRU at the origin of the suspensory ligament, these cases respond very well to ESWT, topical Surpass, and Naquasone and can be maintained in active training. However, if there is marked IRU or if a core lesion is appreciated, then a prolonged rest and rehabilitation program is warranted. Proximal palmar cortical fractures may develop at the origin of the suspensory ligament and are a source of significant lameness. Two common types of bone injury occur in this region. The first is a circular resorptive region in which there is usually an avulsion of the suspensory ligament. The other type consists of palmar cortical vertical fractures, which typically do not involve the suspensory ligament. Suspensory injuries have a favorable prognosis with conservative management and monitoring to assess healing unless an avulsion fragment is present, which carries a guarded prognosis. A convalescence period of 3–6 months is usually sufficient for training to restart.

Fractures involving the splint bones are more common in the forelimbs than hindlimbs. Initially a focal swelling is present with heat and sensitivity to palpation. As training continues, a large focal firm swelling becomes evident. Initially an obvious fracture is difficult to appreciate on radiographs; however, based on the reactivity of the bone and periosteum, a fracture is almost always present. Unless the fracture is very distal, surgical removal is rarely indicated. Horses are removed from training, and the splint can be injected with corticosteroids, and sarapin or cryotherapy is useful if the periosteum is highly reactive. Horses are walked for 2–4 weeks before intense training is resumed. The prognosis is good and recurrence is rare.

Carpus

Carpal lameness is extremely common in Thoroughbred racehorses of all ages, especially in juveniles. The carpus is a complex joint that is susceptible to a multitude of injuries associated with training and racing. Stress-related pathology is involved in most of the bone, cartilage, and other soft tissue injuries of the carpus.

Common conditions include synovitis and/or capsulitis, cartilage wear and erosion, and stress-related bone injury of the third carpal bone. Numerous chip fractures of the radial carpal and middle carpal joints also occur (Figure 9.5).[42]

Clinical signs of carpal lameness vary depending on the disease process, but generally the horse tracks in a base-wide manner to facilitate lateral placement of the knee. The middle carpal joint is far more frequently affected than the proximal joint. Young horses with

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synovitis of the middle carpal joint display minimal lameness. The joint is often warm and there may be a change in behavior or performance. Radiographs are normal and the condition is self-limiting with a reduction in training. Joint injection is frequently performed using a corticosteroid or IRAP in conjunction with a chondroprotective agent. If the condition persists or recurs after treatment, a rest period of 30–60 days is indicated.

Third carpal bone disease is very common in juvenile thoroughbreds and is considered to be an integral component in the development of third carpal slab or sagittal fractures. Radiographs including a skyline projection of the third carpal bone should be performed and are diagnostic for third carpal disease. Common radiographic changes are sclerosis, bone resorption beginning at the radial facet, and slab and sagittal fractures (Figure 9.6).[21] Prognosis is correlated with the amount of displacement of the fracture and degree of damage to the articular cartilage.[32]

Other fracture configurations of the middle carpal joint include chip fracture of the distal radial carpal bone, the proximal third carpal bone, and the distal intermediate carpal bone. Prognosis for return to racing soundness depends on the extent of damage to the articular cartilage and amount of exposed subchondral bone at the load-bearing surface. Poor prognostic signs include extensive cartilage damage, a void on a load-bearing surface, and third carpal bone involvement. Free fragments in the palmar aspect of the joint are also considered a poor prognostic indicator.[24]

The radial carpal joint is less frequently involved than the middle carpal joint, but pathology affecting this joint occurs routinely. Bone and cartilage injury usually manifests on the distal lateral radius, which often develops large (0.5–1.0 cm), partially or completely displaced fragments. In many cases, the fragment on the distal lateral radius occurs first, and the horse is very lame (grade 4+/5) initially but becomes relatively sound in a couple of days. If the horse continues to train, it will often develop a fragment on the proximal intermediate and/or radial carpal bones as a kissing lesion. Fragments of the distal medial radius and proximal radial carpal bone also commonly occur. In general, there is a good prognosis associated with removal of fragments in the radial carpal joint.

Radius

Lameness associated with the radius is probably the least common location in the forelimb region of the Thoroughbred racehorse. However, an occasional finding on nuclear scintigraphy would be an IRU in the center of the radius classified as an enostosis-like lesion or bone islet. These findings are seen in the radius, with the tibia and cannon bone also affected.[1] Lameness is attributed to this finding in approximately 50% of cases and correlated with the intensity of the lesion. Therefore, it is important to distinguish between a stress fracture and an enostosis-like lesion, as the rehabilitation program is dramatically different (Figure 9.7). Typically, enostosis-like lesions involving the radius rarely result in lameness, whereas these lesions in the tibia are more likely to result in lameness. Horses with enostosis-like lesions are maintained in training on aspirin. These horses typically walk until they are sound and jog for 1 month before high-intensity work is resumed. A repeat nuclear scintigraphy examination



Figure 9.6. Thirty degree flexed dorsoproximal dorsodistal oblique radiograph showing a slab fracture of the third carpal bone with frontal and sagittal components.



Figure 9.7. Nuclear scintigraphy image demonstrating two focal areas of IRU in the mid-radius corresponding to an enostosis-like lesion that was not associated with lameness.

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will often reveal normal uptake, and the prognosis for a successful career is excellent.

Humerus

Humeral fractures are a common type of injury and are a result of an imbalance in stress remodeling. Typically these horses demonstrate a significant lameness that resolves quickly with a couple days of walking. Often a single limb is affected; however, this can be a bilateral condition in which the horse will often demonstrate a reluctance to train and a markedly shortened cranial phase of the stride. Clinically these horses present very similar to a scapular stress fracture; however, stress fractures of the humerus are more common.[32,40] It is important to note that horses with proximal suspensory desmitis will often demonstrate a shortened cranial phase of their stride and diagnostic anesthesia is important in differentiating between the different regions. The most common time to see lameness associated with the humerus is in a horse that is returning to exercise following a lay-up period. It is not uncommon to see this type of lameness while the horse is jogging or galloping and before they complete their first breeze. The author utilizes the humeral snap test to help diagnose horses with humeral stress fractures. The test is performed by holding the radius while quickly flexing the elbow joint. A horse with a humeral stress fracture will often resent this type of manipulation and should increase the index of suspicion (Video 9.1). However, nuclear scintigraphy is the most reliable method for detecting this injury. The most common location for humeral stress fractures is the proximocaudal aspect of the humerus or caudal aspect of the distal humerus (Figure 9.8). Complete fractures of the humerus are more commonly associated with stress fractures of the proximocaudal aspect than the distal aspect of the humerus.[16] Interestingly racetrack surface has an impact on the location of humeral stress fractures. A study performed in Southern California demonstrated that horses on synthetic surfaces had a greater proportion of distal humeral lesions, whereas horses on dirt surfaces had a greater proportion of caudoproximal lesions.[16] Another interesting observation was that none of the horses that sustained a complete humeral fracture and were euthanized underwent nuclear scintigraphy prior to the injury. This suggests that scintigraphy is very good at identifying horses at risk for complete fracture and helps minimize their occurrence in racehorses.[16] Because complete humeral fractures are always fatal, horses with IRU in the humerus are always given a rest and rehabilitation period. Typically these horses are given 3 months off if no callus is seen radiographically and 4 months off if a callus is seen and have an excellent prognosis for making a complete return without recurrence as long as care is taken not to rush them back following rest and rehabilitation.

Scapula

Scapular fractures are the most common type of injury to the scapula although uncommon when compared with all injuries in the forelimb of the racehorse. Typically these injuries are seen in horses 3 years old and older and initially present as a significant lameness that resolves quickly with a couple of days of walking. This is not unique to scapular stress fractures as this is a characteristic that is common with all types of stress remodeling lameness. Often a single limb is affected. The right is more commonly affected than the left; however, it can occur bilaterally.[56] The clinical presentation is very similar to a humeral stress fracture. The most reliable method of identifying a scapular fracture is by nuclear scintigraphy. The most common site for this injury is at the distal aspect of the spine of the scapula (Figure 9.9). Because this injury occurs in a very characteristic location, firm digital palpation at this location can sometimes result in a painful response, and a positive result would increase your index of suspicion. Additionally, ultrasound examination of this location can demonstrate an increase in thickness of the spine of the scapula with a rough bone margin as a result of the woven bone callus that is present. Typically these horses are given 3–4 months off work and have an excellent prognosis to return to race training without recurrence, provided that the rest and rehabilitation period is not rushed.



Figure 9.8. Scintigraphic image that demonstrate an IRU on the caudodistal aspect of the humerus.

Tarsus

Lameness localized to the distal hock joints (TMT/DIT) is a common source of lameness and typically seen with horses that have sickle hock conformation. Clinically these horses demonstrate an exaggerated collected stabby gait. The rider will often complain that the horse is cross-firing or cross-cantering and not changing

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Figure 9.9. Nuclear scintigraphy image demonstrating an IRU on the distal aspect of the spine of the scapula.

leads behind. Often nuclear scintigraphy and radiographic abnormalities do not correlate well with clinical disease. However, unless a fracture of the central or third tarsal bone is present, the clinical response to intra-articular corticosteroid and a chondroprotective agent is very good and accompanies a favorable long-term prognosis for a successful career despite radiographic changes. Exceptions include overt pathology such as fracture of the third or central tarsal bones or tarsal crushing or wedging.[30,55] Reports suggest a poor prognosis for return to racing with slab fractures of the third and central tarsal bones; however, clinical experience indicates that the prognosis is favorable as long as healing occurs with preservation of normal articular margins and normal shape of cuboidal bones. If there is advanced distal tarsal disease, internal fixation in conjunction with hock drilling may produce good results.

Lameness associated with the high suspensory region is not as common as distal hock OA and is less common compared with the forelimb. It can be localized with diagnostic analgesia of the lateral plantar nerve or local infiltration. Care should be taken when performing a local infiltration as an impending slab fracture of the central or third tarsal bone can also be desensitized. Because of the retinaculum, horses with primary lameness associated with the high suspensory will often show more improvement in lameness when treated with Naquasone compared with NSAIDs. Treatment options are similar to that of the forelimb, and the prognosis is typically more favorable.

Occasionally horses will demonstrate a stringhalt gait that is most commonly associated with a traumatic event; however, the true etiology is unknown. Typically this abnormality in gait is noticed when the horse is walking at the barn or to the racetrack and is not demonstrated while the horse exercises during high speeds. Oftentimes this exaggerated gait bothers the trainer more than the horse and rarely interferes with their athletic potential. For these traumatic or sporadic cases, a lateral digital extensor tenectomy utilizing a standing approach often results in a good prognosis for improvement in gait; however, the exaggerated flexion of the rear limb is often not completely eliminated.

Tibia

Tibial stress fractures are very common in 2-year-old racehorses as they approach their first race. Occasionally these fractures are seen in older horses; however, this is much less common. Typically horses demonstrate a hindlimb lameness following a gate work. The lameness is most commonly identified 1–2 days following the work, and the affected horse will demonstrate a short-strided, toe-first, heel-slapping gait when jogging in hand in a straight line. Tibial stress fractures can occur unilaterally or bilaterally; however, even in the bilateral cases, one leg is often more severely affected. Nuclear scintigraphy is the most reliable method for diagnosing a tibial stress fracture.[33] Typically these fractures occur proximally or distally. Once a tibial stress fracture is identified on a nuclear scintigraphy examination, a radiographic examination is necessary to confirm the presence of a fracture. If a fracture is visible on radiographs (Figure 9.10), horses are given 60 days of stall rest with hand walking followed by a gradual return to exercise. If a fracture is not visible and only periosteal or endosteal reactivity or callus is noted (Figure 9.11), trainers are instructed to walk the horses until they are sound, which typically occurs within a week. The horses are then jogged for 1 month and are maintained on daily aspirin. The prognosis is excellent and the risk of recurrence is low.

Stifle

Lameness in the stifle is commonly diagnosed by clinical evaluation. It is largely believed to be associated with strain and subsequent injury, primarily of the soft tissue structures of the medial femorotibial joint with occasional involvement of the femoropatellar joint. Manifestations of this lameness include the following gait disturbances: the horse travels wide behind, often does not push off the affected limb normally, and has an unsteady appearance of the hindleg. The medial femorotibial compartment usually has joint effusion, but radiographs are typically normal. Intra-articular anesthesia is not typically performed but does improve the lameness if used as a diagnostic aid. Treatment with intra-articular placement of corticosteroids and chondroprotective agents improves the lameness 5–7 days after therapy. If the gait worsens following initial improvement, a tibial stress fracture should be suspected.

Upward fixation of the patella is usually seen in 2-year-olds but may be seen in older horses as well. Most horses with upward fixation of the patella respond

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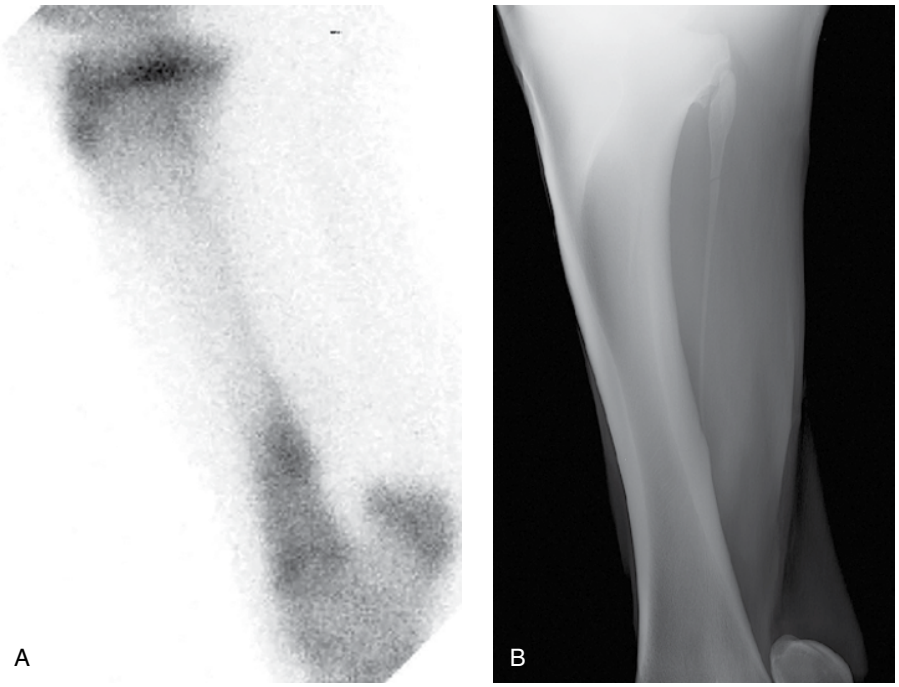


Figure 9.10. (A and B) Nuclear scintigraphy images that show a mild IRU on the mid tibia (A) with corresponding radiographic projection demonstrating a periosteal callus and fracture (B).

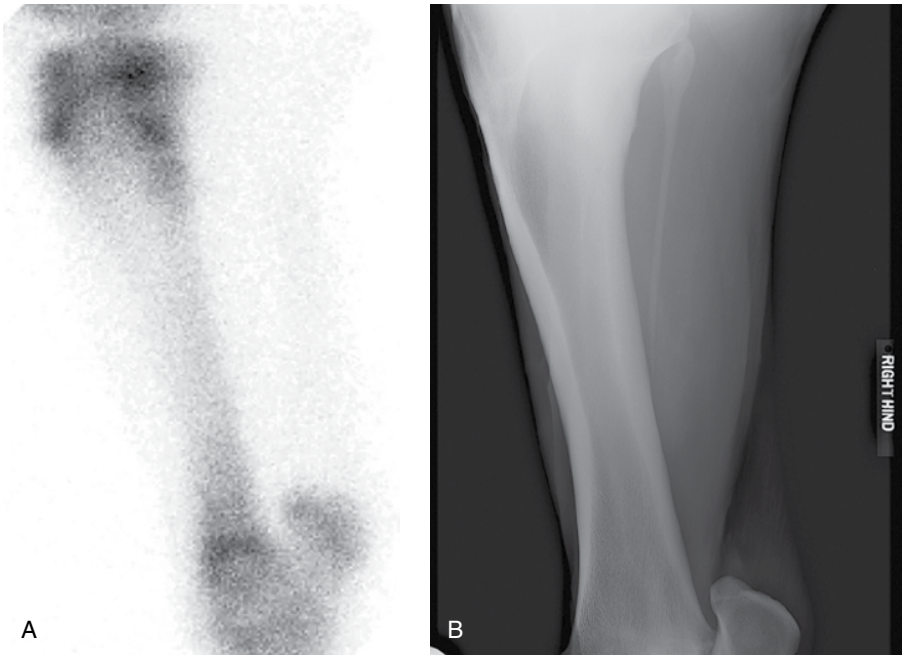


Figure 9.11. (A and B) Nuclear scintigraphy images demonstrate a focal IRU on the proximocaudal tibia (A) with corresponding radiographic projection demonstrating a periosteal callus without fracture (B).

favorably to treatment with systemic intramuscular administration of 3.0 mL of 30 mg/mL mephentermine hemisulfate (90 mg/horse) for 3 consecutive days. Treatment options for refractory cases include internal blister of the patellar ligaments or quadriceps muscles or hormonal therapy. Prognosis for racing is good after resolution of the fixation. Occasionally a fractured patella is the source of lameness; however, this injury is almost always accompanied by a traumatic event and is far more common in the steeplechase discipline. The skyline radiographic projection of the stifle is critical in order to obtain a complete clinical picture.

Pelvis

The pelvic and sacral regions are plagued with a number of soft tissue and bone injuries, which are usually stress induced by excessive exercise. All types of pelvic fractures described in the literature may occur as a result of some form of acute trauma.[50] However, stress fractures of the wing of the ilium and sacroiliac (SI) injury are the most common sites of bone injury of the pelvis in racehorses.[47] Subjectively, female racehorses tend to have an increased incidence; however, some reports have found no sex association.[50] Often horses

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with pelvic stress fractures demonstrate a significant lameness following a gate work and are often pulled up by the rider shortly after breaking out of the gate. Additionally these horses demonstrate a very sore, tucked up hind end, and short-strided gait, and the trainer often believes the horse is tied up. Horses with suspected pelvic fractures should be maintained tied to a wire in the stall to keep them standing for a minimum of 30 days. Recumbency increases the risk of complete fracture, which can result in laceration of closely associated vessels causing fatal hemorrhage. Special care should be taken to monitor these horses for pneumonia as this is a complication of maintaining a horse on a tie wire with the head elevated. The most common location is the ilial wing, and nuclear scintigraphy is the most reliable method of detecting this injury and its location (Figure 9.12). Ultrasound examination is often important in characterizing the type of fracture. Prognosis for most injuries in this region is favorable with rest as long as the fracture is not displaced. Typically horses with ileal wing fractures are given 3–4 months off and have an excellent prognosis with minimal risk of recurrence, whereas fractures that are comminuted, articular, or involve the acetabulum have a poor prognosis for return to racing, and an alternative career should be considered.

Less commonly, lameness will be localized to the SI joints. SI pain is often secondary to a primary lameness lower in the limb, resulting in a change in gait and the way they carry themselves during training. Typically the exercise rider will experience a bunny hop gait when the horse transitions from a jog to a gallop. Often these horses will jog sound when examined in hand. The SI region is medicated with corticosteroids and sarapin; if the primary lameness is identified, this region should be addressed as well.

Soft tissue injuries are recognized in the hip area and are believed to occur during propulsion at fast work. Synthetic surfaces with increased traction have been incriminated in development of this condition; however, there is no current evidence to support this theory. Treatment consists of rest, and the prognosis is good for full recovery.

Tendons and Ligaments

Tendon and ligament injuries in racing Thoroughbreds occur predominantly from intrinsic sources, although extrinsic trauma or injury occasionally occurs. The etiology is multifactorial, but cumulative excessive strain rates in addition to the gradual demise of structural stability incurred with degenerative aging changes that are accelerated by exercise are the primary components.[52] Repetitive loading of a compromised tendon (one that has sustained subclinical, microscopic damage) may be responsible for the often progressive nature of these injuries.[47] Incidence of tendon injury is high and in one study was reported to be responsible for up to 46% of all racetrack injuries,[2] although in clinical experience the rate is not this high.

The forelimb superficial digital flexor tendon (SDFT) is most frequently injured (Figure 9.13) followed by the suspensory ligament, including the origin of the suspensory and medial or lateral branches of the front and hindlimbs, and the distal sesamoidean ligaments. The SDFT develops several characteristic lesions in different locations. A common site is the mid-metacarpal region where it usually develops a core lesion.[45] Central core lesions may be due to unequal strains on fibrils within the tendon. It has been suggested that strain levels on central core fibrils are greater than on peripheral fibers.[3] The second most common lesion of the SDFT recognized clinically involves tearing of the peripheral fibers located on the lateral aspect of the tendon. Juvenile



Figure 9.12. Nuclear scintigraphy image demonstrating an IRU on the right ilial wing.



Figure 9.13. Typical bowed tendon appearance associated with superficial digital flexor tendinitis.

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tendonitis presents as uniform enlargement of the SDFT without fiber disruption and is often unilateral, although bilateral involvement does occur. These cases are identified in 2-year-olds within the first several weeks or months when intensive training begins. As long as these cases are identified prior to fiber disruption, the prognosis is excellent for return to training after a 60–90-day rest period, and recurrence is rare.

Physical evaluation often reveals warmth, mild to moderate edema, and pain upon palpation of an affected area. Horses may exhibit palmar metacarpal swelling without SDFT swelling, usually with very low-grade involvement of the tendon.[22] Lameness may be nonexistent, subtle, and transient or overt. Ultrasound examination is the gold standard to gauge the tendon injury. Caution must be used in interpretation, especially in the acute phase, in which fluid accumulation between fibers may resemble a tear. The follow-up ultrasound examination several weeks later is critical for accurate determination of the degree of tendon injury. Tears are recognized as hypoechoic areas within the tendon, range dramatically in diameter and length, and may be partial or complete. Complete tears appear as anechoic areas, and partial tears appear as hypoechoic, or echogenicity is mixed (Figure 9.14). Affected tendons and ligaments may have a larger cross-sectional diameter than unaffected contralateral structures, and periodically this is the only visible indicator of the problem on ultrasound.[22] More refined imaging may be obtained via MRI and most likely will influence the future management of tendon injuries.

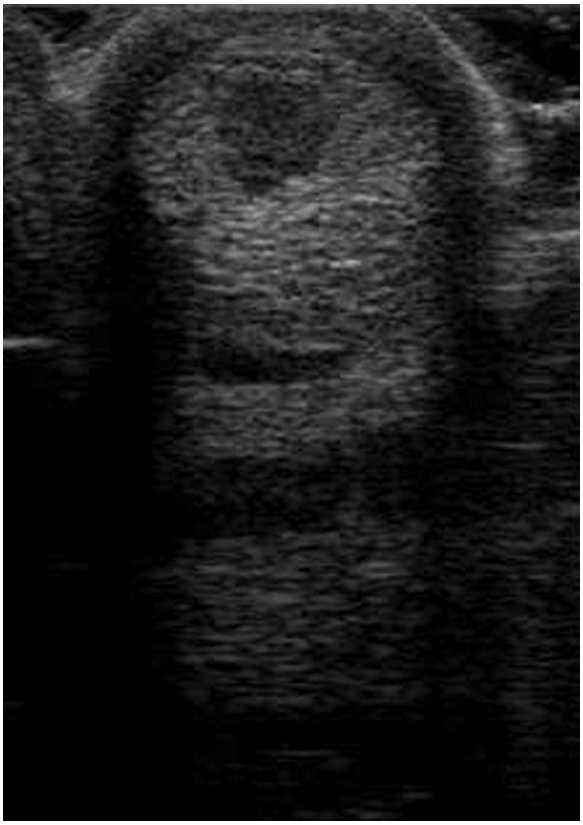


Figure 9.14. Ultrasound image of the flexor tendons showing a core lesion of the superficial digital flexor tendon.

Treatment varies according to the degree of injury. Severe injuries may require complete rest coupled with systemic NSAIDs, hydrotherapy, sweating, regenerative therapies, superior check ligament desmotomy,[28] and gradual return to work, whereas minimal lesions may only need a reduction in training. Serial ultrasonographic examinations help determine the rate of healing and ability to return to full training. Very large defects may benefit from surgical intervention in the form of tendon splitting to permit fluid drainage from the tendon lesion.

The prognosis for return to racing is guarded following tendon injuries. In general, the more severe the tear, or the more distal the location of the lesion, the worse the prognosis. Horses with superficial digital tendonitis in the region of the pastern have a guarded prognosis for future racing. Tendons undergo repair in phases, and the final result is remodeled, mature scar tissue.[45] Frequently, healed areas contain scar tissue that does not have the elasticity of an uninjured tendon, and therefore there is a possibility for repetitive injury. The majority of tendon injuries tend to recur in racehorses, regardless of the treatments employed. Recurrence may be at the original site but is often in a region distinct from the original injury.

Additional Considerations

When considering lameness in the racehorse, one must not fail to consider potential neurologic conditions such as equine protozoal myeloencephalitis (EPM), OA of the cervical vertebral facet joints, or spinal cord compression. EPM is known as the disease that has no characteristic clinical signs. Oftentimes this is seen in young horses that originated from an endemic area, and the stress of shipping and onset of active training may cause enough stress on the individual where the clinical signs become apparent. These horses often present for a lameness issue and failure to train or poor performance. Musculoskeletal evaluation does not reveal any areas of concern, and other diagnostics are unremarkable. The immunofluorescent antibody (IFA) and Western blot blood test is a quick useful way to identify these cases. They typically respond well to treatment and are able to maintain active training once an improvement is noted, which is often within the first week.

OA of the cervical vertebral facet joints is a common cause of hindlimb lameness and poor performance in the Thoroughbred racehorse. These horses demonstrate a stiff neck or altered gait relative to changes in head and neck position and a hindlimb lameness or occasionally a shifting front limb lameness. Diagnosis is typically confirmed with nuclear scintigraphy demonstrating an IRU in the C4–C5, C5–C6, and/or C6–C7 region without any uptake in the hind end. The treatment of choice is ultrasound-guided injection of the articular facet with corticosteroids and a chondroprotective agent. The response to treatment is often very good and rapid. Spinal cord compression cases are relatively uncommon because these cases are often identified prior to active training at the racetrack.

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THE STANDARDBRED RACEHORSE

Kimberly Johnston and Frank A. Nickels

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DESCRIPTION OF THE SPORT

The name Standardbred originated in 1879 in the United States with registration of horses capable of trotting a mile within a standard time of 2:30 minutes. Now the Standardbred is a distinct breed with foals eligible for registration if both sire and dam are registered Standardbreds, regardless of racing performance. Today's Standardbreds currently race much faster, with pacers completing a mile in less than 1:50 minutes and trotters only a few seconds slower. Racing is done on an oval track, usually a mile in length, and horses pull lightweight carts with a single driver. In the United States racing is counterclockwise and commences from a moving start with horses at their post position behind a vehicle with a collapsible partition. Standardbreds race at either the trot or the pace. In the United States, pacers outnumber trotters 4:1, while in Europe and Australia, races are held at the trot only. A horse may have the ability to perform both gaits, but Standardbreds tend to race at only one gait for their entire career. In general, pacers seem to tolerate lameness better than trotters due to the perpetual swinging motion of the pace itself.

The Standardbred resembles the Thoroughbred; however, it is longer and does not stand as tall, averaging between 14.2 and 16 hands. Adequate body length is essential for a long stride and to allow free movement of the limbs without interference. Conformation plays a pivotal role in the success of the racehorse, and many conformational faults predispose to interference or lameness problems. Toeing out is undesirable in the front limbs, especially when accompanied by a base-narrow stance. This conformation affects the flight of the hoof and can cause interference. Toeing in causes less interference but results in an inefficient gait and increases the strain on the medial side of the limb, leading to carpal pain and the development of splints. Horses that are offset or back at the knees are at risk of carpal chips and slab factures. Sickle hocks used to be common in pacers and were even selected for, but sharp angulation of the hindlimb predisposes to curb and hock arthritis. In contrast, many trotters are cow hocked, but unless severe, this conformation may be advantageous because it allows a passing gait in which the imprint of the hindlimb falls outside the imprint of the forelimb, providing a longer stride.

Standardbreds are broken to harness as yearlings and train for 7–9 months before racing as 2-year-olds. Horses are jogged at a slow trot/pace for several miles daily to gain fitness and stamina. Jogging is done "the wrong way" (clockwise) around the track, and a few times a week, a horse is turned to work "the right way" for short bursts of speed training. During peak season, Standardbreds may race as often as once a week. This pounding on hard tracks for many miles and the fact that Standardbreds race and train in different directions makes them prone to repetitive stress and maladaptive bone injuries (Figure 9.15).



Figure 9.15. Although the pace is a natural gait, horses often wear hobbles to help maintain the gait while racing. This equipment commonly causes hairless areas on the legs, making a Standardbred pacer readily identifiable.

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LAMENESS EXAM

Lameness is a leading cause of poor performance that plagues nearly every Standardbred racehorse at some point during its career. Compared with many other disciplines, a high percentage of hindlimb lameness problems are encountered as a result of the Standardbreds' symmetrical gaits and having to pull a draft load.

History taking should include age, stage of training, and whether the horse is a trotter or pacer. Owners should be asked if the horse is lame or simply not performing well. Young Standardbreds experience a subset of problems not commonly seen in the mature horse, and examination of unproven 2- and 3-year-olds with poor performance must be sufficient to rule out the cardiovascular or respiratory systems as the cause.

Lame horses should be examined for signs of localized inflammation (heat, swelling, or pain), and the trainer asked about the duration of the problem. It is important to ask about the size of the track that the horse races on and whether lameness is worse in turns or on the straightaway. Most harness races are a mile long, but track lengths do vary. On smaller tracks, horses must negotiate more turns and the turn diameter is less. Lameness from the medial side of the limb is more evident in the turns, whereas lameness from foot pain, splints, and curbs worsens with distance and is likely more obvious at the end of a race. A change of racing venue may prove successful for horses with chronic lameness issues. For example, a trotter with stifle arthritis may perform better on a 1-mile track where it only has to negotiate two turns.

It should be noted whether the horse "breaks stride" (gallops instead of maintaining the trot or pace), which is a serious fault for the harness horse. Hock pain causes horses to get uneven coming into or out of the turns, but they usually maintain the gait, whereas fetlock and stifle lameness often cause horses to break stride in the turns. Horses can break for many reasons other than lameness, including excitement, immaturity, and incoordination, and interference is a major cause of breaking. In this case, shoeing or tack changes may correct the problem. The "bute test" can be helpful to differentiate lameness from interference: a horse is prescribed a modest course of phenylbutazone and then reexamined. It is believed that lameness improves with the nonsteroidal anti-inflammatory medication while interference remains unchanged. Neurologic disease also must be considered in any horse with unresolved gait abnormalities.

Several terms unique to Standardbred racing provide information about a horse's performance. "Bearing in" or "bearing out" means a horse drifts toward the infield or the outside rail, respectively. Drivers may comment that a horse is "on a line," indicating that they must pull harder on one rein to keep the horse from bearing in or out. Horses drift away from a source of pain, so a horse on the right line is likely to have right front lameness. Alternatively, for rear limb problems, the hindquarters may move closer to one shaft of the sulky, and the horse is then described as being "on a shaft" (Figure 9.16). There are exceptions so this information must only be used as a guide. Horses with right front medial heel pain, for instance, are commonly on the left line during a race because they try to avoid loading the inside of the limb (Figure 9.16).



Figure 9.16. This 2-year-old Standardbred pacer demonstrates being on a shaft. The colt's hindquarters are positioned closer to the right shaft of the sulky. This colt is also on the left line, and its head is turned to the left as the driver pulls harder on the rein in an attempt to keep the horse straight. This combination of findings suggests a left hindlimb lameness.

Interference may mimic lameness if horses alter their stride to avoid hitting another limb, and it can result in bruising and lacerations that do cause lameness. Alterations in gait can also cause horses to overload other limbs and develop compensatory lameness, which is so common in the Standardbred that veterinarians must always consider the possibility that new problems have arisen. Both primary and secondary lameness issues must be addressed to achieve soundness. Trotters tend to develop diagonal lameness problems, whereas pacers may develop ipsilateral or contralateral issues. Tack changes such as the addition of hobbles, boots, or a head pole often indicate high-speed lameness and should be assessed.

Finally, the clinician must know the horse's history, including racing performance, prior lameness, treatments, and response to those treatments. Detailed record keeping is imperative, since many owners and trainers prefer jumping straight to treatment rather than pursuing extensive diagnostic tests. Without a complete diagnosis, response to treatment becomes the barometer of clinical accuracy. Standardbred trainers may try to treat problems on their own, resulting in conflicting clinical findings. It is not uncommon to be presented with a horse that has "had all his joints done" (meaning intra-articular injections) but is still not performing well.

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Careful inquiry may reveal that an area has been overlooked or the veterinarian may need to shift the focus to soft tissue structures rather than just injecting the horse again. It is also important to remain aware of the potential for joint infection following intra-articular injections. Onset of clinical signs can be delayed as long as 2 weeks if steroids are used.

An acceptable venue to watch a horse in hand can be difficult to find at the racetrack, and Standardbreds are not always amenable to jogging on a lead, making observation of the horse in the shed row typical. Unsound trotters sometimes prefer to pace at slow speeds; however, the pace is a forgiving gait, and front limb lameness may be difficult to detect. Most horses, including pacers, will finally trot in hand after several attempts. Correlation between lameness seen in hand and that at racing speed is often poor, so observation of horses in harness on the track is routine.

Flexion tests are part of most exams, although their value is dubious. The carpus can be flexed exclusively and this manipulation is generally useful. In the hindlimb, an effort should be made to flex the fetlock separately from the upper limb, because problems are relatively common in this joint.

A few points regarding the use of diagnostic analgesia in the Standardbred deserve mention. Foot pain is so commonplace that blocking of a front limb should always begin with anesthesia of the palmar digital nerves to establish the significance of heel pain in the overall picture. The abaxial sesamoid block is avoided because it can desensitize the foot, pastern, and parts of the fetlock. Inadvertent anesthesia of fetlock pain could be mistaken for lameness of the foot, with dire consequences if a fracture is present. Hindlimb foot pain is infrequent, so digital anesthesia is rarely warranted; however low plantar anesthesia should be used to rule out the distal limb for all but the most obvious cases of hindlimb lameness. Observation of the horse on the track before and after diagnostic analgesia can be useful; but there is risk of further injury, so the driver should be instructed to only drive the horse as far as is necessary to assess the effectiveness of the block and to avoid abrupt stops or turns. Propagation of incomplete fractures or catastrophic breakdown is possible following desensitization of a limb. Finally, the veterinarian must remember to abide by local jurisdictions regarding the use of local anesthetics close to race day.

SPECIFIC LAMENESS CONDITIONS

Foot

Standardbreds race on compacted limestone tracks and are shod frequently, predisposing them to foot pain. It is not uncommon for a horse to be foot sore for several days after a race. The long-toe, low-heel hoof conformation seen in many Standardbreds, along with the repetitive concussion sustained from jogging on hard tracks, predisposes them to corns. Horses that are shod tight or have the heel of the shoe turned in to prevent it from being pulled off if they overreach are likely to develop bruising from the focal pressure placed on the caudal angle of the bars. Fracture of the distal phalanx is a relatively frequent cause of foot pain and should be considered in severely lame Standardbreds that have no localizing signs. Acute lameness is seen after a race, often in the winter months.

Distention of the distal interphalangeal joint is a nonspecific sign that may be noted in conjunction with foot soreness. Radiographic signs of coffin joint arthritis may or may not be present because synovitis can be a reflection of inflammation in nearby structures, including the podotrochlear apparatus. Response to intra-articular medication is generally favorable but often must be repeated, especially if an accurate diagnosis is never made.

Pastern

The pastern region is often overlooked in the Standardbred. Many practitioners are not as familiar with the intricate anatomy of this area, so soft tissue injuries are likely underdiagnosed. Injury to the distal sesamoidean ligaments, collateral ligaments, and flexor tendons in the pastern can all result in lameness and even joint instability, causing ringbone months or even years later.

Fetlock

Synovial or joint capsule inflammation is common and develops as the amount of training increases. The condition can be secondary to altered gait from another lameness issue, and it is frequently seen in horses with sore heels as a result of toe-first landing. Heat and effusion are present along with a positive response to joint manipulation, but overt lameness is not typical. Fibrosis and mineralization of the synovial pad can occur with chronicity resulting in a decreased range of motion.

Osteochondral fragments are frequently encountered in the fetlock, most commonly originating in the dorsomedial proximal margin of the first phalanx.[6] Concurrent thickening of the synovial pad is frequently observed because the two sites make contact during maximal extension of the joint. Small fragments may not be a significant problem, but larger fragments close to the articular surface result in lameness and can produce score lines on the metacarpal condyle. Frequent injection may keep horses going, but fragment removal is the treatment of choice to prevent joint deterioration.

Proximal palmar/plantar osteochondral fragmentation of the first phalanx is a frequent radiographic finding in Standardbreds. These lesions are routinely removed prior to the onset of a racing career. Not all fragments cause lameness, and when they do it is mild. The complaint more often involves an inability to go straight or breaks at high speed. Most fragments are found medially in the left hindlimb, and horses with an outwardly rotated hindlimb axis are predisposed.[6] Demonstration of the lesion requires oblique views angled 30°–45° distad to prevent overlap of the proximal sesamoid bones on the palmar/plantar aspect of the first phalanx. Problematic fragments can often be managed using intra-articular therapy until the end of the racing season without risk of further injury to the joint. Arthroscopic removal of axial fragments is then indicated, but the value of this has come into question.[1]

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Abaxial fragments are not articular and surgical removal should not be attempted.

Fractures of the first phalanx occur and are generally straightforward; however, special mention should be made of incomplete sagittal fractures because they are common in the Standardbred and can represent a diagnostic challenge. Marked lameness after a race subsides quickly but recurs with return to work. Heat, swelling, and joint distention may be absent or minimal. This scenario, coupled with a positive response to fetlock flexion, should arouse suspicion. Radiographs prior to diagnostic anesthesia are warranted because propagation of the fracture can occur. Short fractures may not be radiographically visible until bone resorption occurs 3–6 weeks following fracture. Diagnosis is facilitated by observation of linear lucency in the proximal sagittal groove on the AP view and dorsal periosteal remodeling, which is best seen on the lateral to medial projection. Sclerosis near the midsagittal groove often precedes fracture, and nuclear scintigraphy is likely to demonstrate the lesion much earlier. Until an accurate diagnosis can be made, these cases should be managed conservatively in a stall to prevent catastrophic injury. Short fractures may be treated with confinement alone, whereas longer, propagating, or nonhealing fractures benefit from internal fixation. Healing occurs in 4–6 months, and the prognosis for racing is good, provided significant degenerative changes do not occur.[8]

Cyclic bone fatigue occurs in the Standardbred when bone adapts to the miles of low-speed jogging one way of the track and is then unable to endure the intense loads encountered during speed work in the opposite direction. Common sites of stress remodeling in the fetlock include the distal plantarolateral aspect of the metatarsal condyle, distal palmaromedial aspect of the metacarpal condyle, proximal sesamoid bones, and the proximal aspect of the first phalanx.[3] Physical findings are minimal, and affected horses have a variable response to fetlock flexion. Subtle high-speed lameness or frequent breaks in the turns are typical. Minimal improvement is seen with intrasynovial anesthesia, except in the most severe cases, because intact cartilage prevents desensitization of the subchondral bone. Nuclear scintigraphy is a valuable tool to reveal identifiable patterns of uptake for this disease entity. Radiographic findings offer insight for prognosis, because changes are nearly undetectable initially, and then progress to sclerosis and flattening of the palmar/plantar condyle. A skyline view of the palmar aspect of the condyle is obtained by holding the limb in flexion while the X-ray beam is aimed dorsodistal to palmaroproximal through the fetlock joint (Figures 9.17 and 9.18). A characteristic "gull wing" pattern of sclerosis on the condyle provides the diagnosis. Exercise must be reduced to allow the adaptive process to equilibrate and microdamage to heal.

Arthritis is probably the most common condition of the fetlock. Joint distention, most notable in the palmar/plantar recess, is characteristic along with variable lameness exacerbated by flexion. Range of motion may be reduced due to joint capsule thickening and fibrosis. Some horses subsist surprisingly well, and lameness does not become a prominent feature until the disease is advanced. A subclinical condition often becomes apparent after hard training or racing under adverse conditions. Diagnosis is based on the presence of degenerative changes on radiographs, and treatment is primarily palliative.



Figure 9.17. An example of positioning to obtain a flexed 45° dorsodistal to palmaroproximal oblique (skyline) radiograph of the metacarpal condyle.

Proximal Sesamoid Bones

Sesamoiditis is believed to result from minor tearing of the suspensory attachment to the proximal sesamoid bones, causing inflammation. Diagnosis is based on radiographic changes, including the presence of prominent vascular channels and patchy sclerosis within the bones. Many believe that the condition predisposes horses to sesamoid fracture, but in a serial radiographic investigation of 71 young Standardbreds, horses that developed proximal sesamoid bone fractures did not have radiographic signs consistent with sesamoiditis.[7] Rest and NSAIDs are the mainstays of treatment, but prognosis for chronic cases is poor.

Fractures of the sesamoid bone are associated with trauma and generally occur in areas of attachment of the suspensory ligaments. It is believed that the suspensory ligament responds to conditioning faster than the sesamoid bone, predisposing the bone to failure during overloading. Horses present with lameness, joint swelling, and thickening of the branch of the suspensory ligament attached to the fracture. Pain is elicited with digital pressure over the bone. Treatment and prognosis vary depending on the type of fracture and extent of concurrent soft tissue injuries. Surgical removal of apical and abaxial fragments is indicated in acute cases.

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Figure 9.18. This radiograph of the metacarpus was obtained using the position described in Figure 9.17. This view is useful to demonstrate small fissures or sclerosis of the palmar aspect of the condyle, neither of which are present in this case.

Metacarpus/Metatarsus

Standardbreds are not prone to the bucked shin complex seen in other racing breeds, although the problem is occasionally seen in forelimbs of 2-year-old pacers during late summer. Bruising of the hind shins may occur in trotters that interfere, with the front foot hitting the dorsal surface of the metatarsus.

Injury to soft tissue structures in this region is common. The majority of cases result from repetitive stress injury, although acute trauma and even tendon rupture can be seen in Standardbreds that race without adequate leg protection. Tendon injuries are relatively easy to diagnose in the Standardbred; swelling and pain prompt ultrasonographic evaluation. Injury to the superficial digital flexor tendon (SDFT) is most common, and the severity dictates the course of treatment. Prognosis for SDFT injury appears better in the Standardbred than other racing breeds. Horses with injury within the tendon sheath often develop constriction by the palmar annular ligament and may require transection of this ligament prior to racing.

Proximal palmar metacarpal pain is regularly encountered in the Standardbred, and differentials must include proximal suspensory desmitis (PSD), injury to the inferior check ligament, and sagittal fracture of the cannon bone, with PSD being most likely. Horses with PSD usually have a head nod that is pronounced when the limb is on the outside of a circle. Affected horses can move in a manner that mimics carpal pain, and lameness may be exacerbated by both fetlock and carpal flexion. In the hindlimb, swelling from PSD is minimal, and horses can be positive to any or all hindlimb manipulations, including the Churchill test, making diagnostic anesthesia critical. In all cases, diagnosis is confirmed with ultrasound, but radiographs should also be obtained to look for bone abnormalities at the ligament's origin, proximal sesamoid bones, and the splint bones, which are frequent in racing Standardbreds.

Suspensory branch desmitis is common in the Standardbred, and it is frequently seen in horses that train on tracks with tight turns or excessive banking. Single branch disease is related to hoof imbalance or poor conformation, whereas biaxial injury results from hyperextension of the fetlock joint. Lameness is not a prominent feature, and many Standardbreds continue to race and train, accumulating damage and propagating injury toward the suspensory body. Injury is easily recognized by the visible swelling at the back of the fetlock and palpation of the enlarged branch(es).

Carpus

Carpal pain represents a significant cause of lameness in the Standardbred. One study revealed its presence in one-third of the population and found it to be the top reason for horses to require a layoff longer than 1 month.[17] Lameness results in a distinctive gait characterized by abduction of the limb and a shortened cranial phase of the stride from a reluctance to flex the carpus. Affected horses often stand in the stall with the knee(s) slightly flexed to reduce tension on the dorsal joint capsule. Heat and swelling are invariably present and often recognized by trainers.

Synovitis and capsulitis are frequently encountered in the young Standardbred. Sensitivity to palpation, pain on flexion, and reduced range of motion are typical. Treatment is aimed at returning the joint to normal as quickly as possible; NSAIDs and ice are helpful in the acute stage. Walking should follow to regain joint mobility and limit fibrosis. In some cases, intra-articular hyaluronan (HA) or steroids are needed to control the inflammatory process. Suppression of synovitis is important to prevent the products of inflammation from compromising the articular cartilage and causing arthritis.

Osteochondral fractures in the carpus are a direct result of race training. In the Standardbred, fragments are mainly seen in the middle carpal joint and are almost exclusively located dorsomedially, coming from either the distal radial carpal bone or the proximal third carpal bone.[14] Sudden onset of carpal swelling with lameness is typical. Flexion exacerbates the lameness, and digital pressure along the dorsal aspect of each bone while the joint is flexed may help localize the source of pain. A complete radiographic study of the carpus should be performed to document any chips as well as subchondral sclerosis. Remodeling occurs in both limbs simultaneously; therefore, the contralateral carpus should also be radiographed to look for concurrent disease. In the racehorse, most chips are clinically important, and arthroscopic removal is the treatment of choice.

The third carpal bone is prone to maladaptive stress remodeling that causes pain and fractures. Evaluation of Standardbreds in training revealed middle carpal joint

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lameness in 30% of horses, with severity directly related to the degree of third carpal bone sclerosis.[9] Increased bone density and loss of vascular channels cause ischemic necrosis and predispose to further injury (Figure 9.19).[19] Lucency of C3 is also associated with lameness,[14,17,19] and lesions in bone and cartilage are usually identified at surgery.[14] Etiopathogenesis is similar to that seen in the fetlock and requires similar management.

Incidence of carpal slab fractures is highest in 2-year-olds, with frontal plane fractures of the radial facet of the third carpal bone the most common configuration.[14] Distribution is nearly equal between the right and left limbs. Horses are lame and resentful of flexion, often leaning back or rearing to avoid it. Diagnosis is confirmed using radiography; the tangential view (skyline) of the third carpal bone is the most valuable. Fractures heal with conservative management, but surgery limits the severity of joint deterioration and provides the best chance to return to racing. Up to 77% of Standardbreds return to racing following slab fracture, but earnings may be decreased.[18]

Tarsus

Young Standardbreds with uneven gaits or those subjected to miles of jogging quickly develop tarsitis. Affected horses exhibit a shortened stride and may stab the toe laterally as they land, causing uneven wear to the shoe. Disease is most frequent in the tarsometatarsal joint but can occur solely in the distal intertarsal joint; therefore, these joints should be blocked (and treated) separately. Nuclear scintigraphy of the Standardbred with distal tarsitis reveals increased radiopharmaceutical uptake (IRU) in the dorsolateral region of the joints, in contrast to a medial location seen in most other sport horses.[4] Radiographic findings correlate poorly with disease severity, and they are frequently negative in 2- and 3-year-olds despite a response to diagnostic anesthesia. Intra-articular use of HA and corticosteroids is beneficial, and older horses may require regular injections to remain competitive. Flat shoes with square toes ease break-over, and the removal of all shoe additives (toe grabs) helps to reduce shear strain.



Figure 9.19. A flexed 35° dorsoproximal to dorsodistal (skyline) view of the distal row of carpal bones in a 3-year-old Standardbred filly with grade 2 of 5 right forelimb lameness. Note the generalized sclerosis of the third carpal bone along with focal lucent areas in the radial facet, indicative of bone necrosis.

Distention of the tarsocrural joint is usually associated with osteochondrosis (OCD) that is prevalent in the Standardbred. Frequency and distribution of OCD lesions may differ between trotters and pacers,[11] and early removal of most fragments allows horses to perform equivalently to unaffected counterparts.[11]

Fractures of the central or third tarsal bone are occasionally seen in the racing Standardbred. Lameness can be severe, but other external signs are often limited and do not specifically incriminate the distal tarsus. Both conservative treatment and internal fixation have provided a fair prognosis for return to racing, but arthritis remains an inevitable sequellae.[13,20]

Curbs frequently develop in 2-year-olds as speed training begins. In addition to swelling in the long plantar ligament, injury to the digital flexor tendons, collateral ligaments, and numerous other structures in this region can lead to the appearance of curb.[15] Cryotherapy (freeze firing) is often employed for persistently painful curbs.

Stifle

The stifle is frequently implicated as a source of lameness in the Standardbred, but a thorough work-up is rarely performed at the track, and the true incidence of lameness attributable to this region is unknown. Usually, lameness blocks out lower in the limb. Horses with stifle lameness tend to carry the affected limb wide and land on the toe. Trotters may develop a bunny hop gait, particularly if affected bilaterally.

Standardbreds with so-called loose stifles frequently have quadriceps muscle soreness that makes them resemble horses with intermittent upward fixation of the patella. They develop an abnormal cranial phase of the stride as the patella momentarily "catches" before the limb is brought to the ground. Generally, this is a fitness issue that is cured by trotting up and down hills or working in deep footing to hasten conditioning. Nevertheless, many veterinarians advocate internal blisters over the medial patellar ligament to induce scarring and decrease laxity. Medial patellar desmotomy should be reserved for cases of true upward fixation because fragmentation of the distal end of the patella has been reported following this procedure in normal horses.[12]

Stifle swelling should be a red flag whenever it is found. Marked femoropatellar effusion with minor lameness is characteristic of OCD and should prompt radiographic investigation.[5] The postoperative prognosis is favorable in the Standardbred, as long as lesions are not extensive. Medial femorotibial effusion is a more subtle but ominous sign, often indicating advanced arthritis or injury to the intra-articular support structures of the stifle. These injuries usually end a racing career.

Upper Limb Stress Fractures

Racehorses can experience stress-related bone disease in the proximal appendicular skeleton, including

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the radius, humerus,[10] tibia,[16] and pelvis.[3] A high index of suspicion enhances the ability to diagnose these stress fractures, and a combination of nuclear scintigraphy and radiography is often needed. Tibial stress fractures are the most common and are usually seen in 2-year-olds.[16] Lameness is noted after speed work and abates quickly with rest. Palpation is unrewarding but response to upper limb flexion may be dramatic. The fracture location is unique for the Standardbred, with the majority in the mid-diaphysis of the tibia, whereas fractures in Thoroughbreds are usually in the proximal caudal or caudolateral cortex.[16] Bilateral fractures can be seen, even in horses with unilateral lameness (Figures 9.20 and 9.21). Horses with scintigraphic evidence of stress remodeling should be treated conservatively, even if fracture is not radiographically visible. The prognosis is good for return to racing following appropriate rest.

Sacroiliac Pain

Sacroiliac pain is very common in the Standardbred and may present as poor performance or overt lameness. Drivers sometimes describe a horse's reluctance to take hold of the bit and think the problem is related to the teeth. Injury occurs during slips and falls, and damage to either the ligaments supporting the pelvis or the sacroiliac joint results in a similar clinical picture. Treatment includes rest and NSAIDs or injection of the sacroiliac joint. Both types of injury should be followed by a controlled exercise program with specific exercises to strengthen muscles of the back and croup.[2]

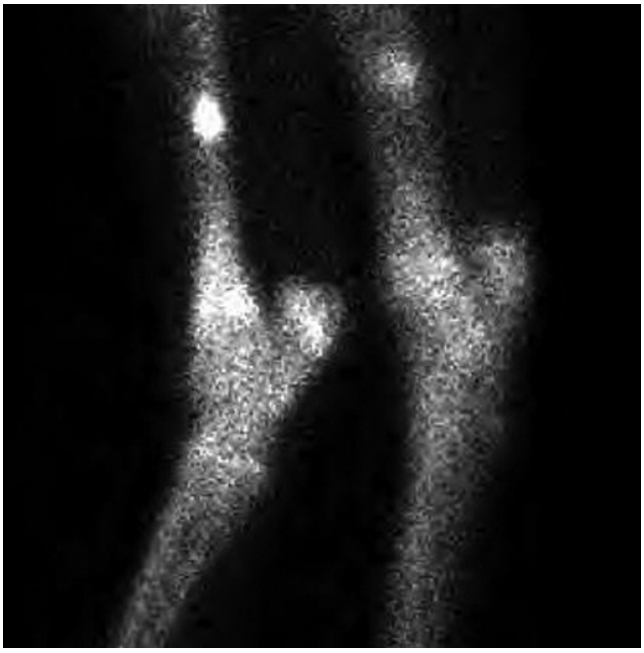


Figure 9.20. A lateral scintigraphic image of the hindlimbs in a 2-year-old Standardbred filly with obscure right hindlimb lameness. Note the focal intense radiopharmaceutical uptake in the mid-diaphysis of each tibia.



Figure 9.21. A dorsolateral to palmaromedial oblique radiograph of the tibia from the same horse as in Figure 9.20. Endosteal sclerosis and callus formation along the caudolateral cortex in the mid-diaphysis indicate the presence of a stress fracture. The horse was rested for 6 months and made a full recovery.

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THE RACING QUARTER HORSE

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INTRODUCTION

Racing Quarter horses are the sprinters of the racing world. They are capable of attaining speeds in excess of 50 mph in less than 21 seconds at a distance of one-quarter mile. The breed originated in colonial Virginia in the 1600s when imported English Thoroughbreds were crossed with native breeds of Spanish descent producing a compact, heavily muscled horse designed for sprinting. They were referred to as Quarter Pathers or Quarter Milers, named after their decided quarter mile racing distance.

The first race records are from 1674 in Enrico County, Virginia, where match races were run down village streets and in small, level fields. The contemporary version of the sport emphasizes 2-year-olds racing in futurities, which run in qualifying heats where horses with the 10 fastest times compete in a final 2 weeks later. Horses must be nominated to futurities, and periodic payments are made in order to maintain eligibility. The same applies to derbies held for 3-year-olds, though the purse money is less than futurities. A very prominent Quarter horse race is the All American Futurity held annually on Labor Day weekend at Ruidoso Downs, Ruidoso, New Mexico, and features a 2-million-dollar purse, with $1 million going to the winner. Los Alamitos racetrack in Southern California, the only predominantly Quarter horse track, offers at least five races during the year with high dollar purses, with the Los Alamitos $2-million-dollar futurity being the richest. The Champion of Champions, which is held at the end of the racing season, is the most prestigious race for older horses. It represents winners of the top races nationwide throughout the year.

Yearling sale prices are driven by precocious, nicely conformed, and well-bred individuals with the potential to compete in these lucrative futurities and derbies (Figure 9.22).

The American Quarter Horse Association (AQHA) studbook has remained open to breeding with Thoroughbreds since the breed was formally established. This outcross has been beneficial in expanding an otherwise small gene pool. It also maintains the classic quarter mile distance of 440 yards, although some Quarter horses run their best at even shorter distances of 300–350 yards.

CONFORMATION RELATING TO LAMENESS

Conformational factors that have been associated with lameness in the forelimb are relatively large body mass, poor carpal conformation (back at the knee and bench-kneed), short upright pasterns, and poor hoof conformation (Figure 9.23). In a scientific study, the most significant finding was that for every inch increase



Figure 9.22. Precocious individuals with popular bloodlines are sought after at yearling sales. This horse (pictured as a sale yearling) won the All American Futurity the following year. *Source*: Courtesy of The American Quarter Horse Racing journal.

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Figure 9.23. Back-in-the-knee conformation on a horse immediately prior to arthroscopic surgery for carpal chip fragmentation. *Source*: Courtesy of Dr. CW McIlwraith.

in toe length, the odds of sustaining a carpal chip fracture increased by a factor of 40.[14] Major hind end conformational defects (e.g. sickle hocks, cow hocks, too straight in the stifles) are undesirable in the Quarter horse racing breed. Breaking sharply from the starting gate is necessary to be competitive, and any serious hind end lameness may inhibit the individual's ability.

TRAINING THE RACING QUARTER HORSE

Horses are usually started under saddle in the latter part of the yearling year to prepare for racing as 2-year-olds. They are not allowed to race before March of their 2-year-old year, and they are restricted from racing 440 yards until later in the year.[1] In California the horses are required to be truly 24 months of age before their first race. The early races are very short (220 yards), and their training schedule is light compared with the racing Thoroughbred. Most of the 2-year-old Quarter horses are very precocious, big bodied, and naturally fast. They can perform well with a low level of fitness, which is possibly a risk factor for injury. Once they are fit, they gallop fewer days than Thoroughbreds, and many older racehorses spend much of their time on a mechanical horse walker at the barn.

SHOEING

The most serious shoeing considerations in the Quarter horse are probably the same as in the Thoroughbred: long toe, low, excessively sloping heels, and medial to lateral hoof imbalance. Most racehorses are shod close to or on race day; consequently, corrective shoeing is not employed as often as needed in many cases because of the risk of sore feet on race day. Horses race in aluminum shoes and various types of pads are employed for foot-sore horses, including rim pads, wedge pads, full plastic pads, and even full aluminum pads with an assortment of hoof packing. Toe grabs are controversial, especially in Quarter horse racing. They have been associated with catastrophic injury in the racing Thoroughbred, and racing regulators restrict their use in some states. There is very little published data regarding toe grabs in Quarter horse racing, and one study found underrun heels as a more significant risk factor for catastrophic injury than toe grab length.[2] Historically, Quarter horse racehorses have used up to 8-mm-(high) toe grabs to prevent stumbling and slipping from the starting gate, but the norm now is 2- to 4-mm toe grabs, with higher ones used only rarely. Races can be won or lost at the break in Quarter horse racing due to the short distance. The action of the front hooves pointing into the racing surface at the first jump out of the gate is different than that of Thoroughbreds, as is the fact that they actually pull with their front legs as well as push from the hindquarters (Figure 9.24). Some leading trainers feel that the use of higher toe grabs may be necessary for a few individuals after the horses have stumbled or fallen out of the starting gate while wearing a low toe grab. A 2-year survey in California found no adverse effect of toe grabs on the rate of catastrophic injury in racing Quarter horses, with a similar distribution of injury to the control population.[10] Further studies specific to Quarter horse racing and toe grabs correlated to injury rates are necessary, as is investigation into racing surfaces and the condition of the racetrack itself.

LAMENESS RELATED TO TRACK SURFACE

Quarter horses race on varying track surfaces around the country; however, trainers prefer a firmer surface because a loose or sandy surface poses problems with breaking at speed from the starting gate and gaining enough traction for sprinting at high speeds. A different set of injuries is associated with racing on a sandier track, particularly tendinitis of the superficial flexor tendon. Hind end lameness, muscle pulls, back soreness, and suspensory ligament injuries also are more prevalent. Although firmer tracks are preferred, they may lead to a higher incidence of joint and bone injury due to greater concussive forces. There are currently no statistics for Quarter horse injuries associated with synthetic tracks.

LAMENESS EXAMINATION

There is no set procedure for lameness examination, but a systematic approach is essential to assure that it

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is thorough. It is important to be efficient because many trainers will have their veterinarian examine every horse before it is entered to race. Soundness is imperative for optimum racing performance in the Quarter horse breed because the races are so short and they are regularly determined by photo finishes. Lame horses may be fractious in the starting gate, and those with hind end lameness may be slow to break from the gate. Quarter horses require a great deal of lameness monitoring to remain at the top of their game. Intra-articular therapy is frequently employed due to the high incidence of joint injury and inflammation associated with speed and concussive forces. Hyperextension of the carpus and metacarpophalangeal joints is common in racehorses and can contribute to lameness (Figure 9.25).

It is very useful to watch the horse walk out of the stall and down the shed row, and this is a good time to obtain a history from the trainer or barn foreman. A sore carpus may be noted immediately by a characteristic wide placement of the leg while walking. Observations such as "The horse gets hotter than usual at the track in the morning" or "He is digging a hole in the stall and standing in it" are useful parts of the history. Observing the horse walk and jog on a hard, level surface may accentuate the lameness and aid in diagnosis. Hoof testers should always be used to define lameness pertaining to the foot. Joint flexion and palpation are particularly useful due to the high incidence of joint problems in the Quarter horse racehorse.

In addition to a conventional lameness examination, the authors pay particular attention to certain examinations



Figure 9.24. Quarter horses break from the starting gate at Ruidoso Downs. Notice the action of the front hooves pointing into the racing surface. *Source*: Courtesy of The American Quarter Horse Racing journal.



Figure 9.25. A champion 870-yard Quarter horse winning a stakes race at Remington Park. This is a good example of hyperextension of the carpus and metacarpophalangeal joints commonly seen in racehorses. *Source*: Courtesy of Dustin Orona Photography/ Remington Park.

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in the racing Quarter horse. This includes flexing the carpus with the leg in a raised position so that the radius is horizontal and looking for an immediate withdrawal response of the neck and shoulder muscles as a response to pain. Directly palpating the carpal joints by placing the thumbs along the individual dorsal borders of the carpal bones while the fingers apply pressure behind the joint can further localize the lameness. The coffin joint is palpated for heat and excessive joint effusion, and the digital pulse should always be checked because it is frequently elevated in horses with acute foot problems. A positive Churchill test may indicate hock soreness and is quickly performed while palpating the distal limb. The medial femorotibial joint is the most common area of soreness in the stifle and may or may not have effusion. The history of a poor performance (especially leaving the gate) usually initiates a more complete examination of the hindlimb. Diagnostic blocks are used when necessary to localize the lameness. See Chapter 2 for more details.

SPECIFIC LAMENESS CONDITIONS

The lameness conditions discussed below are the most relevant for the Quarter horse racing breed. Most of the topics are covered extensively in other chapters; therefore, this section is meant as a review of Quarter horse injuries and how they differentiate from Thoroughbreds.

Arthrosis of the Distal Interphalangeal (DIP) Joint and Problems Associated with the Foot

Coffin joint synovitis is a significant cause of lameness in the Quarter horse.[7] The breed is well known for having undersized feet in relation to the body size, which, coupled with the tendency for racehorses to develop long toes and excessively sloping heels, probably leads to greater stresses to the foot than in other breeds. They also tend to have short upright pasterns, and they race at high speeds on firm track surfaces. Bilateral forelimb lameness is seen, and it can be accentuated by jogging on a hard surface. The stride is shortened with a transfer of weight to the hindlimbs. Horses typically respond to hoof testers over the central third of the frog (as in navicular syndrome). An increased digital pulse is usually evident, and DIP joint effusion may be palpated above the coronet in many cases. Younger horses (2- and 3-year-olds) with synovitis show a greater degree of localizing inflammatory signs than older horses with chronic osteoarthritis (OA).[12]

OA of the DIP joint is occasionally evidenced by the presence of osteophytes involving the distal aspect of the middle phalanx or the extensor process of the distal phalanx. Generalized suspensory soreness as well as soreness in the area of the bicipital bursa is often palpated secondary to inflammation of the DIP joint. Back pain may also be associated with the presence of sore feet and can be detrimental to racing performance due to the horse's reluctance to break sharply and extend its stride.[12] These secondary clinical signs usually disappear after resolution of the foot soreness.

Intra-articular anesthesia may be used to localize the lameness. Radiographs are frequently normal but may show some degree of pedal osteitis, and rarely, navicular syndrome changes. Problems associated with post-racing arthrosis usually occur 1–2 days after racing, when lameness is quite severe. Anti-inflammatories, ice, and systemic corticosteroids tend to correct the lameness within 12–24 hours.

Anti-inflammatory medications and corrective shoeing are often used to treat the condition. The shoeing is in accordance with individual needs, most commonly backing up the shoe as much as possible and protecting the sole. A wide variety of pads are employed with various sole packings. Wedge pads or shoes may be used to correct the low-heel conformation, but care must be taken as often the heel pain is exacerbated. Some horses are trained in bar shoes until they are ready to race. NSAIDs (phenylbutazone in particular) are useful, and the feet are iced twice daily during the acute stage.

Intra-articular corticosteroids are effective in relieving the lameness. Betamethasone esters (Betavet®) or triamcinolone acetonide (Vetalog®) and isoflupredone acetate (Predef®) with or without HA are commonly used, especially if frequent joint injection is necessary. Frequent use of methylprednisolone acetate (Depo-Medrol®) in the coffin joint can produce severe cases of OA over time.

Medial heel bruising is very common. Treatment by quartering shoes short term can become a major source of lameness in the long-term due to the time required to grow out heels and correct shoeing imbalance. Other differential diagnoses of the foot include bruises, abscesses, grabbed quarters, quarter cracks, and laminitis. See Chapters 4 and 11 for further details on lameness conditions of the foot.

The Metacarpophalangeal (MCP) Joint

The MCP joint is another frequent site of lameness in Quarter horses. The most common conditions are synovitis/capsulitis, osteochondral chip fractures, osteochondritis dissecans (OCD), OA, and fractures of the proximal sesamoid bones.

Heat and synovial effusion are the first signs of synovitis, along with a varying degree of lameness. The condition is often bilateral and radiographic examination is negative. Capsulitis does not occur as frequently as in the Thoroughbred because Quarter horses in training gallop less than Thoroughbreds do; therefore, much less stress is placed on the soft tissue structures of the fetlock joint. Symptomatic treatment includes the use of ice, leg sweats or poultice, and NSAIDS. Intravenous HA (Legend®) or IM PSGAG (Adequan®) are often used as systemic treatments. Intra-articular therapy is very effective in these cases, with a good response from corticosteroids with or without HA. If the condition does not resolve with intra-articular therapy or if it recurs after a brief period of time, the training program should be altered or the risk of further joint damage is likely, with OA as the end result.

Many 2-year-olds are entered in multiple futurities, so training revolves around these races. Trainers try to keep horses on schedule for their race dates without sustaining injuries that jeopardize their careers or require extended lay-up periods.

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Intra-articular chip fractures of the dorsal aspect of the proximal phalanx are commonly seen in the forelimb of racing Quarter horses (but less than carpal chip fragments). These fractures are considered to be traumatic hyperextension injuries. They occur primarily on the medial aspect, but may also occur laterally.[8] In some cases, they are quite large, especially compared to the fragments that occur in the Thoroughbred, with a long frontal component (Figure 9.26). Horses usually exhibit lameness and synovial effusion, and are positive to flexion of the fetlock joint.

OCD of the sagittal ridge of the metacarpus and metatarsus is usually noticed when the horse is in early training or still at the farm. It may be seen in either the MCP or metatarsophalangeal joints (more commonly, the latter). The joint is radiographed due to synovial effusion, and varying degrees of lameness may be noted. The defects are recognized on the sagittal ridge, along with varying degrees of fragmentation and loose bodies. Not all cases require surgical intervention.

OA may be seen in association with proximal phalanx chip fractures, especially in older horses, and is manifested as wear lines and erosions on the distal metacarpophalangeal articular surface.[8] Defects of the palmar surface of the distal metacarpus are far less common than in the Thoroughbred, but they do occur in older Quarter horses, and the prognosis is similarly poor.

Fractures of the proximal sesamoid bones (apical, abaxial, mid-body, basilar, and comminuted) are relatively common in the Quarter horse, despite such fractures being considered fatigue-related injuries by other authors. Although most often associated with Thoroughbred racing, biaxial and comminuted sesamoid fractures occur in the Quarter horse, resulting in disruption of the suspensory apparatus.



Figure 9.26. Radiograph showing a large P1 osteochondral chip fracture with a large frontal component. *Source*: Courtesy of Dr. CW McIlwraith.

Dorsal Metacarpal Disease

Bucked shins and stress fractures of the dorsal metacarpus are mainly problems of 2-year-old racehorses but are occasionally seen in 3-year-olds. Dorsal metacarpal disease (DMD) is a bone remodeling phenomenon of the dorsal metacarpus along the lines of stress, resulting in various degrees of periostitis and osteoporosis.[15] The incidence is less now that trainers understand this bone remodeling process as it relates to exercise. Stress fractures are most often seen in the 2-year-old year, in contrast to those in Thoroughbreds, which often sustain stress fractures from 3 to 5 years of age.[15] Many of the fractures are longitudinal and cannot be seen to exit the cortex. They may be present bilaterally with the dorsolateral cortex being the most common location, as in the Thoroughbred. Surgery is not indicated with longitudinal fractures because they heal well with rest. Saucer fractures are generally treated with lag screw fixation.

The treatments for DMD are variable, depending on the owner, trainer, and the horse's racing schedule. Some veterinarians are using a treatment combining regional perfusion of tiludronate disodium (Tildren®) with systemic use of clodronate injection (Osphos®) and extracorporeal shockwave. There is no proof at the present time of it speeding healing, and it remains controversial. While extracorporeal shockwave alone is a popular treatment, some of the older methods including electrical hyfrecation, pin firing, and periosteal scraping continue to be employed. They are all used with varying degrees of rest, depending on the level of disease present.

Proximal Suspensory Desmitis

Proximal suspensory desmitis is usually seen as an acute lameness the day after a workout or race, with profound lameness (4 on a scale of 5). Often it is the fastest horse in the trainer's barn that suffers from this injury. In the acute stage, the horse may walk on the toe without dropping the heel down to contact the ground. Perineural anesthesia of the lateral branch of the palmar nerve may be utilized to localize the lameness without blocking the middle carpal joint of the carpus. Radiographs should be taken and an ultrasound examination performed. Treatment of this condition varies depending on severity, ranging from rest to shockwave to subcutaneous injection of corticosteroids depending on the degree of desmitis.

Tendinitis of the Superficial Flexor Tendon

Superficial flexor tendinitis may be seen in the racing Quarter horse but tends to be related to racetrack surface. The incidence increases with sandier tracks, and the condition is more prevalent in horses that race at the 870-yard distance. Diagnosis and treatment are the same as for the racing Thoroughbred.

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Arthrosis of the Carpus

Carpal synovitis is the most frequent condition seen in the young racing Quarter horse. Back-in-the-knee conformation is common, and this predisposes to carpal injury during hyperextension of the joint while running. Many 2-year-olds have a large body mass for their age and reach very fast speeds without much prior conditioning. The condition is characterized by heat and synovial effusion of the affected joints with the absence of radiographic changes. Lameness may be present but is generally not severe. Carpal flexion and palpation are used to localize the affected joints. The treatment is the same as for synovitis of the MCP joint.

Osteochondral Chip Fractures of the Carpus

The incidence of osteochondral chip fractures of the carpus is very high in racing Quarter horses. Multiple chip fractures are often seen and many times they are bilateral. The distal radial carpal bone is the most common site for chip fracture, followed by the proximal intermediate carpal bone. It is not uncommon to have distal radial carpal fragments in both middle carpal joints and proximal intermediate carpal bone fragmentation in both antebrachiocarpal joints in the same horse (Figure 9.27).[13]



Figure 9.27. Radiograph demonstrating osteochondral chip fragments off the proximal intermediate and distal radial carpal bones (a common combination in the racing Quarter horse). *Source*: Courtesy of Dr. CW McIlwraith.

The diagnosis is generally made by physical examination and digital radiography. The lameness is fairly obvious; most horses have a characteristic wide placement or circumduction of the involved limb at the walk. Most cases are sensitive to flexion of the carpus and palpation of the dorsal surface of the carpal bones. Heat and synovial effusion are often present.

Arthroscopic surgery is the treatment of choice. Distal radial carpal chips are particularly associated with progressive cartilage damage and OA if the horse continues to race without removal of the chip fracture. Many Quarter horses have multiple surgeries during their racing careers due to the high incidence of osteochondral chip fragmentation.

Other important injuries include the more severe fractures (slab fractures and comminuted fractures), many of which are amenable to surgery. Whenever significant lameness is evident in the carpus, a thorough radiographic examination including skyline views of the carpus is necessary to ensure that degenerative lesions, incomplete sagittal fractures, and any nondisplaced fractures may be seen before a catastrophic injury occurs. All of the reported third carpal bone fractures can be seen in the Quarter horse, with a higher percentage of large frontal slab fractures involving both the radial and intermediate facets (Figure 9.28). The slab is often displaced in these cases, with the distal margin of the radial carpal bone collapsing into the proximal fracture site (Figure 9.29). Lag screw fixation is necessary for proper healing and to prevent further collapse of the joint.

Fractures involving both rows of carpal bones may be seen with some injuries. These can be challenging surgical cases due to the severe damage to the carpal bones and associated joint instability, requiring partial or panarthrodesis and the use of multiple bone plates. Most of these injuries can be managed successfully by using aggressive internal fixation techniques to produce pasture-sound breeding animals. Prompt surgical intervention is necessary due to the high risk of laminitis in the contralateral limb.[9]



Figure 9.28. Skyline view of the third carpal bone with a slab fracture involving both radial and intermediate facets. *Source*: Courtesy of Dr. CW McIlwraith.

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Hock Lameness

Effusion of the tarsocrural joint in the hock may be indicative of OCD lesions associated primarily with the distal intermediate ridge or medial malleolus of the tibia. Often these cases are operated before arriving at the track. Lameness associated with the distal tarsal joints, as well as other hind end lameness, is associated with a failure to break sharply from the starting gate. Upper hindlimb flexion tests may be equivocal, but a positive Churchill test along with the history of a poor performance may indicate tarsitis, although a negative test does not rule out the problem. The condition is generally bilateral, and the horse may track close behind or cross midline when observed from behind.

Horses that wear patches behind to protect from scalping or horses with laceration marks seen on the medial aspect of the hock are highly suspect for hock soreness. Radiographs are negative in many instances, or subtle changes may be seen. Intra-articular corticosteroids are effective at relieving the lameness and/or improving performance. Even though the tarsometatarsal and centrodistal joints are low-motion joints, maintaining articular cartilage is important because these joints rarely fuse on their own. Betamethasone esters (Betavet®) and triamcinolone acetate (Vetalog®) have been shown to have fewer deleterious effects on cartilage than methylprednisolone acetate (Depo-Medrol®).[5,6]



Figure 9.29. Radiograph of a collapsing slab fracture of the third carpal bone with the distal margin of the radial carpal bone collapsing into the proximal fracture site. *Source*: Courtesy of Dr. CW McIlwraith.

If methylprednisolone acetate is used, low doses should be considered (20–40 mg).

Stifle Lameness

The most common site of stifle pain is the medial femorotibial joint, which is the same in racing Thoroughbreds. This can be another cause of poor performance. The horse may be positive to upper hindlimb flexion or palpation, but most often intra-articular anesthesia is required to localize the lameness. It is not always clear whether the condition is synovitis or early OA. Radiographs are useful to assess the joint and rule out certain conditions. Ultrasound examination is useful to pick up soft tissue conditions.[4]

Most stifle lameness responds well to intra-articular therapy; however, if lameness persists, diagnostic arthroscopy is necessary to make a definitive diagnosis. Lameness conditions involving the femoropatellar joint are often accompanied by the presence of joint effusion, but in recent years most OCD lesions are operated on before the horse starts racing. Upward fixation of the patella can be an issue in immature racehorses in early training. Subchondral cystic lesions of the medial femoral condyle are a relatively rare but painful condition in the racehorse.

Tibial Stress Fractures

Stress fractures involving the tibia are seen in young horses around the time of their second qualifying work or first race. Diagnosis of the condition has become more common with the advances in imaging techniques and the access to nuclear scintigraphy. The lameness is unilateral and quite severe; the left hindleg is predominantly affected. This may be due to the fact that the horse pulls up quickly from high speed before entering a left-hand turn on the racetrack. If a tibial stress fracture is suspected, nuclear scintigraphy is the best way to demonstrate the injury. Alternatively, digital radiographs taken 1 week to 10 days after injury may show a lesion on the tibia. The horse is usually rested for at least 90 days before resuming training.

Catastrophic Fractures

Proximal sesamoid fractures are the greatest cause of catastrophic injury in the racing Quarter horse. A retrospective study of racing fatalities found that carpal bone and vertebral body fractures were more common in Quarter horses than Thoroughbred racehorses. Sprinting vs. distance racing may play a role in a different distribution of skeletal injuries, but the greatest cause of death for both breeds was found to be fetlock injury.[16]

Lumbar vertebral fractures of the spine have been associated with serious injury to jockeys due to the fall of horse and rider.[3,11] The fractures are located at the L5–L6 vertebral junction. Pathology of the lumbar vertebral region appears to be present before catastrophic injury and is likely associated with maladaptive pathology at the L5–L6 location (Figure 9.30).[3]

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Figure 9.30. Parasagittal (A) and transverse (B) computed tomography images illustrate the cranioventral to caudodorsal oriented fracture and abnormal bone proliferation (arrows) on the ventral aspect of L5. *Source*: Reprinted with permission from Collar et al.[3]. Reproduced with permission of Equine Veterinary Journal.

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THE WESTERN PERFORMANCE HORSE

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INTRODUCTION

Western working stock horses are highly talented and bred to be extremely athletic. Its genetic instinct to control a cow, plus overall athletic ability, produces the near-perfect scenario for training- and competition-related injuries with subsequent pathologies. The role of attending veterinarians is to diagnose injuries accurately and initiate appropriate therapy as soon as possible, thereby minimizing recovery time. A thorough understanding of the nature of the horse's competition is necessary for advising the trainer and owner in the proper management of each athlete.

TEAM ROPING HORSES

Description of Event

Team roping is a timed event that begins with a horned steer weighing approximately 200–300 kg that is contained in a chute at the end of the arena. The heading box is to the left of the steer, and the heeling box is to the left of the chute. The first member of the team, the header, asks for the steer to be released from the chute, and the steer is allowed a head start, termed the score. The team is assessed a 10-second penalty if the header leaves the heading box before the steer crosses the score line. When cued by the rider, the heading horse leaves the roping box and chases the steer at maximum speed, much like a racehorse leaving the starting gate. As the header approaches the steer, it "rates off" or slows slightly as it reaches the steer's hip to allow the rider to rope the steer's horns. After the steer is roped, the rider wraps the rope around the saddle horn (dally), and the heading horse drops its hindquarters to slow the steer's momentum. The heading horse is then turned 90° to the left and pulls the steer across the arena, maintaining a constant reduced speed that allows the rider of the heeler to get into position to rope the hindlimbs of the steer. After the heeler's rider ropes the hindfeet and slack is drawn out of the heel rope, the rope is dallied around the saddle horn, and the heel horse is signaled to drop its hindquarters and come to an abrupt stop. The heading horse turns 180° to face the heeler, which signals the end of the run, at which point time is taken. A good time for the completed run is 7–8 seconds; the world record is 3.2 seconds.

Musculoskeletal Injuries

One study that identified the prevalence of specific musculoskeletal injuries sustained by horses used for team roping also showed differences between horses used specifically for heading vs. heeling. Horses used for heading were significantly older (12 vs. 9 years) and heavier (545 vs. 500 kg) and at greater risk of lameness than horses used for heeling.[1] All were Quarter horses, and all that presented for poor or change in performance were lame.

The type of performance change reported by the owners differed between horses used for heading or heeling. The primary owner complaint in horses used for heading was that the horse stopped pulling the steer after it was roped, followed by the horse acting nervous in the heading box and the horse failing to slow down (rate) after reaching the steer to allow the roper to throw his rope. The primary owner complaint in horses used for heeling was that the horse quit stopping after the steer's feet were roped or the horse would nicker when asked to stop.

The right forelimb was most frequently affected by lameness in horses used for heading, whereas the left forelimb was most frequently affected in horses used for heeling. Horses used for heading had significantly more bilateral forelimb lameness, and those used for heeling had significantly more bilateral hindlimb lameness.

As the heading horse sets the steer and initiates the 90° turn to the left, the right front limb is placed cranially and laterally to decelerate and brace against its forward motion and the weight of the steer, placing a tremendous amount of weight and strain on the structures of the right forelimb. These actions and forces may explain the frequency of right forelimb problems in horses used for heading. As the heading horse turns the steer to the left, the heel horse changes directions and makes a quick left turn to position itself to the inside of the steer's left hip so the roper can throw his rope, which may account for the left forelimb involvement. The horse then stops abruptly which could place stresses on the hindlimbs (Figure 9.31).

The most common musculoskeletal problems diagnosed in horses used for heading were:

1. Pain in the navicular area only
2. Navicular area pain with distal tarsal joint osteoarthritis (OA)
3. Soft tissue injury in the pastern region of the right forelimb
4. Carpal joint OA

Heeling horses were most commonly affected by:

1. Navicular area pain
2. OA of the metacarpophalangeal joint
3. Distal tarsal joint OA
4. OA of the left forelimb proximal interphalangeal joint

The joints of the majority of horses were treated with intra-articular medications; the distal interphalangeal joint was most frequently treated.

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Figure 9.31. A team roping run just after the steer has been roped and the head horse and rider turned 90° so the heeler can rope the heels. Note the right front limb on the heading horse and left front limb on the heeling horse.

TIE-DOWN AND BREAKAWAY ROPING

Description of Event

Tie-down roping, previously called calf roping, is another timed roping event; however, it involves only one horse and rider and one calf without horns. Similar to team roping, the rider backs his horse into the roping box located to the right of the calf, which is confined in the chute. The calf is given a head start, the score, which varies depending on the length of the arena and the level of the roping event (professional calf ropers have a longer score compared with amateur ropers). A 10-second penalty is assessed if the horse and rider leave the roping box before the calf reaches the predetermined score line. The horse and rider chase the calf and the rider ropes it around the neck. The rider dismounts as the rope comes tight and must grab the calf's flank and put him on his side on the ground. The roper then ties three legs together with a pigging string that he carries during the run. The rider gets back on his horse, and the calf must stay tied 6 seconds or he is disqualified. As the rider dismounts, the horse comes to a sudden stop (Figure 9.32) and backs up to take excess slack out of the rope. This allows the roper to catch and flank the calf. The calf roping horse constantly works the rope or keeps it tight throughout the entire run, placing a tremendous amount of pressure on the hindlimbs. A good tie-down roping time is 8–9 seconds.

Breakaway roping is similar to tie-down roping except the ropers are women or children and the rider does not dismount and tie the calf. The roper ropes the calf around the neck with a breakaway rope—a normal rope equipped with a breakaway device that releases the calf as soon as the rope becomes tight. Horse and rider chase the calf, the calf is roped around the neck, and the horse stops quickly, allowing the rope to come tight, at which point the breakaway rope opens. Time is stopped when the rope breaks free. A good time is 3–4 seconds for the run.



Figure 9.32. Tie-down roper after roping the calf. As the roper dismounts, the horse comes to a quick stop and backs up to take the slack out of the rope, allowing the roper to tie the calf.

Musculoskeletal Injuries

The stop is critical and demanding in these horses. The horses are running at top speed and then must come to a complete stop very quickly. Hindlimb lameness issues are very common. The most common musculoskeletal injuries seen by the author are:

1. Distal tarsal joint OA
2. Medial femorotibial joint pain or arthritis
3. Hindlimb suspensory ligament desmitis and hindlimb flexor tendinitis

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4. Hindlimb fetlock OA
5. Hindlimb pastern joint OA
6. Forelimb foot pain

BARREL RACING

Description of Event

Barrel racing is a timed event. The pattern consists of three barrels (55-gal. steel drums) in a cloverleaf pattern. The distance covered for the pattern varies with arena size, but generally the distance from the starting line to the first barrel is 14–18 m. The distance between the first and second barrel is 21–27 m and between the second and third is 27–32 m. The horse and rider start at the alleyway or entrance into the arena, run at full speed to the first barrel (the contestant chooses to take the right or left barrel first), and then slow down (rate) and complete a 360° turn around the first barrel. The horse and rider then speed up and run toward the second barrel and turn 360° around it (Figure 9.33), repeat the process with the third barrel, and then sprint to the finish line. The fastest time to complete the pattern varies with arena size, but a good run for a large arena pattern is 15–16 seconds. If a rider gets too close to a barrel and tips it over, a 5-second penalty is assessed. If the horse and rider fail to negotiate the pattern correctly, they are disqualified.

Although the event is called barrel racing, it is important for the veterinarian to realize that these horses do not sustain the same type of injuries encountered by flat racing horses. The barrel horse does not run at top speed over long distances like flat and steeplechase racehorses; thus, injuries generally associated with speed and fatigue (such as superficial flexor tendinitis or metacarpophalangeal and carpal chip fractures) are usually not common in barrel-racing horses.

A recent study evaluated musculoskeletal injuries sustained by more than 100 horses used solely for barrel racing.[2] The median age was 9 years and the horses weighed an average of 509 kg. Sixty percent were examined due to an owner complaint of lameness, and 40% because of a change in the horse's performance. The most common performance problem reported by owners was that the horse refused to turn correctly around the first (usually right) barrel or ran past it and down the arena. Other complaints included a decrease in the horse's speed, failure to enter the arena, and failure to take the correct lead around the barrel.



Figure 9.33. Barrel horse and rider turning a barrel.

Musculoskeletal Injuries

The right forelimb was most frequently affected by lameness (48%), followed by the left forelimb (43%), left hindlimb (26%), and right hindlimb (21%). Bilateral forelimb lameness (26%) was more common than bilateral hindlimb lameness (5%). The majority of horses that had a performance problem related to the first (right) barrel had right forelimb lameness.

The most common musculoskeletal problems diagnosed in horses used for barrel racing were forelimb foot pain only (33%), distal tarsal joint OA only (14%), suspensory ligament desmitis (13%), forelimb foot pain with distal tarsal joint OA (10%), and bruised feet (9%). Eighty-one of 118 (69%) horses had the affected joint(s) treated with intra-articular medications.

The majority of barrel horses begin the cloverleaf pattern by going to the right barrel. The horse runs at full speed and then slows to turn 360° around this first barrel. The first barrel is the hardest to turn because at this point in the pattern, the horse is at maximum speed. As the horse turns to the right, the inside right forelimb may be under great strain. As the horse approaches the two left turns (second and third barrels), its speed is slower than it was for the initial approach to the first barrel, which may account for slightly less left forelimb lameness. Perhaps these horses refuse to turn sharply to the right because they have subtle right forelimb lameness and are trying to avoid a painful stimulus. This has been previously suggested by other veterinarians who work on barrel racing horses.

REINING HORSES

Description of Event

Reining horses are judged individually as they complete 1 of 11 specified patterns (Figure 9.34). One or more judges score each horse between zero and infinity, with 70 denoting an average score. Each horse automatically begins the pattern with a 70. The judge can either add or deduct up to 1.5 points on each maneuver in half-point increments based on the quality of the maneuver. Penalties are also allocated for minor deviations from the pattern; major deviations result in a zero score for the go. As the judges watch the execution of the pattern, individual scribes keep track of each judge's maneuver scores as well as penalty marks on a score sheet. Scores are tabulated and announced at the end of each run.

The judge's sheets with individual maneuver scores, penalties, and total scores are then posted for the benefit

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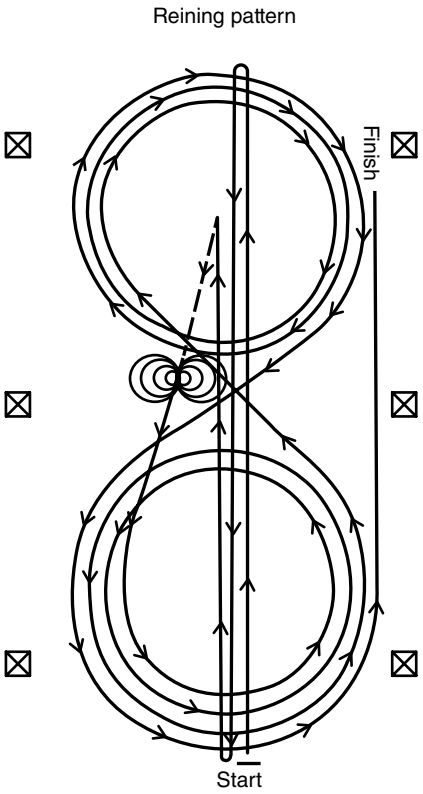


Figure 9.34. Reining horse pattern.

of the exhibitors following each class. In scoring, credit is given for smoothness, finesse, attitude, quickness, and authority when performing the various maneuvers. Controlled speed in the pattern raises the level of difficulty and makes the reining horse more exciting and pleasing to watch. An increased level of difficulty is rewarded with higher scores if the maneuvers are performed correctly.

Musculoskeletal Injuries

The precision maneuvers performed at top speeds by reining horses require implementation of good preventative and balanced shoeing principles. These horses start from a standstill, gallop at top speed, and come to a 30-foot sliding stop. They perform turnarounds in which their body whirls around the inside hindfoot as the forelimbs reach and cross over in a precise and smooth motion.

These horses are often plagued with distal limb bone bruises, fractured splints, and coronary band injuries from the turnaround spins. The high-speed and lateral movements also lead to unilateral hoof impaction and forces that can cause high quarter cracks and blow out cracks.

Half-round shoes are often used on the front feet to ease break-over in all directions, especially with all of the lateral movement that these horses are required to perform. However, some trainers feel that the web of the half round is not wide enough for support and prefer the rounded edges of polo or rim shoes on the front feet. The sliding stops require a sliding plate on the hindfeet



Figure 9.35. Sliding plate used on the hindlimbs of reining horses so that they will slide after a stop. *Source*: Courtesy of Jeff Ridley.

(Figure 9.35). These are usually made from 0.75- to 1.25-inch × 0.25-inch flat steel. The extended heels add support and balance to the stop, and the extended heels rarely extend past the heel bulbs so that they do not cut the opposite leg during the turnaround. Most seasoned horses wear a 1-inch sliding plate throughout the shoe with equal length branches, which provides good support and adequate traction.

Many reining horses are plagued with hindlimb tendon and ligament injuries, of which hindlimb proximal suspensory desmitis is most common. This is likely due to the extensive sliding stop that they are asked to perform.

CUTTING HORSES

Description of Event

Cutting horses were born of necessity on the open grass plains of West Texas. They provided big ranches without barbed wire fences with the only means of working vast cattle herds. The task of the horse was quite simple, at least by definition. Guided by his rider, the cutting horse would enter a herd of cattle quietly and deliberately. A single cow was cut, or separated, from the herd. The cow's natural instinct is to return to the safety of the herd. The cutting horse, through breeding and training, controlled the calf with a series of moves and counter moves. The speed, agility, balance, and quickness of the cutting horse kept the cow from the herd, where other cowboys would hold the cut. The horse and rider would reenter the herd again and again, cutting cattle out until the work was done.

Training of today's cutting horse begins early in its 2-year-old year with the goal of having a competitive 3-year-old futurity horse. After 90 days under saddle, the horse is introduced to cattle. The objective is to have the horse instinctively anticipate and mimic the movements of the cow, using quick stops and turns, while receiving minimal instruction from the rider. Reining the horse is permitted only to make the cut of a single

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cow from the herd. After the cut is successfully made, the reins are placed in a relaxed position on the horse's neck, and only leg cues are permitted from the rider during the actual working time. The instinctive ability of the working cow horse to contain the individual cow provides the excitement of competition in cutting. During the competition, the horse and rider are judged on how smoothly the cow is separated and kept from the herd, keeping the cow in the middle of the arena and having the horse work without hand cues from the rider. Each contestant gets three cows per performance. The rider and horse as a pair begin the competition with a score of 72, going up or down from there; a 74–76 is a good score.

Musculoskeletal Injuries

Due to the twisting and quick side-to-side movements (Figure 9.36), these horses are plagued with numerous hindlimb, pelvis, and thoracolumbar problems. One retrospective study has documented poor performance and musculoskeletal injuries in 200 horses used solely for cutting (Dabareiner RM et al., unpublished data). The majority of horses were Quarter horses with a median age of 5 years. The most common performance complaint by owners was that the horse would not reverse direction to follow the cow (55%), followed by improper stopping (18%). On physical examination, 70% of horses were noted to have thoracolumbar pain upon back palpation. Eighty percent of horses had peripheral or intra-articular diagnostic anesthesia performed to localize the source of lameness. One hundred twenty-six of 200 horses (63%) had hindlimb lameness only. The most common hindlimb lameness was pain originating from the medial femorotibial joint in the stifle (40%) followed by distal hock pain (20%) and hindlimb suspensory desmitis. Forelimb lameness was uncommon with proximal suspensory desmitis and foot pain being most common.



Figure 9.36. Twisting and rotation of the body as the cutting horse mirrors the movement of the cow.

Radiographic abnormalities of the joints may not be apparent in young cutting horses with stifle or distal tarsal joint inflammation. Treatment options consist of short periods of rest combined with NSAIDS and often IV sodium hyaluronate. Most trainers and owners prefer to keep the horse in work and elect for intraarticular medication of the medial femorotibial joint and/or both the distal intertarsal and tarsometatarsal joints with a combination of corticosteroids and sodium hyaluronate. Many of these horses will also be painful to palpation in the lumbar region of the back.

In horses with evidence of soft tissue injuries or radiographic changes, longer periods of rest are usually required. Hindlimb proximal suspensory desmitis does occur in cutting horses. Mild cases with minimal sonographic changes respond well to 6–8 weeks of stall rest combined with local injection of hyaluronic acid and corticosteroids. Horses with chronic, severe lesions require additional therapies and usually have a guarded prognosis for returning to athletic function.

Young cutting horses with hindlimb lameness may suffer concurrently with thoracolumbar myositis. These horses are painful on palpation of the back. They are often agitated when saddled or mounted and unwilling to stop or perform. Therapy is aimed at reducing the local inflammation and preventing muscle spasms. Rest is indicated but rarely is an option due to the horse's performance schedule. Systemic use of muscle relaxants such as methocarbamol (10-mg/kg PO BID for 10 days) may be beneficial in mild cases. Others may respond to a single injection of triamcinolone acetonide (12–16 mg, IM) in addition to the muscle relaxants.

Specific localized pain may be treated successfully by injecting anti-inflammatories directly into affected tissues. The combination of methylprednisolone acetate (200–400 mg) and sarapin (50 mL) locally infiltrated aseptically with 2- to 3.5-inch needles is the author's treatment of choice. The injections are placed into areas of palpable pain by local infiltration.

Other management considerations are important to the recovery. Horses with low underrun heels in the hindfeet should be shod to encourage additional heel support and raise the heels. Typically, a 2°–3° lift is indicated in the stock horse. Evaluation of the saddle fit, type of pad, and specific pressure points when ridden should be considered.

Other modalities such as pulse electromagnetic field and ultrasound therapy have been very useful in keeping these horses in competition. Long warm-up periods of ponying the horse at a walk and trot for 30–45 minutes without the rider are always indicated. The trainer must be cautioned to avoid overwork and severe fatigue at all times.

Strain and subluxation of the sacroiliac joint are not uncommon in the working stock horse. Unlike other types of horses that incur sacroiliac desmitis as a result of slipping or falling, the stock horse often incurs this type of injury from the twisting and rotation of the back and pelvis during work. This rotation is complicated by the weight of the tack and rider, who is attempting to maintain balance and remain stationary on top of the horse during sudden hard stops, turns, and bursts of speed.

Many of the symptoms observed with thoracolumbar myositis are also common with sacroiliac desmitis.

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A painful reaction to digital palpation of muscles in this region is common because of the bridging effect. The muscles undergo bridging during contraction to provide stability to the traumatized sacroiliac joint. This constant state of contraction leads to muscle fatigue, spasms, and pain. Unlike thoracolumbar myositis, observable pelvic limb lameness is often associated with unilateral sacroiliac strain and subluxation.

Clinical signs are extremely variable but include stiffness and alteration of gait, either bilaterally or unilaterally. Often the horse will show a right hindlimb lameness when the left hindlimb undergoes an upper limb flexion test or vice versa. Infiltration of local anesthetic into the suspected area of trauma may help in diagnosis, but seldom is 100% improvement achieved.

Therapy is similar to previously described treatments for thoracolumbar myositis. Deep intramuscular injections of methylprednisolone (400 mg) and sarapin (50 mL) into the region of the sacroiliac joint have been very effective. Disposable needles at least 4 inches long are necessary to reach the affected area. Strict aseptic technique must be rigidly followed. Concurrent systemic therapy with NSAIDs is beneficial in sacroiliac desmitis. All classes of significantly lame horses with sacroiliac instability and subluxation must have rest. Two to six months out of training is often necessary, with strict stall confinement for the first 30–45 days.

The unpublished study by Dabareiner RM et al. is the first clinical study that documented an increased incidence of hindlimb lameness versus forelimb lameness in any horse discipline. It is also the first to document the stifle as the most common joint affected in a specific discipline. Given the increasing popularity of the cutting discipline, this information should be very useful to clinicians working on cutting horses.

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JUMPING, EVENTING, AND DRESSAGE HORSES

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INTRODUCTION AND HORSES USED FOR THE DISCIPLINES

Jumping, dressage, and 3-day eventing constitute the three Olympic equestrian disciplines, included since 1912. Their modern form developed mostly from military institutes and in recent decades became a civilian sport. All three disciplines have become increasingly popular and are widely practiced at various levels of performance. The United States Equestrian Federation (USEF) assures the governance of the sports in the United States, and the Federation Equestre Internationale (FEI) at the international level worldwide.

Many breeds are used for these disciplines, especially at lower levels. As the level increases, particular breeds better suited for the disciplines become predominant.

The degree of specialization, adoption of specific training methods, the structure of the competitions, the selection of horses bred for them, and the level of competition predispose these horses to specific occupation-related injuries.

Dressage

Dressage is a challenge of combining the need for balance, obedience, suppleness, and hindlimb engagement. The objective is to have the horses move as asked giving the impression that the horse is doing it willingly and generously. The horse needs to move upon the slightest command from the rider with rhythm, looseness and suppleness, and energy while remaining straight and in contact with the bit.

Grace, power, athleticism, and balance are needed along with the compatibility for complete obedience. High-level dressage horses are almost exclusively within the Warmblood breeds, with a few Thoroughbred crosses and a few Iberian horses (Spanish and Lusitano).

A study comparing gait and conformation of several Warmblood and Spanish horse breeds (3 years old) including Hanoverians, Westphalians, Oldenburgs, and Selle Francais and Spanish saddle horses concluded that the German Warmbloods, in particular Hanoverians, were the most adapted for competitive modern dressage.[1] This is confirmed in the show rings, as Hanoverians are the most represented breed at the elite level.

Jumping/Hunting

Show jumping involves the athletic effort of jumping added to speed. This will require balance, power, and agility as well as the carefulness not to hit the rails from the horse. The objective is to jump a course obstacle with set distances between them, without knocking rails down or having the horse refuse a jump, in the minimal amount of time.

The level of horse/rider coordination is of extreme importance especially as the courses get more technical (turns and distances between obstacles) and the jumps higher.

Show hunting is specific to North America, and although it still requires jumping of fences without knocking down rails, more importance is given to the elegance of the jumping style of the horse and rider (Figure 9.37). The horses need to travel elegantly, always in balance with barely noticeable command from



Figure 9.37. A show hunter. Manners, way of going, and style of jumping are being judged. *Source*: Courtesy of Dr. Eric Swinebroad.

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HORSESthe rider and jump fluidly with ease, style, and scope. Competitions are scored subjectively.

Thoroughbreds, Warmbloods, American Quarter Horses, and their crosses are commonly used as hunters and jumpers. Several breeds of ponies are often used for children. The appearance of the horse, its proportions, and the jumping styles are important, especially for the hunters as they are judged subjectively. Many European Warmblood breeds (Selle Francais, Hanoverians, Dutch Warmbloods, etc.) have been selected for several decades, with the help of government run programs (i.e. Haras Nationaux in France), using objective data and rigorous selection programs to produce competitive show jumpers and are dominating the show circuits.

Eventing

Eventing consists of the consecutive combination of dressage, cross-country (named previously speed and endurance), and jumping tests. Horses must show proficiency in each one of these disciplines with the cross-country being the most important (Figure 9.38). Horses need to show some of the qualities needed for dressage and jumping, with added emphasis on speed and endurance. Competitions especially at higher levels are extremely demanding on the horse's physic as well as relatively dangerous for both the horse and rider.

Partially because of their relative lesser financial value, and because of the need to be proficient at the different tests, event horses often get into eventing as a second career (often after being suboptimally successful racing). Because of the accrued need for endurance and speed for the cross-country phase, most of the elite event horses are Thoroughbreds or Thoroughbred crosses. Australian, New Zealand, and Irish Thoroughbreds are particularly appreciated at the elite level because of their bigger frame.

STRUCTURE OF TRAINING AND COMPETITION

In comparison with racehorses, training of horses used for dressage or jumping starts much later. Horses are usually broken at 3 or 4 years of age. Often it is not until they are 10–12 years old that they reach their prime in performance.

Dressage

Training of the dressage horse after it has been backed follows a pyramid training scale. At the bottom of the pyramid is "rhythm and regularity" (Takt) that should remain the same through the different exercises and their combination.

"Relaxation" (Losgelassenheit) or looseness is the second level: the aim is to have the horse relaxed, loose at the pole, chewing at the bit, swinging his neck and tail along the strides, and making smooth transitions between gaits.

The third level is "contact" (Anlehnung) at the bit and should result from the horse's forwardness instead of the pulling by the rider's hand.

Fourth level is "impulsion" (Schwung), which is created by storing the energy of engagement. "Straightness" (Geraderichtung) and "collection" (Versammlung) are the last two levels. Collection is the horse's ability to move its center of gravity more backward, by taking more weight on the back end, lowering it, and lightening the forehand. This requires more energy and muscular strength and allows the performance of more advanced exercises such as "piaffe" and "passage."

Competition usually starts at the age of 5; these horses can reach medium levels by age 7–8 and for some of them Grand Prix level by age 8 or 9. Under FEI rules, no dressage horse can show under the age of 6. Lower-level competitions require mostly balance and freedom



Figure 9.38. A cross-country obstacle.

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of movement, and then as the level raises, so does the demands on lateral work and collection. Under the USEF governance, competitions are progressively ranked as "training" and then first level through fifth level that corresponds to international competitions. In Europe it is divided in three progressive levels (L, M, and S). S corresponds to international competitions. International competitions (under FEI governance) starts at Prix St Georges, Intermediate I, Intermediate II, Grand Prix, and Grand Prix Special. Shows are also classified by order of competitiveness and difficulty as CDI* (Concours de Dressage International) to CDI***. Competitions are judged by anywhere from one to five judges who score each one of the movements/figures, giving a score from 0 to 10. The scores of each figure are added and converted into a percentage.

The training and competition surfaces are usually well cushioned, most often artificial. Between show grounds, the surfaces are very consistent and close to ideal and therefore rarely blamed for contributing to lameness problems.

Dressage horses do not perform any speed work or high impact work, which shields them from acute traumatic orthopedic injuries. Their training does involve repetition of similar exercises, and this makes them more prone to chronic accumulative stresses, resulting in subclinical injuries that can ultimately or periodically cause decreased performance, unwillingness to perform some tasks and sometimes chronic lameness.

The collected and lateral work (Figure 9.39), together with the fact that the saddle is placed relatively far back and that the rider is almost exclusively sitting, places unique strains on the dressage horse. This is accentuated by the fact that they have relatively long careers; as many compete until 15–20 years of age, most are at their peak between 8 and 14 and compete after that at lower levels with less experienced riders (as schoolmasters).



Figure 9.39. A dressage horse executing a half pass.

Jumping

Young jumper prospects are often free jumped before getting broken (between ages 2 and 3). In some breeding programs, weanlings are free jumped, and a first selection is done at 6 months, based on their jumping style. One study found that superior jumping style could indeed be detected at such age, although with some variability.[34] Also, foal jumping technique was found to improve with training and had a positive impact on their jumping technique at age 4 when compared with untrained foals, although the difference did not carry over when horses reached 5 years of age, rendering the early training unnecessary.[35]

After they are broken, training of the hunter jumper involves basic flat training, similar to basic dressage training, emphasizing collection and engagement work. After a few months of training on the flat, they are brought over fences. As the training continues, the fences usually get higher, but also horses are trained to jump combinations of fences and jump fences at different angles, at different speeds, and from different distances. A lot of work is put into teaching them to modulate their stride length and speed while approaching the fences.

Hunters are trained to jump in a relaxed fashion, giving the impression of jumping effortlessly, with the jump being a smooth continuation of the previous strides (jumps "in stride"). Completions are scored subjectively, on the style and cadence of the horse and rider. The height of the jumps starts at 2′6 and increases with the levels but does not exceed four feet. It is not uncommon practice to ride hunters or lunge for a long time just before the competitions to "take the edge off."

For jumpers, less attention is given to the style of moving and jumping. The competitions are scored objectively based on fallen rails, possible refusals and time of completion of the course. Height of rails varies between 0.90 and 1.50 m depending on the level of competition at the USEF level (although can go higher in Puissance), and up to 1.70 m for FEI competitions.

There is a multitude of classes and levels depending on the status of the rider (amateur vs. professional) and his age (adult vs. junior) and the age, experience, and quality of the horse (i.e. young horse classes). As the level rises, so do the height of jumps and the technicality of the courses.

The competition season can be exceptionally long for hunter and jumpers as there are shows running every single week of the year. It has become the norm for

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Figure 9.40. A show jumper. Note the hyperextension of the hind fetlocks.

horses to show several weeks every month and often travel long distances in between shows.

At higher levels horses will be conditioned and prepped for a few key events a year. At each show, hunters will tend to be ridden in several classes every day for up to 4 days of the show week. Jumpers will typically only be ridden in one to three classes that week. The longevity of jumpers' and hunters' careers and the particular strains placed by the jumping (Figure 9.40) also contribute to the development of occupational injuries.

Eventing

Event horses start their dressage and jumping training at the age of 4. Some of them have had prior career as a racehorse. The cross-country training includes the familiarization to the cross-country obstacles but more importantly the horse's fitness. Horses start competing at lower levels around the age of 5 and can reach international level by age 7–8.

Competitions start at the lower levels (pre-novice and novice) with 1-day events, during which all three tests are conducted. Different levels for the USEF-regulated competitions include in order of increasing difficulty, pre-novice, novice, training, preliminary, intermediate, and advanced levels.

International competitions are sanctioned by the FEI and are divided between Concours International Combine (CIC) and the more advanced (more jumps and faster rate) Concours Complet International (CCI). CIC competitions are further rated with one to three stars, increasing with the level of difficulty.

The top level of international competition is the 3-day event or "Concours Complet International." It is conducted over 3–4 days: 1 or 2 days of dressage, 1 day for the cross-country, and 1 day for the stadium jumping. The cross-country use is divided in four phases (classic form of the competition), including a trotting warm-up (phase A), the steeplechase (phase B), a cool down (phase C), and finally the proper cross-country course with 30–45 fixed obstacles (phase D). There is significant veterinary involvement before and during each phase of the competition because of how strenuous it is on the horse's physic.

The new format of the CCI competition eliminated phases A, B, and C in an attempt to make this less strenuous on the horses starting in 2002. However, there is still debate on the real effect on the horse by changing the format. The difference between CCI and CIC became only the number of jumps, distances, and level of difficulty.

CCI competitions are rated with one to four (highest-level) stars. There are very few CCI**** worldwide every year: the Olympic games, the FEI World Equestrian Games, the Burghley Horse Trials (UK), the Badminton Horse Trials (UK), the Australian International Three-Day Event in Adelaide (Australia), the Rolex Kentucky Three-Day Event (USA), the Luhmuhlen Horse Trials (Germany), and the Stars de Pau (France). A horse usually competes in only 1–2 CCI**** per year.

Horses competing at this higher level usually use 1-day events and lower-level competitions for training and as fitness indicators.

Overall, fences for the jumping and cross-country phase vary between 1 and 1.30 m in height and 1.40–2 m in spread (Figure 9.41). The cross-country distance varies from 2,500 to 7,200 m. The length and required speed for these competitions, especially at high levels, pushes these horses close to their metabolic and musculoskeletal limits.

TRAINING SURFACES AND SHOEING

Training surfaces between the three disciplines vary considerably. Dressage training and competition arena are relatively standardized, are most often made of artificial material, and provide great cushion. Three-day event horses are at the other end of the spectrum as they have to tackle various surfaces from hard ground to wet mud. Jumpers compete in a variety of surfaces, meant to be somewhat controlled, varying from sandy rings to grass fields, with increasingly synthetic geotextile footings.

Deep soft footing predisposes them to multitude of soft tissue lesions related to fatigue, including suspensory desmitis, superficial digital flexor tendonitis, inferior check ligament desmitis, and gluteal myositis. Hard surfaces predispose to impact injury, such as foot soreness, and joint trauma, bony bruising, and also axial skeleton pain. Competition on grass fields promotes the use of studs. Studs are placed (one to three on each shoe) on the shoes of the front or both front limb and hindlimb. The use of only one stud per foot offsets the lateral medial balance and results in abnormal torque forces.

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Figure 9.41. An event horse landing after a cross-country jump. Note the hyperextension of the front fetlocks. *Source*: Courtesy of Dr. Eric Swinebroad.

The use of studs on both sides of the shoe is less problematic from a lateral medial point of view but might exacerbate hyperextension of the forelimbs by preventing, upon landing from a jump, the normal minimal sliding of the front feet. The addition of a third stud usually on the lateral toe side of the shoe is also common in front shoes.

Synthetic geotextile footings are becoming increasingly prevalent. Although they are rarely too deep or slippery, they tend to negate the natural slipping that happens upon landing from jumps, increasing the torque forces and to some extent the impact at that phase.

Typically, a horse will get shod at least 1 week before any significant competitions, which will allow correction and sufficient rest in the case of a shoe set to tight or a nail issue. An added constraint on the competition horses from a shoeing stand point is that the constant traveling to shows may result in various farriers trim and shoe them (when the regular one is not set to travel). This can result in lack of familiarity of the farrier to the particular horse's feet and the loss of subtle fine-tuning.

LAMENESS DIAGNOSIS

Lameness evaluation on performance horses should be preceded by a good knowledge of the medical/orthopedic history, the current and past performances, and any changes in training, riders, or shoeing.

Palpation of the horse's joints, tendons, and back is performed routinely as well as limb flexion tests and finally evaluation of the spinal range of motion. Hoof testers are then applied. Horses are evaluated in motion, at the walk, on straight line, and on short circles and then at the trot and canter, ideally both on hard and soft surfaces. It can be extremely fruitful to evaluate some horses under saddle. Many elite barns have horses evaluated on a weekly basis to detect any small changes enabling them to address problems early on and modulate workload accordingly.

Barring constraints from the timing of future shows or regulations at the particular show (FEI vs. USEF vs. national regulations of other countries, for the limits of detection of traces of local anesthesia agents) and depending on the severity of the gait abnormality, local and/or regional blocks could be performed. Perineural nerve blocks are often combined with synovial blocks as needed to gain more specificity.

This process might take several days as it is often needed to let a block wear off before using more specific ones. This is especially true when trying to gain more information about foot lameness (after a positive PD nerve block, using intrasynovial distal interphalangeal (DIP) joint, navicular bursa (NB), or digital flexor tendon sheath (DFTS) blocks) or differentiating hindlimb proximal suspensory ligament desmitis from lower hock joint pain.

Results of the blocks are evaluated on the horse in hand or ridden. Having a rider is especially helpful for

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some mild lameness that are better felt than seen or when the lameness is best seen under saddle. It has been shown that some lameness can be unconsciously well hidden by riders and some would be exacerbated by such riders.[24] Experience of the veterinarian with the particular discipline plays a major role in the detections of such subtleties. This is especially true with dressage horses, when the veterinarian will also have to evaluate the trainer and rider as they could be causing a particular gait irregularity or not be able to guide the horse properly to perform a certain task. Having an experienced rider available to help evaluate such a horse can prove invaluable.

If the lameness cannot be localized, nuclear scintigraphy may be used. If the lameness is localized with nerve blocks, the whole array of imaging modalities could be used beginning with radiographs and ultrasound and when necessary cross-sectional imaging such as magnetic resonance imaging (MRI), computed tomography (CT), contrast-enhanced computer tomography (CECT), and positron emission tomography (PET) scan. MRI performed standing has gained in popularity and is often repeated to follow and monitor pathology even in competing horses.

COMMON LAMENESS PROBLEMS

All three disciplines confounded, the most common sources of lameness are:

1. Foot pain, including heel pain, solar pain, DDFT within the foot, DIP joint synovitis/arthritis, collateral desmitis of the DIP joint and navicular bone, and associated ligament injuries
2. Lower hock pain
3. Suspensory desmitis (fore- and hindlimbs, origin, and branches)
4. Axial skeleton: cervical, thoracolumbosacral, and sacroiliac pain
5. Superficial digital flexor tendon (SDFT) and inferior check ligament injuries
6. Fetlock osteoarthritis (OA)
7. Stifle injuries

There are marked variations regarding lameness problems not only between disciplines but also within disciplines between high- and low-level horses.[28] Dressage horses are significantly more prone to proximal suspensory desmitis (PSD) in the hindlimbs at both low and high level. Those competing at lower levels are more prone to navicular bone and associated ligamentous (impar sesamoidean and suspensory ligament of the navicular bone) injuries. Elite dressage horses are more prone to distal hock OA than the low-level horses. High-level show jumpers are more prone to DDFT injuries within the DFTS and the foot. The low-level horses are more prone to navicular bone and associated ligament injuries. Both have a relatively high incidence of distal hock OA. High-level event horses and high-level jumpers are significantly more prone than others to SDFT injuries. This is likely related to the fact that strain of the SDFT increases significantly with speed, fatigue, and height of the fences being jumped.[5,26]

Foot Pain

Foot pain is the single most common cause of lameness in performance horses. Assessment of foot pain starts with the observation of its conformation. Low-heel, long-toe conformations, underrun heels, and lateromedial imbalances predispose horses to soreness. Evaluation of the symmetry of the collateral ligaments of DIP joint and the presence of DIP joint effusion is better done by palpation. Hoof testers are applied carefully over the heels and sole and across the frog and the heels.

Most of the lameness of the foot will respond to PD nerve blocks, although such a block is not completely specific to the foot as it has been shown to diffuse and block out lesions in the pastern area even when performed the most accurately.[29,37] A positive PD nerve block should be interpreted as the lameness had its origin somewhere in the foot or pastern area. A positive DIP joint block will localize it to the coffin joint and the rest of the foot including the navicular apparatus and the sole, excluding the heels.[10,36,37]

A positive navicular bursa block will localize the origin of the lameness to the navicular apparatus or the sole.[38] A positive DFTS block if interpreted within 10 minutes should limit the origin of the lameness to its content with DDFT lesions[17] and sometimes distal sesamoidean ligament lesions found most commonly.

Heel Pain and Solar Pain

Heel pain and solar pain are more common in the event horses and jumpers than they are in dressage horses. Part of this is probably due to the type of surfaces they work on. Proper farriery guided with radiographs will often be sufficient. For solar pain, the use of full pads is often helpful. Pour-on fillers are very popular as well, but care must be taken not to over fill the shoe or under the plates, as it would cause undue pressure on the sole. Full plates are commonly used on hunter/jumper and dressage horses but pretty unpopular among the eventers as they can promote shoe loosening.

Heel pain and corns can be managed fairly well using onion shoes (open shoe with wide flare at the heels). Occasionally, in more severe cases of underrun heels, loading the frog using dental impression material, taped in without a shoe for 4–6 weeks, may help to promote some heel growth in a more vertical direction. Alternatively using frog-loading pads or a rolled heel can be useful.

Sheared heels affect mostly jumpers and starts often insidiously. The medial heel is often displaced proximally. Floating the affected heel and improving breakover by setting the shoe back and adjusting the lateral medial balance will usually resolve the problem, although this process can take 6 months to a year. Addition of a hoof supplement rich in Biotin seems to speed up hoof growth and improve hoof wall quality but is most often already being used.[23,32]

Navicular Syndrome, DIP Joint, and DDFT Lesions

Navicular bone and associated ligament injuries, DDFT lesions within the foot, DIP joint synovitis/arthritis,

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and DIP collateral desmitis can be difficult to differentiate using diagnostic analgesia only. Usually navicular bone and associated ligament injuries (impar ligament and suspensory ligament of the navicular bone) will usually block out not only to a PD nerve block but also to a DIP joint block and a navicular bursa block. Horses with DDFT lesions will block only partially to a PD nerve block and to a DFTS block if located in the distal tendon sheath and will block well to a PD nerve block and to the navicular bursa block or a DIP joint block if located within the navicular bursa and to a PD block and DIP joint block if the lesion is located at the level of the insertion.

Horses with DIP joint arthritis/synovitis will usually display effusion of the DIP joint and block well to a PD nerve block and to a DIP joint block. DIP collateral desmitis rarely displays palpable asymmetry, tends to block only partially to the PD block and a DIP joint block, and often requires a pastern ring block or a basisesamoid nerve block to resolve totally.

Navicular bone injuries such as subchondral bone cysts, fragmentation of the distal border, increased size of vascular channels, and enthesiopathy of the attachment of the suspensory ligament of the navicular bone are common and can be diagnosed easily using radiography. Early and/or less obvious lesions without a radiographic representation could be detected using nuclear scintigraphy or cross-sectional imaging such as CT or MRI. Bone bruising or edema is best seen on MRI STIR sequences.

Abnormalities of the impar ligament and distal aspect of the DDFT could be identified using transcuneal ultrasonography, although image quality is not always ideal, and there is a chance to miss subtle lesions as well as other lesions within the foot.

Effusion within the navicular bursa and some lesions of the DDFT proximal to the navicular bone can be seen with ultrasonography between the heel bulbs. This is enhanced when a microconvex probe is used because of the smaller footprint, which allows for a better contact. Injuries of the suspensory ligament of the navicular bone are harder to evaluate using ultrasonography.

Only 50% of the injuries to the collateral ligament of the coffin joints can be seen using ultrasonography. A small percentage has typical radiographic changes with osteolysis at the site of insertion onto the third phalanx.

Initial management usually entails rest and corrective shoeing, trying to adjust the hoof pastern angles and lateral medial unbalance. Bar shoes or onion shoes are often used as they support the heels and help decrease the sinking of the heels during the caudal phase of the stance on soft ground, theoretically diminishing pressure on the navicular apparatus and coffin joint. For suspect collateral desmitis, asymmetric shoes with a wider bar on the side of the injury are often used. Pads or fillers are often used as well. Onion shoes with a plate open over the frog are becoming increasingly popular.

DIP joint injection and navicular bursa injections with corticosteroids and hyaluronic acid or glycosaminoglycans are often part of the initial treatment. Alternatively, the use of biologics such as interleukin-1 receptor antagonist protein (IRAP),[13] platelet-rich plasma, or protein solutions (Pro-Stride™)[3] is becoming increasingly popular, as it seems to deliver clinical results on cases on which corticosteroids have failed. The other advantage is that you can use these products around shows without the worry about possible testing.

In recent years, polyacrylamide gels (Arthramid™ and Noltrex™) have become available as a new intra articular injection.[42] The gel acts initially as a joint lubricant and then gets integrated in the synovium improving its elasticity. Clinical results on OA cases have been impressive even in cases that were refractory to other modalities.

When this initial management fails or fails to last long enough, cross-sectional imaging such as MRI or CECT is warranted. MRI and CECT are both very valuable imaging modalities for injuries of the foot. In the author's experience both MRI on horses anesthetized and CECT are equally sensitive for most soft tissue lesions and structural bony lesions. MRI on horses standing has been (in the authors' experience) less sensitive and more susceptible to motion artifact. MRI presents the advantage over CECT to detect physiologic bony lesions (bone bruising or bone edema) on STIR. CECT offers the major advantages over MRI to be much faster and more importantly to allow CT guidance of needles to perform intralesional therapeutic injections.

Once a definitive diagnosis is made, the most appropriate treatment is indicated. This can consist in treatment of bone injuries (such as navicular bone edema or bruising or pedal osteitis) using rest, anti-inflammatories, and potentially bisphosphonates (tiludronate, clodronate, zoledronate). Collateral ligaments and DDFT lesions can be further treated using extracorporeal shockwave therapy (ESWT) high-intensity lasers or intralesional injections. Intralesional injections are performed under ultrasound guidance if the lesions can be seen or when available under CT guidance. The reader is referred to Chapter 8 for further information on these therapeutic options.

Lower Hock OA

Lower hock joint OA is also extremely common in sport horses. Most common complaints include lameness but more often the lack of engagement, loss of stride length, and poor impulsion. Complaints about jumpers and eventers will often include loss of scope jumping and the inability to stay centered while jumping. Dressage riders will complain about the inability to perform advanced figures necessitating significant collection such as piaffe or passage. Lameness is usually bilateral. Positive spavin flexion test is usually part of the findings. Radiographs can show various degrees of bone spurring, sclerosis, and joint space thinning with little correlation to the degree of lameness. In most cases intra-articular blocks of the tarsometatarsal (TMT) and distal intertarsal (DIT) joints improve significantly the lameness. In some cases, a tibial and peroneal nerve block is necessary to see an improvement. Most often, these joints are medicated with corticosteroids and hyaluronic acid or glycosaminoglycan as a therapeutic trial without blocking. Using a shoe with a small lateral plantar extension is thought to normalize the landing pattern of the foot and is often used in conjunction with joint injections.

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The choice of corticosteroid is often imposed by the show schedule and regulations: e.g. methylprednisolone has to be used at least 60 days prior to an FEI-regulated show, 15 days prior to an USEF-regulated show. Such treatment is often repeated at regular intervals to avoid losing performance and weeks of showing and help prevent the development of secondary issues.

Horses are often treated also with systemic injections of "maintenance" therapy such as glycosaminoglycans or hyaluronic acid.

In some cases refractory to steroids, biologics such as IRAP have been used with some unexpected success. In advanced cases, fusion of the lower hock joints could be performed either chemically (using ethyl alcohol[39] or monoiodoacetate[4,8,33]) or surgically. Horses will usually be able to perform at lower classes in such case.

When hock injections seem to improve the lameness but only for short periods, some thoughts should be given to the possibility of other injuries in the close proximity of the TMT and DIT joints such as proximal suspensory ligament desmitis.

Suspensory Desmitis

PSD is the single most common soft tissue lesion of especially dressage horses and hunter/jumpers. In the hindlimbs, PSD is likely due to the increased load placed by collection work in dressage and by jumping in hunter/jumpers and eventers. In the forelimbs, it is often seen with overzealous movers and related to the marked metacarpophalangeal hyperextension upon landing from jumps. Suspensory branch lesions are most common on the forelimbs in eventers and high-level show jumpers.[14]

PSD appears most often insidiously in the hind limbs, and lameness or loss of performance seems to subside temporarily to small periods of rest. The condition is often bilateral, and this delays recognition of the problem further. In the forelimbs, there is often a more acute lameness initially. Lameness is often more obvious on the circle with the leg on the outside. Palpation often can reveal sensitivity at the site on forelimbs, but interpretation can be difficult. Flexion tests can be positive especially carpal flexion test and spavin test, which can mislead the diagnostician.

Diagnosis is made using diagnostic analgesia: either direct infiltration of the ligament origin or specific nerve blocks. If subtle or no obvious lameness is present, using bilateral nerve blocks and reevaluating the horse for a change in its freedom of movement or seeking the help of an experienced rider to better evaluate the horse's ability to perform particular tasks may be necessary (especially for dressage horses) to aid diagnosis. Analgesia of the lateral palmar nerve at the level of medial aspect of the accessory carpal bone in the forelimb[6] and perineural injection of the deep branch of the lateral plantar nerve in the hindlimb[21] are gaining popularity as they are easy to perform and seem to decrease the incidence of unintentional analgesia of the carpometacarpal and TMT joints. Especially in the hindlimbs, the differentiation of distal hock pain and suspensory desmitis can be difficult. Performing a tibial nerve block can help differentiate between the two as it would theoretically block the proximal suspensory and not the lower hock joints. It also offers the advantage of not interfering with potential ultrasound evaluation. Such blocks could be performed on horses in hand or ridden if the lameness is most obvious then or only felt.

Ultrasonography is the most used way to diagnose PSD, evaluating possible enlargement, loss of fiber pattern, and core lesions. Ultrasound evaluation can be challenging especially when evaluating hindlimb suspensory ligaments, as the lesions are often subtle, and there is significant variation in the muscle fibers of the suspensory ligament that could appear as lesions to even experienced ultrasonographers. The transducer should be applied from medial plantar, and the ligament should be evaluated on cross-sectional and longitudinal planes. Comparisons with the opposite limb should be performed if there are any doubts about potential muscle fibers.

Radiographs could show bony remodeling at the level of the proximal palmar or plantar cortex and in more severe cases avulsion fractures. Nuclear scintigraphy can also be useful detecting increased radiopharmaceutical uptake at the level of the insertion of the proximal suspensory ligament, indicating a bony component to the lameness.

In questionable cases, the use of cross-sectional imaging MRI and CECT is extremely useful, not only to confirm lesions but also to rule out other causes of lameness responding to local analgesia of the site.

Treatment of PSD includes rest and controlled exercise and therapeutic shoeing (shoe with wide toe and thin branches to load the DDFT more), which is often sufficient treating for forelimb PSD but rarely resolves hindlimb desmitis. The addition of ESWT, laser therapy, and intralesional injections (of biological therapies) is helpful when dealing with hindlimb desmitis and has somewhat improved the prognosis. Ligament splitting is performed not only when a fresh core lesion is present but also in chronic cases.[19]

Fasciotomy and neurectomy of the deep branch of the lateral plantar nerve and neurectomy alone[2] have shown extremely promising results in horses with chronic hindlimb PSD as these horses are believed to have a neuropathy accompanying the compartmental syndrome created by the enlargement of the ligament.[43] Both fasciotomy and neurectomy are believed to reduce the compartmental compression: directly with the fasciotomy and indirectly by the neurectomy as it causes atrophy of the muscular fibers within the ligament, resulting in a reduction of the cross-sectional area.[30] Neurectomy of the deep branch of the lateral palmar nerve has also become increasingly used in recurrent front suspensory cases.

Suspensory branch injuries often have an acute onset. Enlargement, heat, and pain to palpation are usually features of the early stages. Definitive diagnosis is made using ultrasonography and periligamentous fibrous tissue; core lesions or enthesiopathy at the insertion on the proximal sesamoid bones can be seen. Rest and controlled exercise programs usually result in improvement. Laser therapy seems to help reduce clinical signs. Large core lesions could be treated with splitting or intralesional injections. Enthesiopathies respond well to ESWT. In some cases, the branch lesions communicate with the

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fetlock joints creating some degree of synovitis, requiring intra-articular medication of the joint and in certain cases arthroscopic debridement of the branch lesions.[27]

Axial Skeleton Pain

Cervical Pain

Cervical facet OA is an increasingly recognized condition affecting horses in several disciplines but more common in event horses and jumpers. A wide range of clinical signs can be linked to this condition including decline in jumping quality, resistance to sharp turns, and advanced dressage figures involving exaggerated lateroflexion such as canter pirouette, resistance to work in a frame, resistance to the bit, abnormal head carriage, forelimb lameness, neck stiffness, and in the most advanced cases neurological proprioceptive deficits.

Evaluation of range of motion (lateroflexion, ventroflexion, and extension) should be performed with minute attention to any range limitations, hesitation, or slowness. Some horses will compensate and avoid maximal flexion by turning their heads at the level of the atlantoaxial joint. Radiographs of the cervical column can be difficult to interpret as many horses will have evidence of some degree of OA of the last two intercervical facets (C5–C6 and C6–C7).[9] Nuclear scintigraphy will confirm the suspicion or reveal the sites when evaluating a horse for either poor performance or forelimb lameness that does not block out to the most commonly performed blocks.

Ultrasound is useful to evaluate the articular facets and identify osteophytes and effusion but is most often used to guide needles for therapeutic joint injections.

Horse with cervical pain associated with cervical facets OA can respond to physical therapy and osteopathic and chiropractic work. The vast majority will respond to intra-articular injections of corticosteroids using ultrasound guidance.

Back Pain

Back pain is frequent in performance horses and can manifest clinically through various performance limitations. The riders and trainers most often recognize it when it is accompanied by sensitivity to palpation, grooming, saddling, reactivity to tightening the girth and to the rider's weight, and bucking. Horses with chronic back pain will often loose some of their epaxial muscle mass.

Evaluation of reported behavior, back palpation and manipulation (inducing lateroflexion, dorsiflexion and extension at various levels), and observation of the horse lunging at the canter on soft ground (to evaluate the amplitude of the dorsiflexion) as well as the observation of the horse ridden will provide significant information.

Treatment of back pain will involve treatment of the initiating cause if identified, treatment of the primary lesion, and treatment of the associated muscle spasm and pain. Chiropractic work, physical therapy, and acupuncture, along with training modification, are complementary to the medical management.

The most common causes for back pain include the following.

Hindlimb Lameness Causing a Compensation Muscle Spasm in the Thoracolumbar Area

Treating the lameness usually will result in the resolution of the back pain within a few weeks. Treatment of the muscle spasm and pain will speed up the return to optimal performance and might be needed depending on the timing of shows.

Suboptimal Saddle Fit Creating Pressure Points or Restricting the Horse's Movement

Horses need to be comfortable in their tack to be able to provide optimal performance. The saddle should ideally be fitted to the individual horse. Adjustments should be made when the horse is in a state of fitness, and the fitting should be evaluated at rest but most importantly during work when the muscles are tighter and more voluminous to ensure that the saddle is not constrictive. Saddle should have a good area of contact with the horse and should not create pressure points. A veterinarian or saddle fitter experienced in the particular discipline should perform the evaluation and recommend/perform the adjustments as the movements that the saddles need to allow will vary depending on the discipline. Some saddle companies provide pads with pressure sensors to help evaluate the fitting of saddles. Alternatively, thermography could be used.

Rider

Evaluating the rider as a potential cause of back pain is always a delicate task and requires the veterinarian to be very knowledgeable about the particular discipline. Horses will have back issues that will be treated but would recur regularly. This situation presents itself most often with lower level riders that either sit crookedly or are heavy (relatively to the horse) and unbalanced.

Primary Muscle Strain and or Spasm

Primary muscle strain is usually acute, most common in jumpers and eventers, and presents with significant pain, swelling, and spasm. It usually is appreciated shortly after a significant workout. Treatment will involve anti-inflammatory therapy (icing and NSAIDS or a systemic dose of corticosteroids depending on the severity), rest, and muscle relaxants.

The more insidious and also more frequent muscle spasm requires finer back palpation to identify. Treatments are varied and include NSAIDS, muscle relaxants (methocarbamol), and epaxial muscle injections of trigger points or mesotherapy.

Epaxial muscle injection can be performed using 80 mL of sarapin, 10 mL of DMSO, and a dose of short-acting corticosteroid, injected using a 1.5-inch 21-g needle, following three parallel lines on either side of the dorsal spinous processes spaced inch apart, from mid-thoracic level, caudally to the sacroiliac area.

Kissing Spine

Kissing spines or dorsal spinous process impingement is one of the most common causes of primary back pain.

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The pain is chronic, and diagnosis is made using radiography (evidence of overriding spinous processes, with areas of bony sclerosis and bony lysis) or nuclear scintigraphy. The supraspinous ligament can also be affected and should be evaluated ultrasonographically.

Interpretation of radiographs, nuclear scintigraphy, and ultrasound of the supraspinous ligament should be performed with caution as many horses with no clinical signs of back pain will have abnormalities.[12,18] Using local anesthetics around suspected areas could be useful but should also be performed cautiously as it will also increase the amplitude of movement of normal horses.[20]

Treatment of impinging spinous processes involves injections of corticosteroids and sarapin in and around the affected interspinous spaces, muscle relaxants, NSAIDS, and bisphosphonates. Shockwave therapy is efficacious in some cases. Ancillary therapies (acupuncture, chiropractic work, etc.) and adaptation of the training aimed at encouraging dorsiflexion will complement the treatment. Refractory cases can be treated surgically (interspinous ligament desmotomy or partial resection of the spinous processes.[22]

Intervertebral Facet OA

Intervertebral thoracolumbar facet OA is to be suspected when horses have recurring back spasm and pain. Diagnosis can be made using nuclear scintigraphy, radiograph, or ultrasound. Facets are best evaluated on lateral oblique (horizontal 20° ventral–dorsal oblique) views taken from left to right and from right to left.[15] The use of local anesthesia and reevaluation helps confirm the diagnosis.[16] Treatment consists of intra-articular or periarticular injections of corticosteroids under ultrasound guidance and/or bisphosphonates added to the basic treatment of the associated muscle spasm. Physiotherapy to improve muscling is helpful for long-term management. Core strengthening exercises (such as baited stretches), riding or lunging aides to engage the hind end (Pessoa lunging rig, Equi-Band, and equivalents), and functional electrical stimulation (FES) are used most routinely.[31] FES has been really helpful to lower the spasticity of the muscle, as well as the quality of the muscling around the spine.

Sacroiliac Pain

Sacroiliac pain is relatively common, more so in jumpers and eventers than in dressage horses. The disease is usually bilateral but one limb is often more affected than the other. Severity varies widely but the most severely affected horses stand parked out and resent bearing weight on the most affected limb (complaint is often that the farrier is having a hard time shoeing the horse). Mild to moderate lameness is sometimes present, and some of the horses tend to "bunny hop" at the canter. Affected horses will often resist or show pain when the hindleg is picked up and raised enough to tilt the pelvis and are often sore to palpation/pressure over the tuber sacrale region.

Diagnosis can be reached using local analgesia around the joint (this should be done with extreme caution and never bilaterally, because of the risk of anesthetizing the sciatic nerve that will affect the horse's ability to walk and potentially stand). Nuclear scintigraphy using motion correction software can be diagnostic but is somewhat insensitive. Ultrasound evaluation of the dorsal sacroiliac ligament (transcutaneously) and of the sacroiliac joint itself (transrectally) can provide supportive information.

Therapeutic trials using periarticular injections of corticosteroids are used most often to confirm the diagnosis. Such injections are carried out blindly or with ultrasound assistance, using 8–10-inch spinal needles.[7,11] Treatment involves periods of rest for the most severe cases (4–6 months) and periarticular injections using corticosteroids and potentially sarapin for the least affected ones.

SDFT and Inferior Check Ligament Injuries

SDFT Injuries

SDFT injures are most common in event horses, somewhat common in jumpers especially at higher levels and rare in dressage horses. They almost exclusively affect the forelimbs. Presentations range from subclinical progressive tendonitis and peritendonitis (most common in eventers associated with cyclic repetitive loading and damage) to acute severe tendonitis with large core lesions (eventers and high level jumpers) that are most often related to one misstep on deeper footing and especially associated with the use of studs.

Diagnosis is easily made by palpation. Ultrasonography further characterizes the injury and allows follow-up.

Treatment is aimed in the acute stage at minimizing inflammation, using icing and NSAIDS to minimize edema and further fiber disruption. In extremely painful horses, placing the limb in a cast or a supportive boot seems to increase the comfort level. Supportive bandage and local anti-inflammatory ointments such as DMSO or diclofenac cream alternating with icing are helpful for the first few days.

After a few days, ultrasonographic examination is repeated. Large core lesions are treated using transcutaneous splitting. Treatment after that consists of rest and controlled exercise for 3–12 months depending on the extent of the injury, using ultrasonography to monitor progress and gradually increase the workload. The use of intralesional injection is becoming routine for such lesions using a source of stem cells (bone marrow, bone marrow concentrate, fad derived mesenchymal cells, cultured stem cells), a source of growth factors (platelet rich plasma or bone marrow supernatant), a scaffold (porcine bladder submucosa—Acell), or a combination thereof. Such injections are believed to improve the quality and speed of healing and decrease the rate of re-injury. The use of laser and therapeutic ultrasound is also popular.

Inferior Check Ligament Desmitis

This condition affects mostly jumpers and eventers in their mid-teens and younger dressage horses, almost exclusively on the forelimbs. Lameness presents acutely with swelling and heat at the site. Ultrasonography confirms the diagnosis, and treatments involve mostly rest and controlled exercise for 4–6 months.

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Intralesional injections of the inferior check ligament have not been rewarding in the authors' experience.

Some cases get complicated with adhesions to the SDFT that can be identified during a dynamic ultrasound examination (holding the leg and ultrasounding the area as fetlock flexion and extension are induced). Management of such complications involves surgical separation and 3–4 repeated injections of corticosteroids in between the SDFT and the check ligament 2 weeks apart.

Metacarpophalangeal and Metatarsophalangeal (MCP/MTP) Joint Injuries

Synovitis and OA of the fetlock joints are fairly common in older jumpers and eventers. Lameness is localized by a positive intra-articular analgesia or a low 4- or 6-point nerve block. Fragmentation of the proximodorsal aspect of the first phalanx is not uncommon and is better treated surgically, removing the fragment arthroscopically. Synovitis, with no evidence of radiographically/ultrasonographically visible pathology, usually responds well to intra-articular injections of corticosteroids and hyaluronan or glycosaminoglycan. Alternatively series of IRAP injections could be used. If the response to intra-articular therapy is incomplete or transient, advanced imaging (such as MRI) or exploratory arthroscopy is warranted. Severe bone bruising visible on MRI STIR sequences should be addressed and probably treated using bisphosphonates and rest to help prevent the development of subchondral bone cystic lesions.

Stifle Injuries

Osteochondrosis and Subchondral Bone Cysts

Osteochondrosis of the trochlea ridges of the femur and of the patella along with subchondral cystic lesions of the medial condyle of the femur is relatively frequent developmental disorders in Warmbloods. Therefore, radiographic surveys of yearlings during prepurchase examination are highly recommended.

Osteochondrosis lesions are treated surgically with debridement using arthroscopy, and prognosis is relatively good if the lesions are not too sizable.

Subchondral bone cysts are treated with either surgical debridement or injections of corticosteroids under the lining of the cyst,[44] but prognosis is not as good for a long career especially if the horse is older.[40] Preoperative treatment with a bisphosphonate reduces in the authors' experience the risk of postoperative enlargement of the cyst when debridement is elected.

Intermittent Upward Fixation of the Patella

This condition affects especially larger young horses and horses after a period of rest. The diagnosis is easy in the most severe cases and necessitates a finer observation in the milder ones who only "catch their patella." The best way to observe mild cases is during a transition from canter to trot or when they are being asked to back up.

Milder cases respond well to blistering of the medial and middle patellar ligament to induce scarring and thickening (injection of 0.5 mL of 2% iodine in almond oils at three levels in each ligament) and an injection (1 mL) in the gluteal muscles. More tenacious cases can be treated by ligament splitting,[41] and medial patellar desmotomy is reserved for the most severe ones as it has been associated with complications such as patellar fragmentation.[25]

Synovitis and OA

Synovitis and OA can affect either one or a combination of the three synovial compartments within the stifle. Ideally each one should be blocked during lameness examination separately. Radiographs and ultrasonography especially to evaluate the menisci are necessary. Osteophytes at the level of the medial tibial condyle are relatively common. If no soft tissue injuries are identified, intra-articular injections using a combination of corticosteroids and hyaluronan or glycosaminoglycan are warranted in each of the affected compartments. Alternatively IRAP could be used. If the injections do not provide soundness or if they do only for a short period, diagnostic exploratory arthroscopy is warranted.

Meniscal injuries are diagnosed after identification of one of the femorotibial joints as source of lameness, using ultrasonography or during exploratory arthroscopy.

Debridement of meniscal tears is warranted unless they are amenable to be sutured, and a period of 3–4 months of rest should ensue.[45]

Patellar Fractures

Patellar fractures are mostly seen in event horses and some jumpers. They are associated with moderate to severe lameness and effusion of the femoropatellar joint. Skyline radiographic view of the patella is necessary to fully evaluate the fracture. Most of the fractures are amenable to arthroscopic debridement, and the majority return to full work.

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THE ENDURANCE HORSE

Todd C. Holbrook

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THE SPORT

The sport of equine endurance has grown substantially since the American Endurance Ride Conference (AERC) was established as the national governing body for long-distance riding in 1972. Under current rules, the AERC recognizes endurance competitions that cover distances of at least 50 miles/day up to a maximum distance of 150 miles in 3 days. Pioneer rides may be up to 5 days in duration; in that case, 1 day must be 55 miles, and the remaining are 50 miles.[8]

The sport originated in the United States, where the Western States Trail Ride 100-mile competition (also known as the Tevis Cup) has been held in the Sierra mountain range since 1955. Endurance events covering distances of 80–160 km have been regulated by the Federation Equestre Internationale (FEI) since 1982. The sport has subsequently undergone tremendous growth internationally. In 2010, a new world record was set in a 100-mile (160-km) endurance competition in Dubai, United Arab Emirates. The winning time was 5 hours, 45 minutes, 44 seconds, at an average speed of 27.77 km/hour.[2] The trend of faster speeds associated with less technically demanding competitions in international events in comparison to national events is well recognized.

Typically, AERC rides are composed of both limited distance competitions (25–35 miles) and AERC-recognized endurance competitions, ranging from 50 to 100 miles. Over the past 10 ride seasons (2007–2016) in the United States, the AERC recognized an average of 269 yearly rides in which 19,575 horses competed each year.[8] While completion rates typically vary with ride conditions and level of competition, the average completion rate over the past 10 AERC seasons has been 86.25%.[8] Completion rates for international competitions are not typically this high. In a recent review of 30,741 horse starts at 100–160 km FEI endurance rides—in 47 countries between 2008 and 2011, completion rate was 50.7%.[5] Furthermore, a notable inverse relationship has been documented between mean competition speeds and completion rate.[10] In both national and international venues, the primary reason for elimination from endurance competition is lameness.

ATHLETES AND EXERCISE CONDITIONS

The suitability of pure Arabians and Arabian crosses is well recognized for long-distance aerobic exercise. However, most endurance rides are open to horses of other breeds, with the exception of breed-sanctioned competitions (i.e. Arabian Horse Association).

Frequently, suitable athletes compete from the age of eligibility for 8–12 years. It is not uncommon for some horses to compete well into their 20s. The current minimum age limit is 5 years for AERC and novice levels of FEI competition.[8,9] FEI competitions are categorized by course difficulty: horses must be 6 years old to enter 1* and 2* rides, 7 years old to enter 3* rides, and 8 years old to enter 4* rides.[6]

Although most competitive horses in the sport are of Arabian influence, their individual conformation can vary widely. Those with a long competitive career usually have few striking conformational flaws; however many horses with less-than-perfect conformation (e.g. toed in or out, high/low heel, contracted heels, post-legged, sickle hocked, etc.) can be competitive in the sport. Coupled with the magnitude of distance covered, the environmental conditions in terms of both topographical terrain variation as well as heat and humidity extremes create exercise conditions that are among the most challenging of any equine sport. Lameness and metabolic disturbances are the two most common reasons for elimination during competitions.

VETERINARY CONTROL

Endurance is one of the most stringently controlled equine sports in terms of veterinary oversight during competition. Horses are initially examined prior to the ride to determine suitability to enter competition. The examination includes a basic physical and lameness examination. At the pre-ride inspection, joint or tendon sheath effusion and abrasions or scars are entered on the ride card horse diagram, similar to markings on a Coggin's form (Figure 9.42).[8]

The ride is divided into segments or phases. An examination determines fitness to continue at the end of each phase at a veterinary check point or "vet gate." Short rides may only have one vet gate, whereas 160-km competitions may have as many as 7. Physical examination parameters evaluated and recorded on the ride card at each vet gate include pulse and respiratory rate (PR), mucous membrane character, capillary refill time, jugular vein refill, skin turgor, gastrointestinal sounds, back and withers sensitivity, rubs or sores from the bit or tack, muscle tone, anal tone, gait, impulsion, and overall impression. Criteria are recorded on a ride card using a scale of A to D, with A considered normal (Figure 9.42).[8] Rectal temperature is often only measured and recorded in horses exhibiting signs of heat stress.

Horses must meet a pulse criterion that is set prior to competition (usually 64 beats/minute within 30 minutes of entering the vet gate). A cardiac recovery index is determined in conjunction with the lameness examination by assessing the heart rate before and 1 minute after initiating an 80-m trot (40 m out and back). After the trot-out, if the horse's heart rate increases by four or more beats, metabolic criteria coupled with other physical exam findings are scrutinized in more depth to

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determine fitness to continue competition. Horses with synchronous diaphragmatic flutter coupled with evidence of significant metabolic distress are eliminated from competition. Those eliminated from competition by the control veterinary staff are typically referred to a treatment veterinarian for further evaluation and treatment on site.

Lameness is evaluated on the American Association of Equine Practitioners (AAEP) scale of 1–5 (see Table 2.3). Competitors may not enter or proceed in the ride on a horse with lameness of grade 3 or higher. Typically the footing for lameness examination areas is well planned and controlled at international ride venues, whereas at regional AERC rides, the footing and terrain may be quite variable at the vet gates (Figure 9.43). Occasionally, when level topography is unavailable and the footing is inconsistent, the lameness examination can be more challenging.

Maximum ride times are typically 12 and 24 hours for 50- and 100-mile competitions, respectively. In most rides the top 10 finishers are evaluated 1 hour after completion to determine best condition (BC). In con-

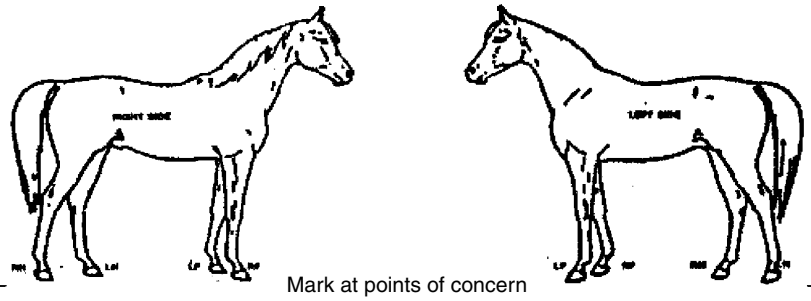
RIDE NAME_____ DATE_____ DISTANCE_____

Rider Name_____ Weight Division_____

Junior Rider____ Sponsor's Name (Juniors)_____

Horse Name_____ Age_____ Breed_____ Color_____

RIDER #



Mark at points of concern
(can use contrasting color at final)

Pre-Ride (First) Examination			Post-Ride (Final) Examination		
P_____ R_____ T_____			P_____ R_____ T_____ Heart Rate Recovery Index		
Parameter	A,B,C,D	Comments	Parameter	A,B,C,D	Comments
Mucous Membranes			Mucous Membranes		
Capillary Refill			Capillary Refill		
Jugular Refill			Jugular Refill		
Gut Sounds			Gut Sounds		
Skin Tenting			Skin Tenting		
Anal Tone			Anal Tone		
Muscle Tone			Muscle Tone		
Back Withers			Back Withers		
Tack Galls			Tack Galls		
Wounds			Wounds		
Gait			Gait		
Impulsion			Impulsion		
Attitude			Attitude		
Overall Impression			Overall Impression		
Signature of Examiner_____			Signature of Examiner_____		

Reason of elimination_____ Signature_____

A. file: RideCard 4/04 1.0

Figure 9.42. (A) Front of a typical AERC ride card. (B) Back of a typical AERC ride card.

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RIDER # _____ NAME _____

CHECK	#1	#2	#3	#4	#5	#6
ARRIVAL TIME						
PR TIME						
PULSE						
OUT TIME						
Mucous Membranes						
Capillary Refill						
Jugular Refill						
Gut Sounds						
Skin Tenting						
Anal Tone						
Muscle Tone						
Back Withers						
Tack Galls						
Wounds						
Gait						
Impulsion						
Attitude						
Overall Impression						
COMMENTS						
Heart Rate Recovery						
#1						
#2						
Examiner						

B.

Figure 9.42. (*Continued*)

trast to examinations during competition, the BC exam often includes limb palpation and mild flexion. Similar to the routine exams during competition, horses are evaluated for lameness at a trot. In addition, during the BC lameness examination, horses are also frequently evaluated in a circle in both directions. Performance-enhancing medications are not allowed during endurance competitions, and both the FEI and AERC governing bodies use random drug testing for prohibited substances.

THE LAMENESS EXAMINATION

It is not uncommon for an individual to perform several hundred lameness exams during any given ride, depending on entries, ride distance, and number of ride veterinarians. This provides a great training ground for students and new graduates as well as an enjoyable working environment for practitioners who are sports medicine enthusiasts.

As indicated above, lameness examinations during competition are typically limited to observation from a distance, with the exception of the foot evaluation. While limb palpation and joint flexion are used during the BC exam, this depth of examination during competition is typically reserved for horses that are eliminated to direct further diagnostics and treatment. If a soft tissue injury is suspected in horses with only a grade 1 or 2 lameness at a vet gate, the flexor surfaces are frequently palpated when considering elimination. Consistent examination of the foot also is important in these horses because removing a rock lodged in the frog sulcus or replacing a lost, displaced, or bent shoe may resolve or improve the lameness and allow the horse to continue (Figure 9.44). Thus, riders have the option to re-present for lameness evaluation after farriery.

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Figure 9.43. (A) Veterinary gate trot-out area for the FEI Young Riders World Endurance Championships in Verona, Italy, in 2017. (B) Veterinary gate trot-out area at an AERC ride near Poteau, OK.

Horses with grade 3 or greater lameness are eliminated from further competition and referred to the treatment veterinarian. Elimination of horses with grade 1–2 lameness is at the discretion of the control veterinarian and depends on the suspected underlying cause. Most horses with undetermined causes of lameness of this grade are allowed to continue, whereas elimination of horses with consistent sensitivity over the suspensory ligament or flexor tendons is prudent. Prior to elimination at FEI and AERC national championship rides, it is common for a panel of three veterinary judges to observe the trot-out and vote with the majority consensus taken.

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Figure 9.44. (A) 50-mile endurance horse with grade 3 lameness of the left front limb. Note the displacement of the medial branch of the shoe and the compression of the heel (arrow). (B) Medial heel bruise that was evident after shoe removal. The medial heel was floated and the shoe straightened and replaced. Subsequently, the horse was sound and continued competing.

COMMON CAUSES OF LAMENESS

Lameness is by far the most common indication for elimination from endurance competition. Based on AERC post-ride statistical reports, of 21,933 starts during the 2007–2008 ride season, 1,410 horses were eliminated for lameness, accounting for 44% of all eliminations. Of these, the majority were unlocalized front limb (45.9%) and hindlimb (23.4%) lameness conditions. Keep in mind that diagnoses at the vet gate are based on brief examination only, usually without even the benefit of hoof testers and definitely without the use of diagnostic nerve blocks. Considering the defined causes of lameness, hindlimb muscle comprised 9.4% of all lameness problems, followed by front suspensory (4.9%), front foot (4.4%), front tendon (3.3%), front muscle (2.5%), front joint (2.1%), hind tendon (1.1%), hindfoot (0.78%), other front ligament (0.71%), other hind ligament (0.64%), hind joint (0.57%), and hind suspensory (0.28%).[8]

Many causes of lameness in endurance athletes are transient, leading to elimination from competition on ride day but resolving after rest with or without the benefit of anti-inflammatory treatment. Often, the exact cause of lameness in these situations is not specifically determined. Recurrent problems or significant acute soft tissue or orthopedic injuries are more likely to result in a full diagnostic and therapeutic approach similar to that performed for any other sport horse.

Muscle Disorders

Certain occupational conditions are common in the sport of endurance, including exertional rhabdomyolysis and other muscle disorders primarily of the hindlimbs, back, and neck. While data regarding the prevalence of exertional rhabdomyolysis in endurance horses is limited, a recent prospective study of 101 endurance horses competing in four 50-mile distance races in the Northwestern United States identified a 4% prevalence of exertional rhabdomyolysis.[7]

Subclinical myopathy may also predispose to the development of compensatory lameness. In a study of 36 elite horses competing in a 160-km FEI 3-star competition, 22 horses that successfully completed had abnormally high mean creatine kinase (CK) activity (22,473 ± 41,192 IU/L) compared with pre-ride concentrations (702 ± 720 IU/L). Although there was no difference in CK activity between finishers and nonfinishers, two horses eliminated for lameness had the highest CK values (240,000–400,000 IU/L).[6]

Exertional rhabdomyolysis usually develops early in the ride (fewer than 15 miles), is often associated with anxiety, and may be influenced by cold weather and inadequate warm-up. Myopathy also develops late in the ride (more than 50 miles) and is associated with exhaustion or abrupt changes in footing (i.e. muddy hill work). Most commonly, the gluteals and lumbar epaxial muscles are affected. The gluteal and/or lower lumbar epaxial muscles may be affected asymmetrically, resulting in pronounced unilateral swelling or firmness in a specific area (e.g. middle gluteals). The most common gait abnormality is a shortened anterior stride phase at a walk. Further exercise is contraindicated with significant rhabdomyolysis, and aggressive treatment is warranted in most affected horses. The primary goal of treatment is fluid diuresis until gross pigmenturia is resolved. Analgesics may be necessary in some horses. Doses of flunixin meglumine and other toxic NSAIDs should be minimized in the face of dehydration. Portable biochemistry analyzers are commonly used by treatment veterinarians, at even small local endurance rides. Field evaluation of serum electrolytes, muscle enzymes, and renal parameters in these patients helps to guide therapy. Occasionally, endurance horses develop rhabdomyolysis upon resuming work after long-distance transport (road and air).

Although the efficacy of dantrolene in the treatment of acute rhabdomyolysis in horses has not been well studied, its role in preventing recurrent episodes is established. In treadmill studies, dantrolene sodium (4 mg/kg PO)

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reduced biochemical evidence of muscle damage in Thoroughbreds with recurrent exertional rhabdomyolysis.[3] In addition, dantrolene reduced recurrent episodes of rhabdomyolysis in racing Thoroughbreds.[1] Although dantrolene use during competition is prohibited, it is used in an attempt to prevent recurrent myopathy during training and/or long-distance transport of elite Arabian endurance horses predisposed to myopathy prior to resuming work.

Muscle spasms or cramps and muscle strains are also quite common in the sport. The gluteal muscles are commonly affected, but other muscle groups, including the gracilis, lumbar, triceps, biceps, forearm, and pectoral muscles, may be involved. Occasionally horses with muscle spasms are acutely painful; they may collapse and show signs similar to those of acute colic. These individuals usually benefit from prompt analgesic treatment. Most often an α-2 agonist (e.g. detomidine, xylazine) with or without a narcotic (butorphanol) is more effective and safer compared with high doses of NSAIDs, considering the likelihood of significant concurrent dehydration. Massage therapy, stretching or walking, and topical heat may help treat muscle cramps. On-site serum biochemical analysis in these patients is prudent because it may reveal electrolyte imbalances, necessitating correction.

Muscle pain affecting the back and skin sensitivity of the saddle and girth regions is common in endurance horses. It is likely that both are related to rider imbalance and poor saddle fit and compensatory back pain may develop secondary to hock lameness. In the neck region, sensitivity of the strap muscles in front of the shoulder (brachiocephalicus m.) is very common and likely develops as a consequence of forelimb lameness (usually in the feet or ankles) or fatigue.

More rarely, frank muscle tears may cause acute lameness. Occasionally, local hematomas may form. Rest and NSAIDs (post-rehydration) usually lead to full recovery.

Suspensory Desmitis

Forelimb suspensory desmitis (SD) is common in endurance horses, affecting both the proximal area and branches. Hindlimb SD is less common but likely has a similar pathogenesis, and the treatment options are identical. SD usually develops when local footing consists of deep sand, soft soil, or mud. Hence, in areas such the United Arab Emirates, SD is the most common cause of lameness in endurance horses. The diagnosis is suspected based on palpation of the limb and is confirmed by ultrasonography (Figure 9.45). The author's impression is that medial/lateral hoof imbalance predisposes endurance horses to suspensory branch desmitis. With branch lesions, usually only one branch is affected; therefore, notable swelling may be evident on palpation compared with the opposite branch or leg. In contrast, lesions of the proximal suspensory and body may be less obviously swollen, and diagnosis is usually based on sensitivity to palpation and ultrasound. Subtle, recurrent lameness associated with proximal SD may require diagnostic nerve blocks followed by ultrasonography for confirmation.

After an acute injury, the character of the ultrasonographic lesion severity may worsen over time, so serial examinations to follow lesion progression are recommended. Response to topical ice therapy can be dramatic and is warranted for the first 24 hours' post-injury. This should be followed by intermittent ice water or cold-water therapy and bandage support. After rehydration, NSAID treatment for the first 5–10-day post-injury is typical. The prognosis for return to function is good in horses with mild desmitis. Those with minimal to no ultrasonographic changes may only need 3–4 weeks of stall rest and hand walking before gradually returning to work under saddle, with an additional 4–6 weeks of walk/light trot intervals prior to returning to training work. With significant lesion size, long-term rest (minimum 6–8 months) is prudent. Horses with significant desmitis are best managed

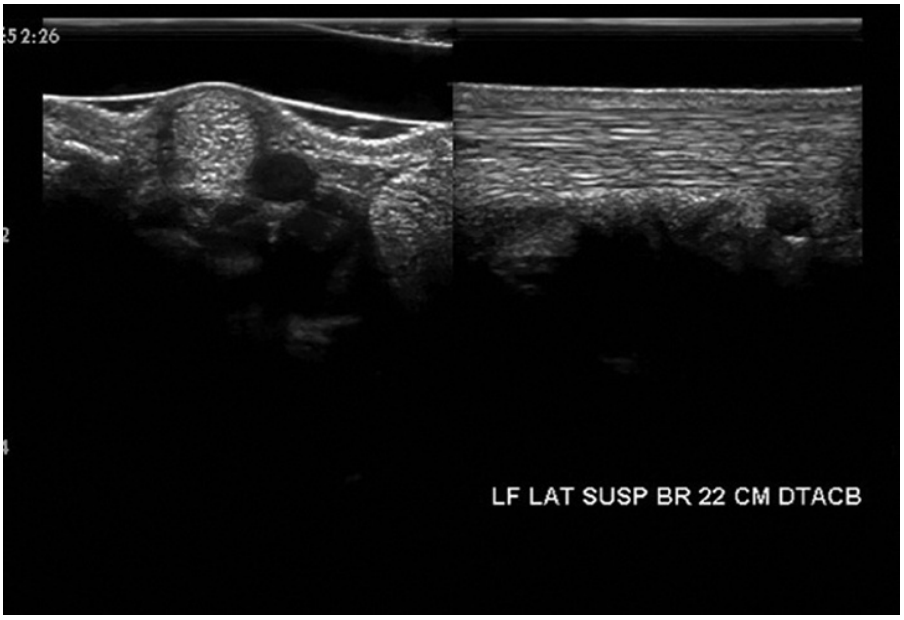


Figure 9.45. Ultrasonographic image of the lateral suspensory branch from an endurance horse that developed grade 3 lameness after 83 miles of competition. Enlargement and fiber disruption were minimal, and the horse returned to work after several weeks of rest. *Source*: Courtesy of Dr. Patty Doyle.

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with a well-controlled exercise program in which the intensity and duration of work is gradually increased over 6–12 months. This is often guided by improvement in lameness and ligament healing based on serial ultrasonographic evaluations.

Foot Lameness

Foot conditions are common causes of lameness in endurance horses. If time permitted more routine use of the hoof tester at the vet gate examination during endurance events, the AERC post-ride statistic results mentioned above would likely be quite different. Often evaluation of the digital pulse and comparison of its pre- and post-trot quality can be informative. Common conditions include foot bruising, corns, dislodged or sprung shoes, and heel bulb trauma from overreaching or other sources of injury.

Endurance horses are also subject to acute laminitis that may be related to repetitive concussive laminar trauma or associated with systemic exertional disease syndromes (e.g. heat stress, exhaustion, rhabdomyolysis, and renal failure). Aggressive medical treatment for acute laminitis is indicated in any endurance horse with prodromal signs thereof (shifting weight, forelimb lameness while turning, bounding digital pulses, etc.). Early treatment is important because consistent clinical prognostic indicators are unclear. It is quite common for certain horses to develop bruising at the coronary band and subsequent horizontal hoof cracks as the hoof grows out that serve as a timeline of when laminitis may have occurred at previous competitive rides (Figure 9.46). The majority of these horses complete the ride and do not develop lameness; thus, it is unclear whether this truly represents a laminitic event. Very rarely subsolar hematoma formation can cause acute lameness that can mimic acute traumatic laminitis in endurance horses.

Tendinitis

Superficial digital flexor (SDF) tendinitis is a common cause of lameness in endurance horses, especially in the forelimb. While tendinitis occurs less commonly in the hindlimb, it appears to be more prevalent in the sport of endurance compared with other disciplines. Deep digital flexor tendinitis occurs less commonly than SDF tendonitis but appears to have a predilection for the hindlimb in endurance horses. Clinical diagnosis is based on palpation and confirmed by ultrasonography. Thermography can be used to confirm regional hyperthermia associated with acute soft tissue injuries (Figure 9.47). Similar to other soft tissue injuries, early topical ice therapy and systemic NSAIDs after rehydration are warranted. Rest duration, rehabilitation, and prognosis depend on lesion severity and location.

Other Causes of Lameness

Acute joint sprain is not uncommon and is often influenced by the condition of the trail footing. Although the fetlock appears to be more commonly affected, slipping on trail can potentially result in soft tissue injury of any other joint. Interference injury, especially of the medial fetlock of the hindlimb, is common, and occasionally lameness can result (Figure 9.48).

Splints can cause acute lameness and are likely more common during training, especially in horses with less than ideal forelimb conformation. This condition typically responds well to conservative treatment and rest. Recurrent lameness is uncommon unless there is impingement on the suspensory apparatus that causes chronic desmitis.

Chronic OA affecting the metacarpophalangeal and hock joints is not uncommon. Prognosis in horses with significant degenerative disease is guarded with the exception of distal tarsitis, which may respond well to intra-articular corticosteroids, enabling continued work.

Catastrophic fractures have become more frequent in endurance horses coincident with the increasing speeds of competition, especially in the Middle East and at international venues. Fractures are most common in the forelimb, especially condylar fractures involving the metacarpo(tarso) phalangeal joint and, less commonly, the proximal phalanx.[4] Stress-related bone injury is presumed to play a role in fracture development in endurance horses. While no large-scale



Figure 9.46. Hemorrhage at the coronary band from a recent 100-mile competition on hard footing in an endurance horse.

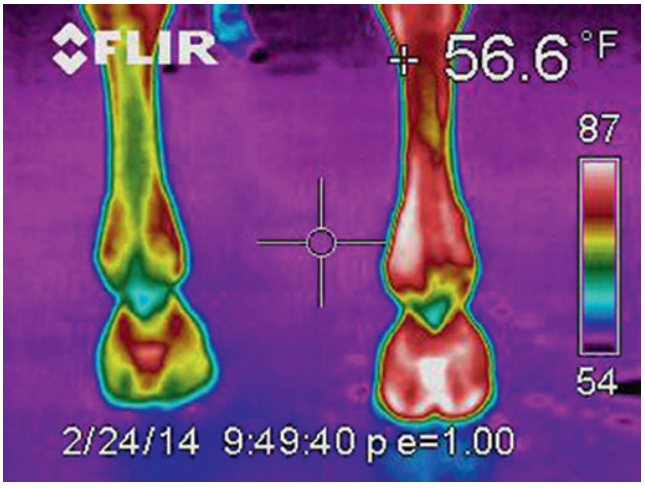


Figure 9.47. Thermographic image of an endurance horse with trauma of the right front medial fetlock region. *Source*: Courtesy of Dr. Ken Marcella.

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Figure 9.48. Interference lesion at the medial aspect of the right hind fetlock.

epidemiologic studies have been published, the prevalence of fractures per start in elite endurance horses appears to be similar to that seen in flat-racing Thoroughbreds.[4] Therefore, treatment veterinarians at endurance events should be prepared to stabilize distal limb fractures.

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THE WESTERN PLEASURE HORSE

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UNDERSTANDING THE SPORT

Western pleasure remains one of the most popular show events within the spectrum of western performance classes offered by such prominent show associations such as the American Quarter Horse Association (AQHA), National Snaffle Bit Association (NSBA), and the American Paint Horse Association (APHA). From its origin dating back to the 1960s, the western pleasure sport served to showcase working horses' athletic ability to execute performance gaits comfortably and synergistically with their rider. The collected, soft gaits and overall calm demeanor of these horses embody the name of their sport and the enjoyable riding experience they have come to represent. The sport has since evolved into the platform for other western classes including trail, horsemanship, and western riding. As with any specialized equine discipline, an effective veterinarian must be closely familiar with the expected maneuvers, training schedule, and demands placed upon the western pleasure athlete.

Similar to other specialized equine sports, western pleasure has undergone several style transitions throughout the years. Excessively low head carriages characterized pleasure horses in the 1980s, while in the 1990s they are remembered for their extremely slow, mechanical motions. The AQHA has since redefined how the western pleasure horse should ideally move throughout the three judged gaits (walk, jog, and lope).[1] The western pleasure assessment criteria can be most easily grouped into the below three categories:

1. Correctness of each gait
2. Quality of the horse's performance including self-carriage, expression, topline, and softness of movement
3. Ease of performance with minimal cuing from the rider

The quality of movement and consistency of gaits remains the most intrinsic and sought-after trait of an elite western pleasure athlete. It is also a source of intimidation for many equine veterinarians who are not familiar with how the various gaits should appear.

When done properly, a western pleasure horse uses its core stabilizing muscles to round its back, lift its topline, and advance its limbs in free-flowing, coordinated motions with suspension and cadence. The horse should exhibit forward motion with significant hind end propulsion, especially while loping. The speed of movement is determined by the frequency and length of strides taken. According to the AQHA, the stride length in all gaits should be "of reasonable length in keeping with conformation." In addition, the horse should be able to cover a proportionate amount of ground with little effort. In essence, slow, deliberate, long-flowing strides are more rewarded than short, choppy, and uncoordinated gaits. Western pleasure horses should carry their heads and necks in a relaxed, natural position, without excessive motion, while allowing for balance and cadence reinforcement. The neck should be at or slightly above the level of the withers, and the nose should not be excessively behind or in front of the vertical. All of the maneuvers should be able to be performed on a reasonably loose rein, with a bright, willing expression (Figure 9.49).

The sport of western pleasure has been criticized for the appearance of horses performing maneuvers incorrectly, potentially leading to strained, unnatural, and uncoordinated gaits in the show ring. Most commonly, exaggerations of the desired gait qualities include head bobbing, a four-beat lope, and most



Figure 9.49. Western pleasure horse being shown at the jog.

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recently an "over-canted" lope. When head bobbing as a consequence of lameness has been ruled out, the abnormal motion is most often the result of decreased forward momentum, lack of self-carriage, and imbalance. Also a consequence of reduced forward momentum is a short, choppy four-beat lope instead of the desired well-defined, three-beat gait. The extra beat has given life to the term "tropping," referring to lack of gait coordination between the forelimbs and the hindlimbs. An "over-canted" lope refers to curvature within the sagittal plane (through the hip) that manifests itself as the outside hindlimb being placed further to the inside of the arena than the inside forelimb (Figure 9.50).[1] This curvature toward the inside of the ring is a tactic employed to enhance the appearance of hindlimb stride length, also known as the "deep hock" appearance. The result of excessive curvature is a labored, mechanical lope instead of the desired fluid, smooth gait. The AQHA has taken proactive steps in establishing judging continuity to ensure that class placements do not favor these altered gaits. As with many equine disciplines, however, there will likely continue to be trends that come and go.



Figure 9.50. An overly canted lope characterized by curvature within the sagittal plane (through the hip) that manifests itself as the outside hindfoot (arrow) being placed further to the inside of the arena than the inside front foot.

WESTERN PLEASURE TERMINOLOGY

In addition to understanding the sport, the equine veterinarian must also be able to effectively communicate with owners and trainers about specific ridden impressions or other lameness-related observations. Being fluent in specific colloquial terms used among horsemen will enable the practitioner to better successfully evaluate, diagnose, and treat the subtleties that distinguish the elite western pleasure athlete.

Western pleasure horses may often present to the veterinarian for decreases in performance, not necessarily overt lameness issues. Common performance complaints include a horse not "holding ground well," or not "engaging behind," for example. "Holding ground" typically refers to the amount of time the forelimb on the outside of the circle remains in late stance phase during a particular gait (usually the lope), before rolling into swing phase (Figure 9.51). Extending the late stance phase of a forelimb increases the amount of time the foot is in contact with the ground, ultimately slowing leg speed. When a horse is not "holding ground" well, the resultant lope can be quicker, choppier, and less fluid than the desired gait. In addition, not "holding ground" can lead to increased carpal flexion while loping, which is in contrast to the preferred "flat knee" or "quiet up front" action (Figure 9.51). Another commonly used phrase in the western pleasure industry is a horse's ability to "engage behind" (Figure 9.52). This phrase refers to the cranial–caudal distance that a horse is driving up underneath of itself (cranial phase of stride), resulting in maximal hind end propulsion and effortless self-carriage. Commonly used expressions will likely vary among horsemen, geographical region, and level of competition.

TRAINING AND SHOWING

Some western pleasure athletes begin their careers as yearling lunge line competitors, while others do not enter the show ring officially until their 2-year-old season. Young horses are first trained to perform the basic transitions and gaits and then are gradually taught the preferred head set and speed while being exposed to different riding environments. Low head carriage is typically reinforced not only through hand cues and exposure to a variety of bits but also through the use of draw reins (Figure 9.53) or martingales. Speed is often controlled through varying amounts of leg and hand pressure and sometimes even with a cue referred to as a "spur stop" where applying leg/spur pressure signals the horse to slow down or stop. Due to the variability in the training methods most commonly used, the AQHA has instituted comprehensive rules that safeguard first and foremost the welfare of the horse.[1]

Asking a horse to perform these gaits with specific limb placement at slow speed adds difficulty to an already technically challenging sport for both horse and rider. A similar comparison can be made to a weight lifter. A weight lifter may easily lift 100 pounds quickly, but when asked to lift that same weight with controlled, slow motion, the task becomes much more difficult. The same applies to western pleasure horses. To master flawless gait transitions with seemingly invisible cues from

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Figure 9.51. A western pleasure horse loping in the left lead. The right forelimb is "holding ground," while the left forelimb is "quiet" with minimal carpal flexion. In addition, the hind end is well engaged, allowing for maximal hindlimb propulsion.



Figure 9.52. Western pleasure horse demonstrating the desired hind end engagement that allows for cadence, self-carriage, and forward propulsion while loping in the right lead.

the rider, significant training is required, and fatigue-related soft tissue injuries are not uncommon. In addition, a variety of orthopedic conditions as a result of rigorous training schedules and strenuous physical demands are seen in western pleasure horses.

COMMONLY ENCOUNTERED MUSCULOSKELETAL ISSUES IN THE WESTERN PLEASURE ATHLETE

Because the art of western pleasure derives itself from subtleties, these horses are typically best evaluated for lameness while ridden or lunged. Diagnostic analgesia and the administration of all therapeutics must be performed in accordance with association-sanctioned guidelines for which the practitioner must be astutely aware. The clinician treating actively competing horses faces a unique set of challenges in managing physiologically complex orthopedic conditions; therapeutic plans must not only reflect the most appropriate therapy for the horse but also remain in accordance with existing medication rules.

FOOT PAIN

Not unlike horses of any other discipline, western pleasure horses experience concussive forces throughout their training. Although this concussion is not endured at speed, the repetitive nature and hours required to gain

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Figure 9.53. Draw reins that allow the rider to contact the horse's bit through a single, movable, pulley-like system are commonly used as training tools to reinforce the appropriate head and neck carriage required of western pleasure horses.

the slow motor control to perform the calculated gaits are a recipe for foot pain. Some believe that the characteristically small feet of today's Quarter horse, which may not accommodate force dissipation during stance phase, exacerbate the effects of concussion.

Specific farrier approaches are often employed to enhance the western pleasure horse's natural, flowing forelimb movements. Western pleasure horse's front feet are typically shod with aluminum shoes, often with 2°–3° wedges and rim pads to achieve positive palmar angles and weightless solar support. Medial to lateral balance is as important in the western pleasure horse as it is in any other discipline, with often special attention paid to aligning the hoof pastern axis and decreasing the toe distance, also known as "easing the break-over." This combination of shoeing enhances the "quiet" carpal action of the western pleasure horse characterized by minimal carpal flexion. The hindfeet are also typically shod with aluminum shoes.

The forelimb lameness of a western pleasure horse with foot pain can present with varying degrees of severity and is usually exacerbated with the limb on the inside of the circle. The typical differential list for a western pleasure horse with pain localized to a palmar digital (PD) nerve block is no different than that for horses of other disciplines, and the major rule-outs include coffin joint, navicular apparatus, or sole pain as a result of concussive forces/shoeing abnormalities.

In a 7-year demographic review of 17,458 horses that presented to a practice focusing on competing western performance horses, diagnostic analgesia was performed 9,329 times. The forelimb PD and abaxial sesamoid were the two most frequently performed nerve blocks (Table 9.1). Over the 7-year period, 24,910 joints were medicated, with intraarticular coffin joint therapy comprising 4,480 of those cases (Table 9.2). The navicular bursa and pastern joint were medicated in 355 and 113 cases, respectively (Table 9.2).

It is the author's experience that MRI abnormalities that occur within the front feet in this population of horses are similar to those of other disciplines.

Table 9.1. Frequency of diagnostic nerve blocks performed over a seven-year period on 17,458 horses in a western performance equine sports medicine practice.

Diagnostic block	Frequency performed
Forelimb PD	1,867 (20.0%)
Forelimb abaxial	1,695 (18.2%)
Low four-point	724 (7.8%)
Tarsometatarsal joint	704 (7.5%)
Forelimb proximal suspensory	648 (6.9%)
Hindlimb proximal suspensory	575 (6.2%)
Distal intertarsal joint	382 (4.1%)
Femoropatellar joint	362 (3.9%)
Fetlock joint (forelimb and hindlimb)	239 (2.6%)
Femoropatellar, medial femorotibial, and lateral femorotibial joints	235 (2.5%)
Scapulohumeral joint	103 (1.1%)
Medial femorotibial joint	100 (1.1%)
Carpus	91 (0.9%)
Sacroiliac joint	80 (0.9%)
Tarsocrural joint	82 (0.9%)
Lateral femorotibial joint	57 (0.6%)
Bicipital bursa	41 (0.4%)
Distal interphalangeal joint	26 (0.3%)
Elbow joint	24 (0.3%)
Carpal canal	18 (0.2%)
Hindlimb digital tendon sheath	15 (0.2%)
Forelimb digital tendon sheath	13 (0.1%)
TMT and DIT joint	8 (<0.01%)
Total diagnostic blocks	**9,329**

Source: Josh Donnell, Unpublished data.

Observed pathologic changes include articular cartilage damage of the coffin joint, deep digital flexor tendinopathy, collateral ligament desmopathy, alterations of the navicular bone itself (sclerosis or bone marrow edema), and navicular bursitis. The array of abnormalities that have been realized with the use of high-field MRI continue to advance what is known about navicular disease in all equine athletes, a paradigm that will continue to evolve with continued research and clinical application.

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Table 9.2. Frequency of therapeutic injections performed over a 7-year period on 17,458 horses in a western performance equine sports medicine practice.

Synovial structure	Frequency of medication
Tarsometatarsal joint	7,183 (28.8%)
Femoropatellar joint	5,133 (20.6%)
Distal intertarsal joint	4,970 (20.0%)
Distal interphalangeal joint (forelimb and hindlimb)	4,480 (18.0%)
Medial femorotibial joint	960 (3.9%)
Fetlock joint (forelimb and hindlimb)	912 (3.7%)
Carpal canal	897 (3.6%)
Sacroiliac joint	807 (3.2%)
Digital tendon sheath (forelimb and hindlimb)	650 (2.6%)
Navicular bursa	355 (1.4%)
Lateral femorotibial joint	338 (1.3%)
Carpus	332 (1.3%)
Scapulohumeral joint	221 (0.9%)
Tarsocrural joint	167 (0.6%)
Femoropatellar, medial femorotibial, and lateral femorotibial joints	126 (0.5%)
Pastern joint (forelimb and hindlimb)	113 (0.4%)
Bicipital bursa	87 (0.3%)
Cervical facet (total)	70 (0.2%)
Cervical facet (C3–C4)	22 (<0.01%)
Cervical facet (C4–C5)	20 (<0.01%)
Cervical facet (C5–C6)	11 (<0.01%)
Cervical facet (C2–C3)	10 (<0.01%)
Total therapeutic injections	**24,910**

Source: Josh Donnell, Unpublished data.

FETLOCK OSTEOARTHRITIS

Similar to the racing Thoroughbred, the western pleasure horses experience pathologic manifestations due to the chronic, repetitive forms of loaded exercise that they undergo. Condylar fractures are far less common in the western pleasure horse compared with the racing Thoroughbred, but osteochondral fragmentation, synovitis, and eventual osteoarthritis with varying levels of articular damage, sclerosis (Figure 9.54), and bone marrow edema are commonly encountered in western pleasure horses.

In the previously described western performance horse demographic review, the fetlock joint was blocked for diagnostic purposes in 239 cases and was the sixth most frequently medicated synovial structure (N = 912; Table 9.1). Interestingly, the frequency of medicating to fetlock joint with interleukin-1 receptor antagonist protein (IRAP) was second only to the femoropatellar joint (Josh Donnell, Unpublished data).

PROXIMAL SUSPENSORY DESMOPATHY

Suspensory desmopathy of the fore- and hindlimbs is frequently encountered in western pleasure horses, albeit commonly with differing presenting histories. In the western performance practice demographic review, forelimb and hindlimb proximal suspensory analgesia was pursued 648 and 575 times, respectively (Table 9.1).

Horses subsequently diagnosed with hindlimb proximal suspensory desmopathy often present with a historic hindlimb lameness that improves for 2–3 weeks following hock injections. Following the transient improvement, however, the lameness returns and seems exacerbated with work in soft, deep ground. Trainers and riders also usually describe a lack of hindlimb propulsion, but this observation is not specific for only hindlimb suspensory desmopathy. The lameness is not consistently worsened when the hindlimb is on the inside vs. outside of the circle and is broadly characterized by a decreased cranial phase of stride and an unwillingness to push during late stance phase. Once the lameness has been localized to the proximal metatarsal region in accordance with ruling out the distal limb, standard radiographic evaluation of the tarsus is typically pursued, followed by both weight-bearing and non-weight-bearing ultrasonographic evaluation of the proximal suspensory ligament as previously described.[6,7]

Commonly observed pathologic changes include ligamentous enlargement (desmopathy), osseous remodeling (enthesopathy), fascial thickening (fasciitis), and loss of fat/muscle/fiber definition assessed with off-angle imaging. Ligamentous enlargement and osseous remodeling of the plantar aspect of the third metatarsal bone (Figure 9.55) singularly or in combination remain the most consistent, subjective findings in western pleasure horses. However, a portion of acutely lame horses with suspected suspensory injuries show no ultrasonographic evidence of abnormalities. The importance of contralateral limb comparisons cannot be overemphasized, permitting the clinician to more easily evaluate subtle changes that may otherwise go undetected. Of the 1,728 ultrasonographic evaluations performed in the practice demographic review, the forelimb and hindlimb suspensory ligaments were the most frequently evaluated anatomic structures with 871 and 365 exams performed, respectively (Josh Donnell, Unpublished data).

Horses subsequently diagnosed with forelimb proximal suspensory desmopathy commonly present with an acute lameness following a period of uncontrolled (excessive) lungeing or exercise. Alternatively, horses present with an acute-on-chronic, insidious forelimb lameness that worsens with work. A common complaint for these horses is not "holding ground" with the limb on the outside of the circle. Regardless of the history, the lameness is usually exacerbated with the limb on the

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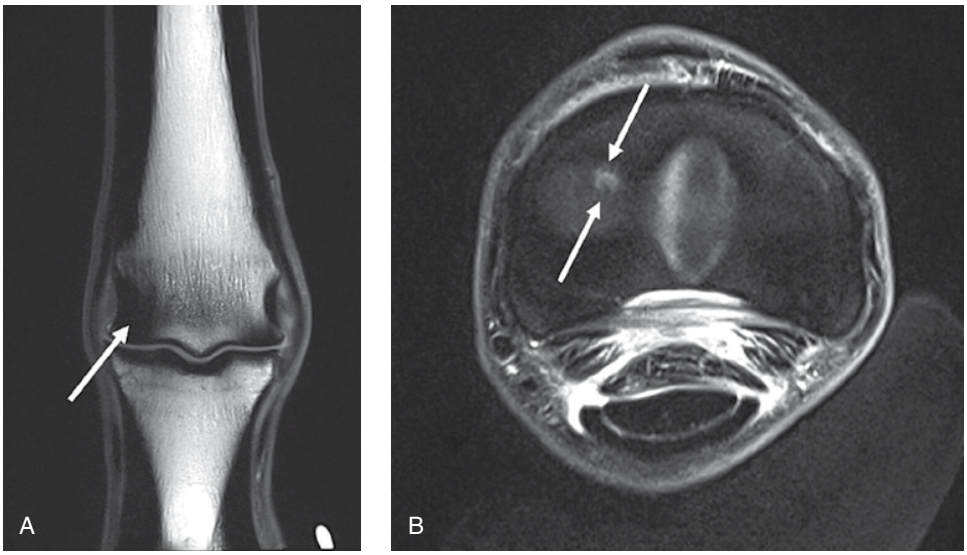


Figure 9.54. Magnetic resonance images from a 9-year-old western pleasure gelding with lameness localized to the fetlock joint that was refractory to intra-articular therapy. (A) Dorsal plane, T1-weighted image demonstrating markedly low signal consistent with severe sclerosis of the medial condyle (white arrow). Image acquisition parameters TR 600 ms, TE 12 ms. (B) Transverse plane, intermediate-weighted, fat-suppressed image demonstrating an ill-defined area of increased signal (white arrows) within the mid to dorsal aspect of the metacarpophalangeal joint. The linear striations in the oblique sesamoidean ligaments (most prominent in the medial distal oblique sesamoidean ligament) are normal architecture. Image acquisition parameters TR 300 ms, TE 40 ms (mildly T2 weighted).

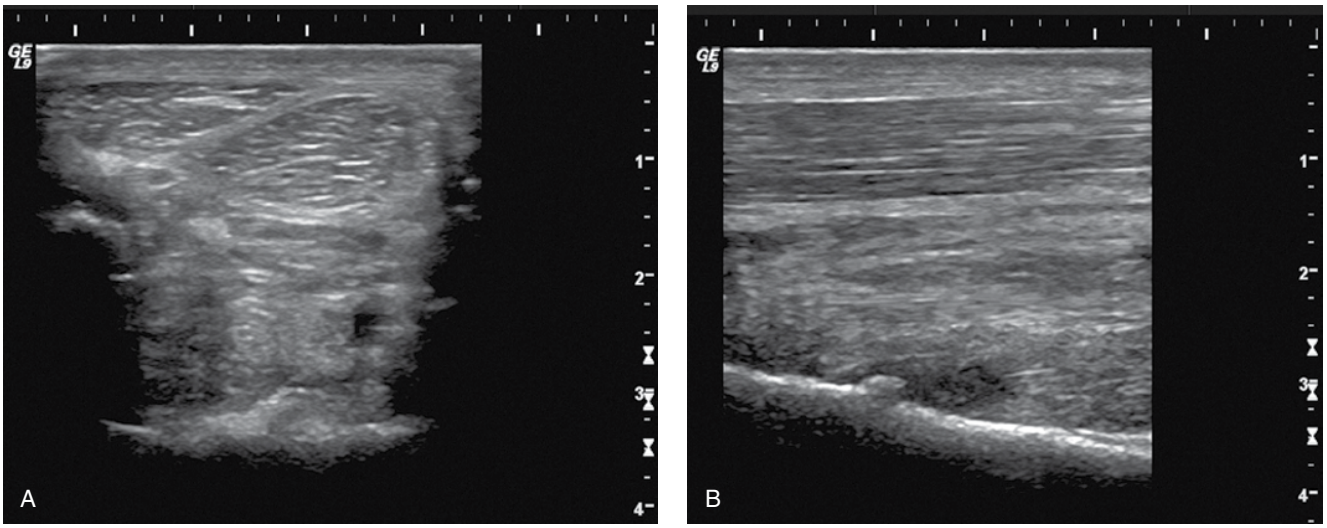


Figure 9.55. Ultrasonographic evaluation (non-weight bearing plantar (A-top) and weight bearing longitudinal (B-bottom)) of a 10-year-old all-around western performance horse with a hindlimb lameness localized to the proximal suspensory ligament. Moderate to severe chronic proximal suspensory desmopathy with associated enthesopathy and fasciitis was subsequently diagnosed and confirmed on MRI evaluation.

outside of the circle and periodically with the foot desensitized.[3] Standard diagnostic analgesia beginning with the distal limb is recommended, followed by radiographic carpal evaluation and ultrasonographic assessment similar to that described for the hindlimb (weight bearing and non-weight bearing).[6,7] Frequently observed ultrasonographic changes of the forelimb suspensory ligament include generalized ligamentous enlargement (desmopathy) with fiber hypertrophy that results in palmar displacement of the fat/muscle tissue. Osseous remodeling at the palmar aspect of the third metacarpal bone (enthesopathy) is also encountered to varying degrees. Subjectively, forelimb enthesopathy changes seem to reflect osseous resorption, resulting in a "scooped" appearance of the palmar metacarpus at the origin of the suspensory. Similar to the hindlimb, however, many western pleasure horses with pain localized to the proximal metacarpus show no ultrasonographic evidence of abnormalities.

If the severity of disease does not warrant cessation of work, forelimb and hindlimb proximal suspensory desmopathy is most successfully medically managed with extracorporeal shockwave therapy in combination with regional corticosteroid treatment, cryotherapy, and

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Figure 9.56. Standing stifle arthroscopy being performed with the use of an 18-auge disposable arthroscope. This technique has been used on numerous western performance horses to identify and characterize pathologic changes within the stifle.

rehabilitation. In the western performance practice demographic review, 1,212 cases received shockwave therapy to medically manage suspensory desmopathy (Josh Donnell, Unpublished data).

DISTAL TARSAL DISEASE

Pursuant to a discussion of hindlimb suspensory desmopathy, dialog regarding distal tarsal (hock) disease naturally follows thereafter as the two conditions are both commonly seen in the western pleasure athlete and often simultaneously.[2] In contrast to horses of other disciplines, a considerable frequency of juvenile (2–3 years of age) osteoarthritis is observed in the distal tarsal joints of western pleasure horses. The high prevalence of disease is attributed to the repetitive loading and torsional forces placed on the hocks of western pleasure horses during the collected lope. In addition, all pivots and overall directional changes in western pleasure are to be performed through hindlimb propulsion, of which the hocks and stifles are the driving force. With age and progressing levels of work, juvenile distal intertarsal (DIT) and tarsometatarsal (TMT) osteoarthritis may progress to partial or complete ankylosis.

Western pleasure horses with distal tarsal (DIT/TMT) disease typically present with a standard history of hindlimb lameness and lack of propulsion. A trainer may also specify that a horse is not "engaging" as they are capable of. It is the authors' subjective experience that primary TMT-related lameness is exacerbated with the affected limb on the inside of the circle, while primary DIT-related lameness is exacerbated with the affected limb on the outside of the circle, although the incidence of simultaneous disease is high. The TMT joint was the most frequently blocked synovial structure (N = 704) in the demographic review (Table 9.1). Subsequently, the TMT joint was the most frequently medicated synovial structure. The DIT joint was blocked second most frequently and was the third most medicated joint (Josh Donnell, Unpublished data).

In a study of 103 hindlimbs assessed with MRI, the majority of which were western performance horses, osteoarthritic changes of the DIT and TMT joints were the most common findings.[2] Other degenerative tarsal changes such as subchondral cystic lesions and intertarsal desmopathy that were underappreciated radiographically were identified with MRI.[2] With the increasing frequency of distal tarsal/proximal metatarsal MRI examinations, there will likely be further insight into the range of pathologic changes that occur in this complex region.

Stifle Disease

Similar to equine athletes of other disciplines, stifle disease is a consistent source of lameness for western pleasure horses. The standard clinical and dynamic lameness exam reveals hindlimb lameness of varying degrees with effusion typically present in both the medial femorotibial and femoropatellar joints. Unlike some of the other lameness conditions, stifle disease does not subjectively seem to be consistently worsened with the affected limb on the inside or outside of the circle, as it can be present during either scenario.

Diagnostic analgesia to investigate the source of the stifle lameness is standard as for other hindlimb gait abnormalities, with the authors routinely anesthetizing all three joints of the stifle simultaneously to effectively rule in or out the stifle as the source of lameness. Anesthesia of the individual stifle joints may then be pursued at a later blocking session if warranted. Over the 7-year study period, all three joints of the stifle were anesthetized 235 times, while the femoropatellar, medial femorotibial, and lateral femorotibial joints were individually anesthetized in 362, 100, and 57 cases, respectively (Table 9.1). Likewise, the femoropatellar joint was the most frequently medicated stifle joint being treated in 5,133 cases. Overall, the femoropatellar joint was the second most frequently treated synovial structure behind the TMT joint. The medial and lateral femorotibial joints were individually treated in 960 and 338 cases, respectively, while all three stifle joints were medicated simultaneously in 235 cases (Table 9.2).

Once the lameness has been localized to the stifle, standard diagnostic evaluation is performed both radiographically and ultrasonographically. The standard ultrasonographic assessment of the stifle should include

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not only a thorough evaluation of the articular environment but also an exhaustive consideration of the character of the medial meniscus and medial cranial meniscotibial ligament (MCMTL) during the non-weight-bearing portion of the exam. Commonly observed pathologic changes include meniscal degeneration, with MCMTL tearing (desmopathy) and associated osseous (insertional) remodeling (enthesopathy). These changes, in combination with synovitis and suspected articular cartilage damage, comprise the traumatic osteoarthritis complex frequently encountered in not only western pleasure horses but also western horses of many disciplines. Repetitive hindlimb loading and torsional forces are suspected, although the exact etiology and the temporal relationship of articular cartilage damage to meniscal instability (or vice versa) remain unclear.

With or without significant imaging abnormalities, a lameness that has been diagnostically localized to the stifle joint that remains refractory to therapeutic treatment usually warrants diagnostic and therapeutic arthroscopic surgery. The use of an 18-gauge disposable arthroscope to evaluate the stifle joint in the standing horse has been used successfully on horses of many western disciplines (Figure 9.56).[4] Used as a diagnostic tool, this procedure helps identify lesions and allows the clinician to characterize lesion severity in the standing, sedated horse. In turn, these findings can be used to decide whether to continue further medical and rehabilitative management or determine the need for standard, therapeutic arthroscopic surgery.

Axial Skeleton

Because precise head and neck carriage are central to the sport of western pleasure, the neck and associated supporting musculature is commonly investigated in cases of poor performance or perceived stiffness. Following a routine, dynamic lameness evaluation, special attention is paid to assessing the cervical spine for pain and functional restriction. Routine diagnostic evaluation of the cervical spine includes radiographic assessment using both lateral and oblique views, followed by ultrasonographic evaluation. The most frequently observed pathologic change of the cervical spine is facet osteoarthritis characterized by periarticular osteophyte formation, joint capsule thickening, and synovitis. In the demographic review, cervical facets within the neck were medicated 70 times with C3/4 being the most frequently treated articular facet (Josh Donnell, Unpublished data).

Although "back pain" is a common presenting complaint of owners and trainers of western pleasure horses, it is often difficult to fully determine whether the back is a primary or secondary performance issue. Once any contributing role of primary hindlimb lameness and/or saddle fit has been addressed, any remaining back pain is usually treated with combinations of a variety of therapies including systemic muscle relaxants, mesotherapy, acupuncture, regional infiltrations of sarapin and triamcinolone or shockwave therapy. The back/lumbar region was the fourth most frequent site that underwent shockwave therapy in the previously discussed demographic review (the suspensory ligament, hock, and palmar heel region underwent shockwave therapy most frequently). Shockwave therapy was also frequently used on the sacroiliac region, and the sacroiliac region was medicated in 807 cases (Table 9.2).

REHABILITATION AND MANAGEMENT OF THE WESTERN PLEASURE ATHLETE

With the increasing popularity and inherent athletic demands of the sport of western pleasure, there is vested motivation for preventing and managing injuries in the longitudinally competing western pleasure athlete. While owners and trainers still commonly pursue routine therapeutic joint injections, the value of physical therapy is also recognized both at home and during show competitions. Horses with previously diagnosed injuries are often aggressively managed with cryotherapy, systemic and topical non-steroidal anti-inflammatories, and support limb wraps (when indicated and as allowed per sanction guidelines). Similar to other disciplines, western pleasure athletes are also commonly maintained on systemic joint medications such as IV hyaluronate sodium* and/or IM polysulfated glycosaminoglycan.** Numerous commercialized therapeutic modalities are also readily accessible at the larger show competitions including cold-water spas, theraplates, electromagnetic blankets, and lasers of various classes. While the mechanism or role many of these modalities play in the treatment of musculoskeletal disease remains unknown, their increased availability, demand, and use by horsemen warrants mention.

When show horses are not competing, graduated, controlled exercise rehabilitative programs that incorporate the use of focused flexibility training, proprioceptive stimuli and core-strengthening exercises are being utilized with increasing frequency. In addition, with the increased availability of underwater treadmill units and the recognized role aquatic therapy can play in mitigating osteoarthritis, aquatic training is increasingly being used as an exercise alternative to maintain musculoskeletal fitness between competitions.[5] As the field of equine rehabilitation continues to evolve, advancements in tailored rehabilitation programs for specific injuries of various severities can be expected.

CONCLUSION

The sport of western pleasure with its deep-rooted traditions and novel stylistic approaches will likely continue to remain one of the most popular events of the western performance athlete. The specific, rigorous training and showing demands placed upon the western pleasure horse predispose them to sports-specific injuries just as other elite equine athletes. The most effective sports medicine veterinarians are able to recognize the physical stresses these horses endure to perform at the highest level possible. Therapeutic plans must not only reflect the most appropriate therapy for the western pleasure athlete but also remain in accordance with existing medication rules.

Notes

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GAITED HORSES

David A. Wilson and Kevin G. Keegan

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EVALUATING GAITED HORSES FOR LAMENESS

Most horses in the United States move in four specific gaits; walk, trot, canter, and gallop. The walk and trot are symmetric gaits, and the canter and gallop are considered asymmetric gaits. Symmetric gaits are the easiest gaits to analyze for lameness because the earliest sign of lameness is gait asymmetry.

Lameness evaluation is more difficult in horses that typically use a gait that is not a walk or trot. There are many other gaits used by horses, either naturally or artificially by training, and these other gaits can be difficult to evaluate without sufficient knowledge or experience. The trot is by far the most common symmetrical gait, and the majority of horses in the United States are evaluated for lameness at the trot. Therefore, most veterinarians are very comfortable evaluating horses at the trot. There are both practical, meaning it is easier, and principled, meaning it is more accurate, reasons for doing so. The trot has a large vertical suspension phase that occurs twice during each stride, and that vertical suspension phase exacerbates a weight-bearing lameness because of the resulting effects on vertical ground reaction force, which is the kinetic measure most highly correlated with pain on weight bearing. The trot is also a symmetric gait not only by side (left vs. right) but also by forelimb vs. hindlimb, which allows more predictable effects on determination of compensatory lameness. All of these reasons make the trot easier and better to evaluate for most veterinarians.

WHAT IS A GAITED HORSE?

Technically all horses are gaited horses. A walk, trot, and canter are "gaits." However, horses are considered to be "gaited" if they perform a gait that is not a typical walk, trot, canter, or gallop.

Gaited horses typically perform one of the many intermediate speed four-beat gaits, which are collectively referred to as ambling gaits. These horses are selectively bred for their natural gaited tendencies and are generally smooth to ride (Table 9.3).[8] In most "gaited" breeds, the ambling gait is a hereditary trait. The gene DMRT3 controls the spinal neurological circuits related to limb movement and motion.[1,2,22] Even one copy of a premature stop codon mutation in the DMRT3 gene (*DMRT3:Ser301STOP*), which diverged from a common ancestral sequence within the last 10,000 years, will produce gaitedness.[2] However, some members of these breeds may not always gait, and some naturally trotting breeds not listed in Table 9.3 may have ambling or "gaited" ability.

A gaited horse moves at a walk, similar to non-gaited horses. They place all four feet down independently. The change in foot placement occurs as the speed increases. Most horses begin to trot at faster speeds, with forelimb and hindlimb footfall synchrony. However, in gaited breeds at speeds a little faster than a standard walk, they disassociate forelimb and hindlimb footfall. In gaited breeds this faster disassociated gait may be faster than a trot in a non-gaited horse. These faster than walking gaits have many different names, the flat walk, the running walk, the rack, saddle gait … the list goes on and on.

CLASSIFICATION OF GAITS

There are many ways to classify the various gaits of the domestic horse. Gaits may be classified as:

Stepping (walking) vs. leaping
Symmetrical vs. asymmetrical
Two beat vs. four beat
Lateral vs. diagonal sequence
Regular vs. lateral vs. diagonal couplet gaits

Stepping (Walking) vs. Leaping Gaits

Stepping (walking) gaits lack an airborne (suspension) phase in the stride. Each limb is on the ground >50% of the total stride duration.[4,10,17] Stance phases of the forelimb and hindlimb pairs overlap, with no period of suspension and no high vertical displacement of the body and no aerial phases of the stride. These are typically four-beat gaits that have a lateral footfall sequence of LH, LF, RH, RF.[20]

By contrast, leaping gaits have at least one or usually two aerial phases during each stride, when none of the limbs are in contact with the ground. With leaping gaits, primarily the trot and the pace, the concussion or ground reaction forces on the limbs are usually greater than those horses using a stepping gait at equivalent speeds. Therefore, this increased concussion in the leaping gaits tends to exacerbate the differences in weight bearing between limbs with pain and those without.

Symmetrical vs. Asymmetrical Gaits

The rhythm or cadence of the right and left footfalls defines gait symmetry. In symmetric gaits, the footfalls of a pair of feet (forelimb pair or hindlimb pair) are evenly spaced in time.[10,17] The forelimbs and hindlimbs move symmetrically, but the left and right sides are 180° out of phase.[9,12] The movement of each pair of feet (fore or hind) is evenly spaced in time. The symmetric gaits are distinguished from each other by the coordination between the fore- and hind limbs, the speed of progression, the relative duration of stance and swing phases, and the presence/absence of aerial phases.[10]

The trot and pace are at opposite ends of the spectrum considering symmetrical gaits, but they are the closest to total symmetry such that the right and left and fore- and hindlimbs move in virtual symmetry. In other

Table 9.3. Breeds of gaited horses.

Breed	Description/origin
Aegidienberger	Small gaited horse from Germany that performs the tölt gait and was created by crossing the Peruvian Paso with the Icelandic horse
American Saddlebred	Some have one or two, four-beat ambling gaits: the slow gait and the rack. Initially developed in the United States in the early 1700s by crossing imported Thoroughbreds to the native Narragansett Pacer
Campeiro	Small, stout Brazilian horse that has an ambling gait similar to the Marchador and was developed in the 1500s from Spanish and Portuguese imported horses
Campolina	Larger Brazilian breed with an ambling gait called the true marcha or *marcha verdadeira*, developed in 1870 by farmer Cassiano Campolina by crossing a Barb mare with an Andalusian sire. Further refinements were made over the years by crossing to various other breeds
Florida Cracker Horse	Also known as the Seminole and has a single-footed ambling gait, sometimes referred to as a "coon rack." Was developed from ancestors of Spanish horses brought to Florida in the 1500s
Icelandic horse or pony	Has an ambling gait called the tölt and was developed in Iceland from horses imported by the Norway settlers almost 1,100 years ago
Kathiawari	Has a swift, lateral pace and is indigenous to the Kathiawar peninsula in western India
Kentucky Mountain Horse	Has a naturally occurring single-foot ambling gait, was developed in Eastern Kentucky, and is related to the Tennessee Walking Horse.
Mangalarga Marchador	Have two natural ambling gaits—the diagonal *batida* and the lateral *picada*. Are native to Brazil and descended from Portuguese Lusitano stallions and Barb mares
Marwari horse	Has a natural ambling gait that is close to a pace, called the *refaal*, *aphcal*, or *rehwal*, and are from the Mariwar (Jodhpur) region of India
Messara horse	Has a naturally pacing gait, also known as a Cretan horse, found on the island of Crete off the coast of Greece
Missouri Foxtrotter	Has an ambling, four-beat broken diagonal gait, developed in the Missouri Ozark Mountains in the early 19th century
North American Single-Footing Horse	Has an intermediate ambling gait sometimes called the rack or paso largo and originated in the southern United States
Pampa, spotted, or pinto horses	Developed from the Brazilian Mangalarga Marchador, Campolina, and Brazilian crossbred horses
Paso Fino	Has a natural four-beat ambling gait and originated from Barb, Spanish Jennet, and Andalusian horses imported to the Caribbean from Spain as early as the late 1400s
Peruvian Paso	Has a natural ambling gait called a "paso largo," and originated in Peru from Spanish Jennet, Barbs, and Andalusian horses.
Racking Horse	Has a distinctive ambling single-foot gait called the rack which is similar to the running walk of the Tennessee Walking Horse from which it descended in the mid-1800s
Rocky Mountain Horse	Has a natural ambling four-beat single-foot gait and was developed in Kentucky in the late 19th century
Spotted Saddle Horse	Has an ambling gait other than the trot and was developed by crossing Spanish–American type gaited pinto ponies with several gaited horse breeds, including the Tennessee Walking Horse, Morgan, and Standardbred horses
Standardbred Horse	Has a two-beat diagonal gait (trot) or two-beat lateral gait (pace); developed in the 18th century in the United States from English bloodlines
Tennessee Walking Horse	Has a four-beat gait with the same pattern as a regular, flat walk, but significantly faster and was developed in the late 18th century from Narragansett Pacers and Canadian Pacers crossed with gaited Spanish Mustangs from Texas
Walkaloosa	Has an ambling gait other than the trot and has been available for many years but just recently was recognized as a breed registry

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words they have a regular rhythm. All other symmetrical gaits move with right and left symmetry (both fore and hind), but the forelimbs do not move in symmetry with the hind limbs.

In asymmetric gaits, the footfalls of a pair of feet are unevenly spaced in time. The canter and gallop are examples of asymmetric gaits.[17] Each asymmetric gait also has two forms; for example, the right and left lead canter. This asynchrony makes it more difficult to evaluate lameness because one must remember the movement in one form and compare it to the other form that must be subsequently observed. It has been shown that weight-bearing pain is displayed at the trot as asymmetry well before the asymmetric gait is substantially altered.[18] Therefore, most descriptions of lameness evaluation are focused on symmetric gaits, most commonly the trot.

The horse performs many symmetric gaits including the walk, running walk, rack, paso largo, paso corto, classic fino, paso llano, sobreandando, marcha picada, marcha batida, fox trot, tölt, pace, stepping pace, trot, passage, and piaffe. The coordination between the hindlimbs and the forelimbs, the speed of progression, the relative durations of stance and swing phases, and the presence or absence of aerial phases distinguish the symmetrical gaits.[10]

Two-Beat Gaits: Diagonal (Contralateral) vs. Lateral (Ipsilateral) Synchrony

The two-beat gaits of horses use a diagonal (contralateral) or lateral (ipsilateral) pattern. In the trot, there is diagonal (contralateral) synchrony, whereby the forelimb and the contralateral hindlimb move in relative synchrony with a period of suspension separating the diagonal stance phases.[11,14]

In the pace, there is lateral (ipsilateral) synchrony whereby the forelimb and the ipsilateral hindlimb move in relative synchrony and each lateral stance phase is followed by a period of suspension.[10,14] Both the trot and pace have a suspension phase, which makes the gaits relatively uncomfortable and more difficult for the rider. Because of the suspension phase, the vertical ground reaction force of each limb is greater than during other gaits at equivalent speed. These gait characteristics increase the severity of the observed lameness, particularly in weight-bearing lameness.

Four-Beat Gaits

Most symmetric gaits, other than the trot and pace, are four-beat gaits. Each hoof contacts the ground separately in a four-beat gait. The footfall sequence may be lateral, diagonal, transverse, or rotary (Figure 9.57).[11] There are no known rotary gates in the horse.

Lateral vs. Diagonal Sequence Gaits

In a lateral sequence gait, the order of footfall is LH, LF, RH, RF. Lateral sequence gaits have the footfall of a given hindfoot followed by the footfall of the ipsilateral forefoot. The fox trot is an example of a lateral sequence gait.

In a diagonal sequence gait, the order of footfall is LH, RF, RH, LF. Diagonal sequence gaits have the footfall of a given hindfoot followed by the footfall of the contralateral forefoot.[17] The marcha batida is an example of a diagonal sequence gait.

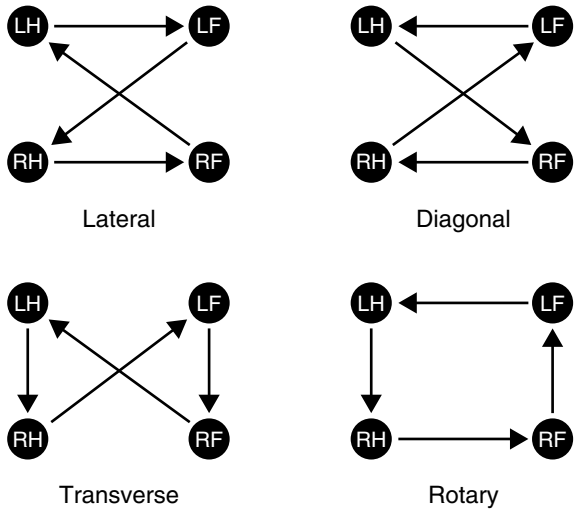


Figure 9.57. Footfall sequences in four-beat gaits. Arrows indicate the order of footfalls. LH, left hind; RH, right hind; LF, left front; RF, right front. *Source*: Reprinted with permission, The Dynamic Horse, Sport Horse Publications, 2004. Reproduced with permission of Cambridge University Press.

Regular Rhythm vs. Lateral Couplets vs. Diagonal Couplets

Lateral sequence gaits can be further divided into regular rhythm vs. lateral couplet vs. diagonal couplet gaits.

Regular (or square) rhythm gaits have a time interval between ipsilateral hindlimb and forelimb footfalls of exactly one-quarter of the stride duration.

Lateral couplets occur when the time between the ipsilateral footfalls of the hind- and forelimbs occurs in less than one-quarter of the stride duration. Lateral couplet gaits, such as the rack, have footfalls of a given fore- and hindfeet on the same side of the body related in time as a pair.

Diagonal couplet gaits, such as the fox trot, have footfalls of the fore- and hindfeet on the opposite sides of the body related in time as a pair. Diagonal couplets occur when the time interval between the ipsilateral footfalls of the hind- and forelimbs occurs in greater than one-quarter of the stride duration (Figure 9.58).[10,17]

Gaits Other Than the Walk, Trot, Pace, and Canter

There are many symmetric gaits that can be difficult to evaluate for a variety of reasons, primarily because most practitioners have not had as much experience evaluating these gaits. In these other symmetric gaits, the fore- and hindlimbs are generally moving symmetrically, but the timing of the fore- and hindlimbs varies depending on the specific gait. These gaits do not have a typical suspension phase and usually have at least one foot on the ground at all times.

These other symmetric gaits fall in a virtual continuum from the trot, which has a diagonal (or contralateral) symmetry, to the pace that has a lateral (or ipsilateral)

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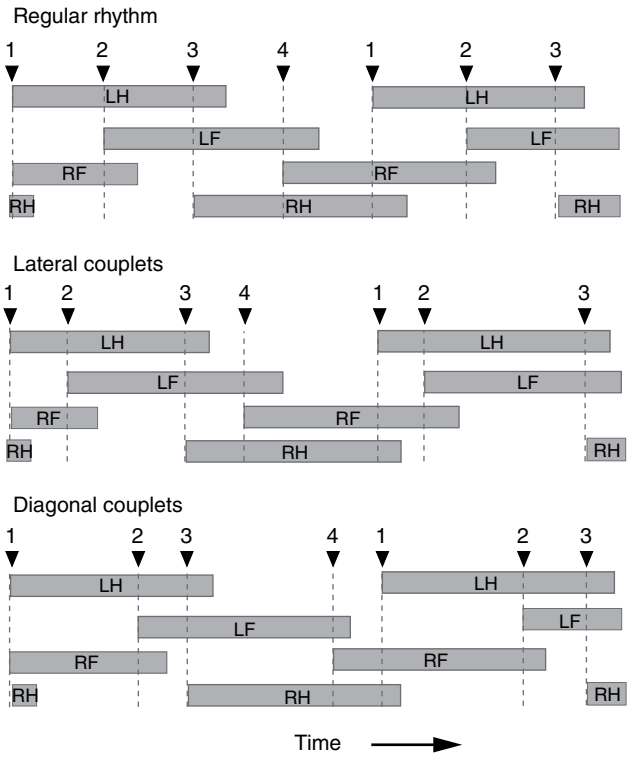


Figure 9.58. Gait diagrams for symmetrical, four-beat, lateral sequence gaits performed with a regular rhythm (above), lateral couplets (middle), and diagonal couplets (below). Shaded bars represent the stance phases of the limbs and open areas between bars represent swing phases. The numbered arrows and vertical dashed lines show the timing of the footfalls. LH, left hind limb; RH, right hind limb; LF, left front limb; RF, right front limb. Note that the contacts of the LH and RH occur at the same time in all three diagrams. *Source*: Reprinted with permission, The Dynamic Horse, Sport Horse Publications. Reproduced with permission of Cambridge University Press.

symmetry.[17,21] The gaits along this continuum, shown in the center of Figure 9.59A and B, are usually considered four-beat gaits or stepping gaits. The boundaries between these four-beat stepping gaits are arbitrary and subjective with gait distinction being based mostly on timing of foot placement, breed, and style of movement. They include the walk, the paso gaits, fox trot, the marcha gaits, the various rack gaits, and the running walk[17,20] (Figure 9.59). The gaited horse gait spectrum (https://www.youtube.com/watch?v=aoB6FFP7a84; Phoenix Rising Saddles, accessed 30 January 2020).

SPECIFIC GAITS IN "GAITED HORSES"

The **fox trot** is a four-beat gait, with an irregular rhythm with diagonal couplets. It is a stepping gait with no periods of suspension.[9] The rhythm of footfalls is characterized by diagonal couplets with forelimb impact being slightly before contralateral hindlimb impact, after an interval of ~15% of the stride duration (https://mfthba.com/education/). The sequence of limb placements is LH, LF, RH, RF. This early forelimb placement and impact causes an amplified vertical movement of the head and damped vertical movement of the rest of the body. It is reported to be an energetically efficient gait. There are typically eight limb support sequences in each stride, which alternates between periods of bipedal and tripedal support (https://mfthba.com/education/).[12] At faster speeds, as seen in the show ring, there may be a suspension phase when both forelimbs are off the ground simultaneously (https://mfthba.com/education/).

The same principles apply for the five-gaited Saddlebred and Tennessee Walking Horse at the rack, slow gait, and running walk (and the Icelandic horse at the tölt), when the hindlimb impacts the ground before the ipsilateral forelimb. The damped vertical movement of the body during these four-beat gaits in these breeds, although creating a more difficult gait to evaluate for lameness, is a desired quality that provides a smooth riding experience. Fortunately, most five-gaited Saddlebred horses readily trot and can be evaluated at this gait when evaluation of the rack or slow gait is difficult.

Running walk is a four-beat stepping gait with lateral couplets (LH, LF, RH, RF) and significant overstep of the forelimbs at speed. The hindfeet overreach the imprint of the front by 15–55 cm in a straight, smooth, gliding motion. It is the classic gait of the Tennessee Walking Horse[7] (https://www.youtube.com/watch?v=usZCp-nJEYI, accessed 30 January 2020).

The racking family of gaits falls into the symmetrical gaits, and almost all have the same sequence or order of footfall. The footfall sequence of the gaits in the rack family is the same as that of the walk (LH, LF, RH, RF). The variation in racking gaits is due to the timing of footfall, the "pick up," and the support sequence. This family of gaits includes the rack, saddle rack, stepped rack, and half rack. These are known by different names in the various breeds: Fino, Corto, Largo, Rocky Mountain Horse, Pleasure Gait, Trippel, Singlefoot, Stepping Rack, Pure Tölt, and Fast Largo.

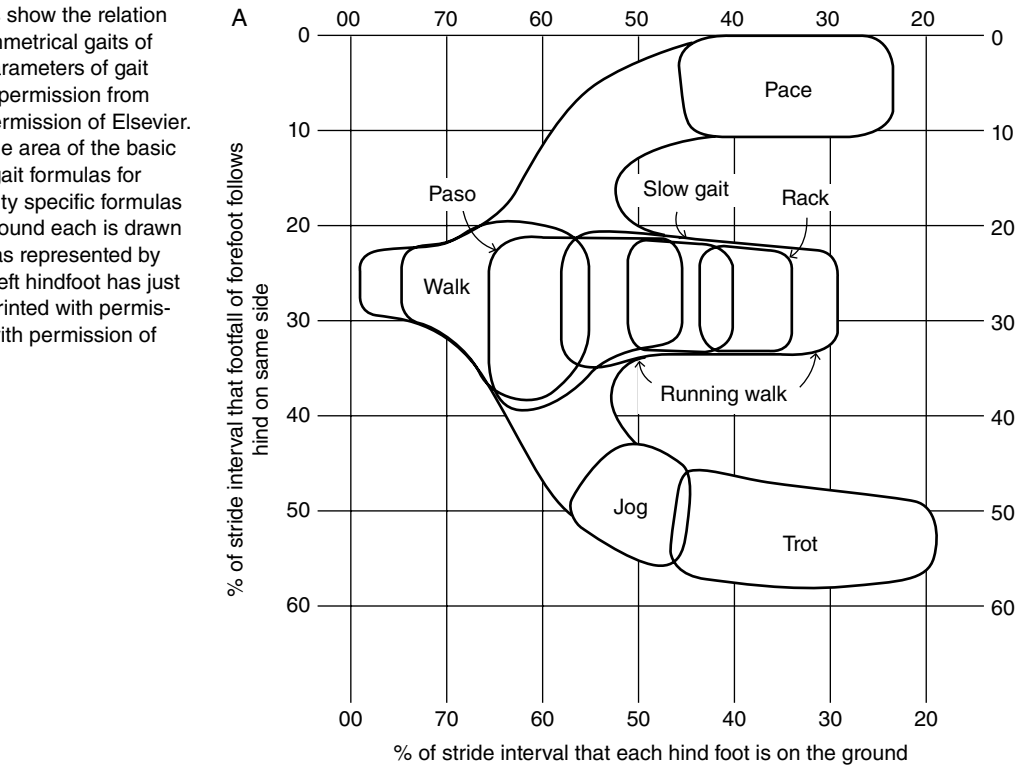
Rack (single foot) is a four-beat stepping gait with a regular rhythm with lateral couplets. This gait is similar to a pace and paso llano as the lateral limbs move in couplets (LH, LF, RH, RF). Each foot hits the ground at equal, separate intervals[5] (https://www.youtube.com/watch?v=vKHjWSn44ZA, accessed 30 January 2020).

Slow gait (stepping pace) is a four-beat stepping gait that is highly collected with a regular rhythm with lateral couplets (LH, LF, RH, RF), similar to but slower than the rack. The ipsilateral fore- and hindlimbs lift almost simultaneously, but the hindfoot contacts the ground prior to the forefoot.[5]

Paso fino gaits are evenly spaced four-beat lateral gaits with each foot contacting the ground independently in a regular sequence at precise intervals creating a rapid, unbroken rhythm. When executed perfectly, the four hoof beats are absolutely even in both cadence and impact, resulting in unequaled smoothness and comfort for the rider (www.pfha.org, accessed 30 January 2020). All four hooves travel close to the ground while in motion and are lifted equally in height as the horse covers ground. At whatever speed the horse travels, the smoothness of the gait ideally allows the rider to appear motionless with little up and down movement.

The paso largo is a four-beat stepping gait with an irregular rhythm with diagonal couplets. According to the Paso Fino Horse Association (PFHA), it is the fastest speed of the gait, executed with a longer extension and

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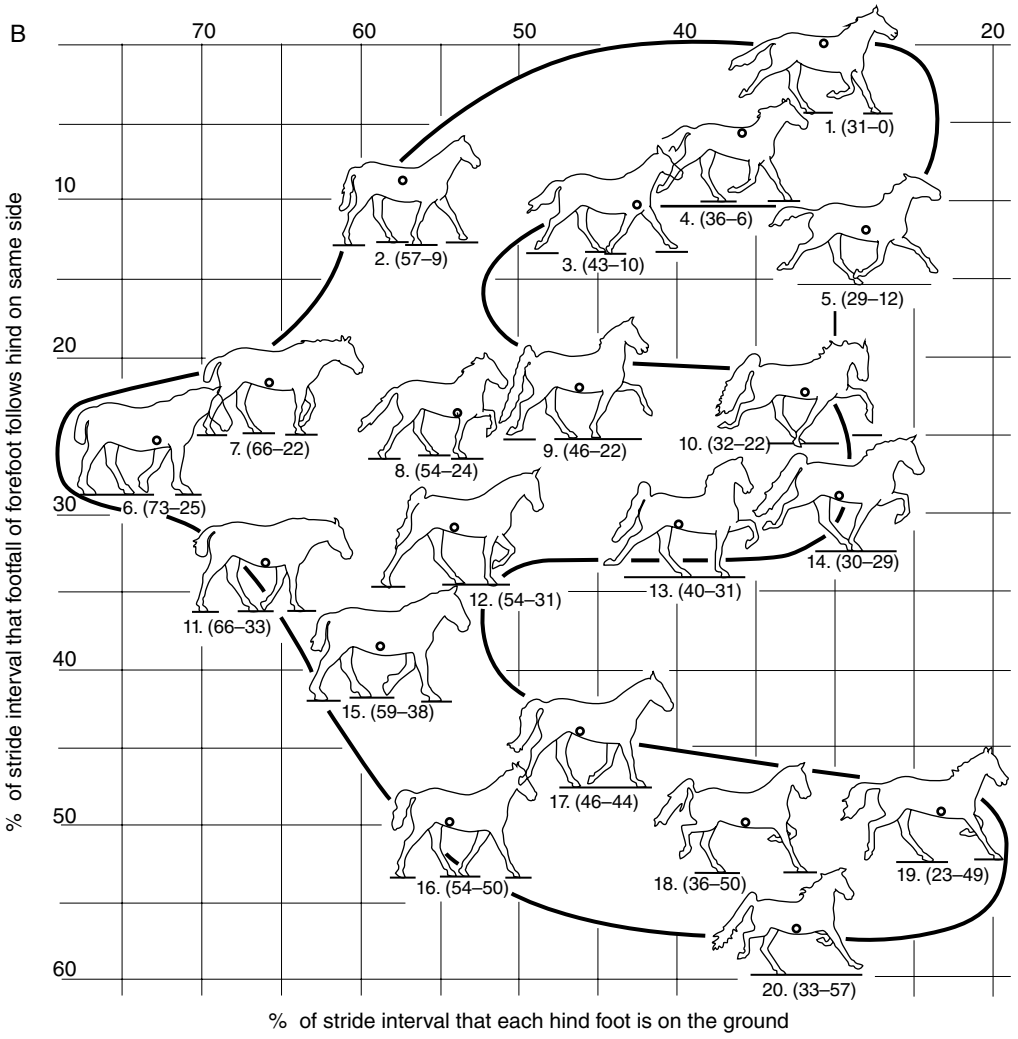


Figure 9.59. (A) Basic graphs show the relation between the principal named symmetrical gaits of horses and their relation to the parameters of gait formulas. *Source*: Reprinted with permission from Hildebrand.[17] Reproduced with permission of Elsevier. (B) In the background is shown the area of the basic graph within which fall nearly all gait formulas for symmetrical gaits of horses. Twenty specific formulas are located (small circles), and around each is drawn a silhouette of the horse moving as represented by the formula. In every sketch, the left hindfoot has just touched the ground. *Source*: Reprinted with permission, Hildebrand.[17] Reproduced with permission of Elsevier.

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stride and moderate to minimal collection. Forward speed varies with the individual horse. The paso largo is a fast, lateral, four-beat gait in which the horse can reach speeds up to 25–30 mph, equivalent to a canter or slow gallop. The paso largo is not just an increase in speed but also shows a distinct extension in stride.

The paso corto is a four-beat stepping gait with an irregular rhythm with diagonal couplets. According to the PFHA, its forward speed is moderate, with full to moderate collection. Steps are ground covering but unhurried, executed with medium extension and stride. The speed of this gait is comparable with the speed of a trot but is much smoother.

The classic fino gait is a four-beat stepping gait with a regular four-beat rhythm. According to the PFHA, it is performed at full collection, with very slow forward speed. The footfall is extremely rapid while the steps and extension are exceedingly short. It is a collected gait with rapid footfalls that covers as little ground as possible. It requires a high degree of collection, the most difficult of the three paso fino gaits.

Peruvian Paso performs a natural ambling four-beat lateral gait, in that it has four equal beats and is performed laterally: LH, LF, RH, RF. A unique trait of the Peruvian Paso gait is *termino*, an outward swinging leg action, originating from the shoulder, in which the front lower legs roll to the outside during the forward movement of the stride, similar to a swimmer's arms.[4]

The paso llano is a four-beat stepping gait with a regular four-beat rhythm. It is the slowest gait of the Peruvian Paso other than the natural walk. This gait is similar to a pace as the lateral limbs move in couplets (LH, LF, RH, RF). The Peruvian Paso can also move in a more diagonal gait, which is called the *pasi-trote* and is similar to the fox trot in foot placement and rhythm without the vertical head movement, characterized by diagonal couplets with forelimb impact being slightly before contralateral hindlimb impact. (https://www.youtube.com/watch?v=I3e3Ea0IDz0, accessed 30 January 2020; https://www.youtube.com/watch?v=ST4QgNsfapc, accessed 30 January 2020).

The sobreandando is a four-beat stepping gait with an irregular rhythm with lateral couplets. It is a fast broken pace. It is faster than the paso llano, but instead of four equal beats, the lateral beats are closer together in a 1–2, 3–4 rhythm (LH-LF, RH-RF), with a longer pause between the front foot of one side to the hindfoot of the other side.

The tölt is a four-beat stepping gait with a regular four-beat rhythm. It is a running walk of the Icelandic pony, sometimes said to be more similar to the rack or paso llano. The horse's hindlegs should move well under the body and carry more of the weight on the hind end, allowing the front to rise and be free and loose. This gait is characterized by the movement of the animal's legs in a lateral sequence (LH, LF, RH, RF), with lateral couplets with a rhythm similar to the sobreandando and paso llano.[6] (https://www.youtube.com/watch?v=RV9P0w8vZi8, accessed 30 January 2020).

Mangalarga Marchador, a Brazilian horse breed, has a natural gait called the marcha, which can be either marcha batida (ba-chee-da), a diagonal movement, or marcha picada, a lateral movement. If the horse ambles equally between the two gaits, it is referred to as having centro movement. Centro is not an actual gait but rather a term used to describe where the horse falls on the gait scale. Of the two gaits, the picada is thought to be considerably smoother than the batida. The marcha picada is a four-beat stepping gait with a regular four-beat rhythm. It is a broken pace with little vertical movement. This gait is characterized by movement in a lateral sequence (LH, LF, RH, RF), with lateral couplets and periods of triple hoof support. The timing of footfalls is similar to the paso llano of the Peruvian Paso Horse. The marcha batida is a four-beat stepping gait with a period of triple support and diagonal couplets (LH, RF, RH, LF). This gait is similar to the trot but shows very little suspension as horses are always in contact with the ground. There is no aerial phase (http://www.marchadorsinmotion.co/gaits, accessed 30 January 2020; http://www.namarchador. org/breed/the-marcha-gaits/, accessed 30 January 2020).

LAMENESS IN THE GAITED HORSE

Lameness in the gaited horse is primarily associated with the hindlimbs. The reason is possibly due to the shift in the center of mass caudally when utilizing these gaits, particularly at speed. The distal intertarsal and tarsometatarsal joints are most commonly affected. Stifle joint osteoarthritis and back pain are also associated with the racking gaits, the running walk, marcha picada, fox trot, etc. However, a study by Axelsson et al. revealed no association of the tölt gait in Icelandic horses being a risk factor for osteoarthritis of the distal tarsal joints. Tarsal joint angles being either too small or too large also may predispose gaited horses to distal tarsal osteoarthritis, but this is not unique to gaited horses.[3,13]

The most unique lameness originally associated with gaited horses is the disorder of degenerative suspensory ligament desmitis (DSLD), first described in Peruvian Paso and Peruvian Paso cross horses. However, this lameness is not necessarily associated with gait. DSLD is a heritable, debilitating disease causing an insidious, progressive onset of bilateral to quadrilateral lameness associated with diffuse enlargement of the suspensory ligaments that are not responsive to rest.[16] Exercise may help to improve or at least stabilize the clinical signs of DSLD.[23] DSLD is now recognized as a more systemic disease of proteoglycan accumulation in connective tissue such as collagen fibrils in tendons and ligaments and smooth muscle cells. Halper proposed to change the name to equine systemic proteoglycan accumulation (ESPA) due to its more systemic effects.[16] DSLD has subsequently been reported in other breeds such as Paso Fino, Thoroughbred, Arabian, Hanoverian, American Saddlebreds, Appaloosa, Quarter horse, and Akhal-Teke (www.akhal-teke.org, accessed 30 January 2020; https://en.wikipedia.org/wiki/Akhal-Teke, accessed 30 January 2020) and in some pony and draft breeds. Akhal-Teke horses are from Turkmenistan, thought to be one of the oldest existing horse breeds. Peruvian Paso horses have been reported to develop the disease at an earlier age than other horses.[15,19]

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Why Is Evaluating Lameness in Gaited Horses Different (More Difficult)?

The vertical movement trajectories of the head and pelvis during the symmetric gaits other than the trot are identical in concept to the trot, which means that they move down during the first half and up during the second half of stance. Therefore, the same principles of altered movement with lameness apply, with "lowest on sound" for both head and pelvic movement for fore- and hindlimb lameness, as an accurate indicator of side of lameness. Also, with the ipsilateral gaits, there is more rolling from side to side than vertical movement down and then up. So, although in principle vertical movement of the head and pelvis should be good indicators of lameness, they may be more difficult to observe. Certainly, the compensatory lameness patterns for ipsilateral gaits will be different than that for the contralateral trot.

There are many reasons why gaited horses are more difficult to evaluate for lameness. The trot is the most common gait of horses in the United States, and horses that trot or pace have a suspension phase to their stride such that the relative impact is greater in horses that trot or pace. Most equine lameness can be considered weight-bearing lameness, with a decrease in peak vertical ground reaction force as the main effect. Therefore, gaits with higher normal vertical excursion of the center of mass, and higher vertical ground reaction forces experienced by the limbs, will more apparently display lameness. Horses that typically move in gaits without a clear suspension phase are more likely to mask low-grade pain on weight bearing.

Those familiar with evaluating horses at a trot recognize asymmetries of movement such as a head nod, hip hike or hip drop, shortened stride, asymmetric hock height, etc., as well as compensatory lameness (ipsilateral forelimb or contralateral hindlimb). Veterinarians must shift their thinking to evaluate gaits that fall in the continuum between trot and pace. One way to do that is to separately evaluate the forelimb and hindlimbs. The gaits are still symmetric, but they are asymmetric between fore- and hindlimbs. The forelimbs should move symmetrically, and the hindlimbs should move symmetrically. Therefore, the evaluator may want to train their mind to look at each half separately. Some gaited horses have an exaggerated head movement, but it still should be symmetric between right and left forelimbs. Hindlimb lameness can be difficult to evaluate in gaited horses because they do not have a typical suspension phase and therefore their hip movement or gluteal asymmetry may often be minimal compared with horses at a trot or pace.

Due to the rapid movement of the limbs in some gaited horses, particularly for horses performing the rack, classic fino gait, or the paso largo gait, the evaluator may wish to improve their chances of identifying a lameness or asymmetry in gait by having the horse travel over a hard surface. The speed of limb movement is often near the threshold of the human eye's ability to accurately discriminate movement and/or placement of the limbs. The combination of eyes and ears may help the evaluator identify the lameness or asymmetry. The sounding board used to show and evaluate Paso Fino horses can be used to help evaluate horses with a rapid gait.

Gaited horses are often evaluated under saddle in their regular environment. The extra weight of the rider and saddle will increase the ground reaction force and therefore may help to exacerbate a mild lameness. They may also more easily maintain a consistent speed or gait when ridden than when moved in hand.

Using Body-Mounted Inertial Sensors (The Q™ and Lameness Locator®) to Evaluate Lameness in Gaited Horses

Gaited horses can be evaluated for lameness using body-mounted inertial sensors, but there are some unique characteristics for acquiring data and important data interpretation facts that need to be understood when using currently available systems.

With the exception of the standard walk, any symmetrical gait that causes a falling and rising of the body twice per stride can be analyzed with body-mounted inertial sensors. This two-phase, sinusoidal-like vertical movement pattern, with downward movement of the torso during the first half of stance (impact) and upward movement during the second half of stance (pushoff), is generally retained even with the four-beat gaits like the fox trot (forelimb hits the ground slightly before contralateral hind limb, i.e. a broken trot) and the stepping pace (hindlimb hits the ground slightly before the ipsilateral forelimb, i.e. a broken pace). Measurement of head and pelvic trajectory and calculation of differences in maximum (between the stances) and minimum (within the stances) heights remain valid for determining lameness. At the standard walk, with a cadence of 1–2–3–4 (LH, LR, RH, RF), and equal timings between footfalls, this biphasic vertical movement is shifted so that maximum torso heights are highest during stance and lowest between stances. Current software does not include algorithms that detect this shift, and, therefore, they are not valid for the walk. Data can be collected at the walking gaits, and an amplitude of asymmetry may indeed be measured and reported, but the side and timing of lameness may not be accurate. However, weight-bearing lameness is less measurable (and less visible) at the walk than at the faster gaits, and the importance of measuring lameness at the walk is minimal. While it may appear that some lameness is better displayed at the walk than at other gaits, this is really only because the movement is slow enough for the limited temporal resolution of subjective observation to pick up the movement disturbance. A horse demonstrating lameness at the walk will have measurable lameness at the trot.

With the ipsilateral gaits (pace, stepping pace, etc.), hindlimb lameness analyses need to be flipped, i.e. left hindlimb lameness is a right hindlimb lameness and vice versa. This is because hindlimb footfall, which is currently determined by the right forelimb gyroscope and the assumption that the horse is moving in a contralateral gait, is one half-cycle out of phase, i.e. right hindlimb footfall is coded as left hindlimb footfall and vice versa. Correct hindlimb footfall sequence can be accurately determined by pelvic rotation from the sensor on the pelvis (generation 5 Q™ sensors include a three-axis gyroscope), but as of the date of this writing, this has not been fully tested and implemented in Lameness Locator®

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software. Amplitude and timing of hindlimb lameness (impact vs. pushoff) are correctly measured, but the side of lameness is opposite of actual. So, why retain the right forelimb gyroscopic sensor? The combination of the right forelimb gyroscope to detect forelimb footfall order and a pelvic gyroscope measuring frontal plane rotation to detect hind limb footfall order allows instantaneous (on-the-fly) determination of ipsilateral from contralateral gait. Future versions of Lameness Locator® are expected to include this capability, so users will not need to specify gait.

Many four-beat gaits are characterized by suppressed vertical torso movement (this is what makes them comfortable to ride) and, especially in the Missouri Foxtrotter, exaggerated vertical head movement. Vertical movement of the head remains the best method of forelimb lameness detection in the four-beat gaits; however a small difference between right and left strides, which, subjectively, are easily seen in a standard trotting horse, may be more difficult to appreciate in the Missouri Foxtrotter and other breeds with exaggerated vertical head movement. It is a question of seeing a small signal against the background of a lot of surrounding noise. Also, because vertical hindquarter movement is damped, detecting small differences in vertical pelvic movement between right and left is more difficult.

These same limitations affect objective measurement with inertial sensors. Lameness Locator® software includes a standardization algorithm that adjusts lameness measures based upon expected vertical movement of the head and pelvis. This is accomplished with algorithms that decompose the vertical movement trajectories into harmonic components, the expected vertical movement being the component at twice stride frequency. This is provided so that a mild lameness measure for a bounding Warmblood in dressage competition (large range of expected vertical movement) will be approximately the same as a mild lameness measure in a miniature horse trotting across the exam room floor (small range of expected vertical movement). Larger asymmetric movements are needed to exceed normal reference ranges when expected vertical movement is high. Smaller asymmetric movements are considered normal reference ranges when expected vertical movement is low. However, at the extreme ends of the total vertical movement spectrum, such standardization may cause discrepancy between what asymmetry is visually apparent and what is actually measured. So, the exaggerated normal vertical head movement in the Missouri Foxtrotter may suppress forelimb lameness results and variation, and the minimized normal vertical pelvic movement (actually in some cases near zero) may exaggerate hindlimb lameness results. Therefore, it is not uncommon for Missouri Foxtrotters (and other breeds that gait with exaggerated vertical head and suppressed vertical torso movement) to measure with forelimb lameness that seems less than and with hindlimb lameness that seems greater than that seen with the naked eye.

Some breeds, most notably the Missouri Foxtrotter and the Standardbred pacer, will not move with a consistent gait when led on a lead shank by a handler, and they do not commonly lunge. Instead, when in hand, they may move at a gait transitional between a walk and fox trot or between a trot and a pace or simply at a very fast walk. Some do not lead at all. This inconsistency of gait is inherently and normally asymmetric, unrelated to pain, so incompatible for determining lameness. For the Missouri Foxtrotter, having the handler force the horse to move faster, by moving faster him/herself, may cause the horse to begin to trot regularly, and the lameness may then be seen or measured. But this may not be ideal, as the owner/trainer is complaining about lameness at the fox trot, and the handler will more quickly tire so that additional observation trials are more difficult to collect. Instead, the lameness evaluation is more likely to succeed if conducted with the horse under saddle and ridden. In this manner, gait and speed are more consistent, and both the fox trot and trot can be evaluated. The Mangalarga Marchador (performing the *marcha batida)* is very similar to the Missouri Foxtrotter both in gait and consistency while led and under saddle (Figure 9.60).

Similarly, it is almost futile to attempt to have a handler lead a Standardbred pacer and expect a consistent gait of either a trot or pace. The best method for evaluating pacers for lameness is to sit behind them in the bike with the horse tacked up fully. Also, simply jogging slowly, even in hobbles, may not cause the horse to gait symmetrically, until a certain speed (usually faster) is attained (Figure 9.61). This can be accomplished with inertial sensors either by placing the receiving tablet computer on the person of the driver or within the sulky, traveling in a vehicle within range of the moving horse, or connecting to and collecting data from sensors as the horse is moving by the data collector holding the receiving tablet. Placing the receiver in the sulky with the driver or traveling next to the horse allows collection of hundreds of strides with low stride-by-stride variability, such that the effect of directional change in the turns is minimized over the entire trial. There is more side-to-side rolling in a Standardbred pacer, but the torso (and head) still rise and fall twice per full stride. However, because the horse is pacing, hindlimb lameness results must currently be flipped, right to left and left to right, but forelimb lameness results are the same as if the horse were trotting at speed. Of course, flipping the hindlimb results is not necessary for the Standardbred trotter and other breeds that trot at high speed, and a more ideal combination of gait (the trot) and circumstance (ability to collect large numbers of contiguous strides at speed) for evaluation with inertial sensors would be hard to find. Because of the presence of the head check and crupper, which occupy the body locations ideal for body-mounted inertial sensors, increased diligence is needed for proper attachment of the head and pelvic sensor. Some users around the world have invented attachment devices that seem to work well.

There is no published information on using body-mounted inertial sensors to evaluate American Saddlebred horses at the rack (an ipsilateral gait) or in other breeds like the Icelandic horse in similar symmetric ipsilateral gaits. These authors have evaluated some American Saddlebred horses for lameness at the rack and have found that the variability in gait is relatively high and trial length (ability to collect multiple contiguous strides) is relatively low, both of which significantly interfere with the ability to find small consistent asymmetries of mild lameness or to detect partial improvements

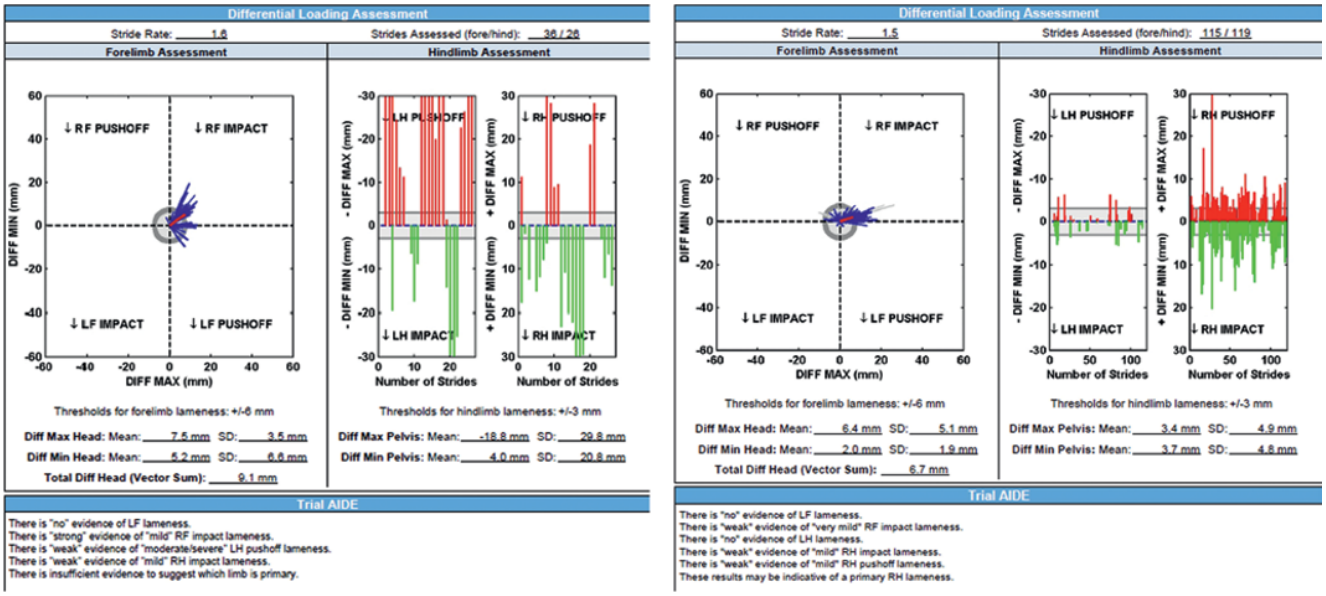


Figure 9.60. (Left) Attempt to measure lameness with inertial sensors in a Missouri Foxtrotter led by a handler. Note: There is no consistent hindlimb asymmetry measured. (Right) Consistent mild right hindlimb lack of pushoff and lack of impact are measured during foxtrot under saddle.

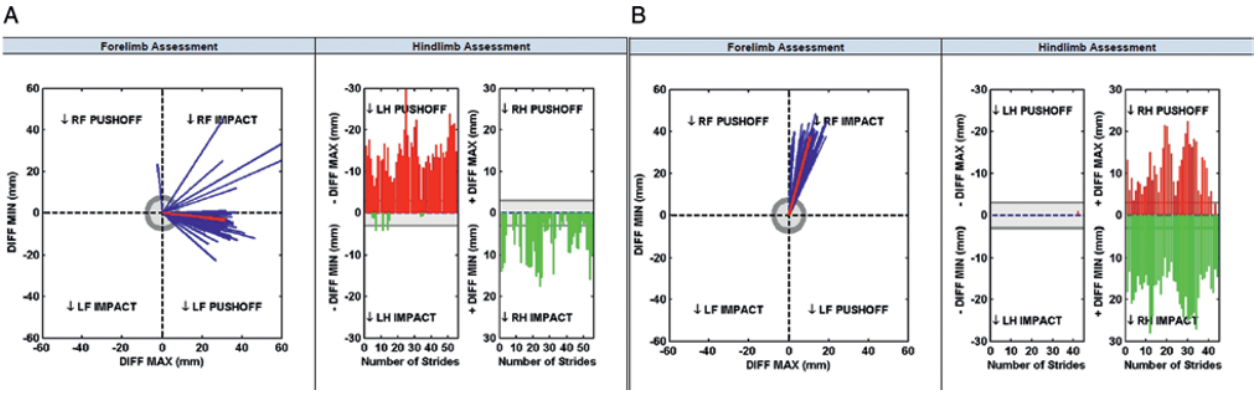


Figure 9.61. Inertial sensor data (A) from pacer that is jogging in hobbles. Note: No clear forelimb lameness, pushing off less in left hind and coming down less in right hind. Difficult to decide which limb is lame. (B) When same horse is pacing at speed. Note: Clear right forelimb lameness with compensatory left hindlimb lameness.

in mild lameness after blocking. Because the rack is an ipsilateral gait, using currently available Lameness Locator® software, the hindlimb lameness results must be flipped for the rack. However, this is of little consequence because most American Saddlebreds trot readily, such that evaluation for lameness at the rack is usually not required (Figure 9.62).

Lameness Locator has been tested in the Missouri Foxtrotter, Standardbred Pace, and American Saddlebred Rack. Based on these findings and clinical observations in these and other gaits, the following general guidelines can be made for some other common gaited breeds.

Tennessee Walking Horse and Rocky Mountain Horse Running Walk

These ambling gaits do not have a small extra duration between second and third footfalls needed to differentiate contralateral from ipsilateral hindlimb foot fall, so they will be OK for forelimb, but hindlimb results may not be reliable.

Paso Finos (Classic, Corto, Largo)

Because the footfall cadence is LH-LF-RH-RF, similar to the walk, the algorithms used in Lameness Locator will have trouble discerning the side of hindlimb lameness. Forelimb evaluation will likely be accurate in the largo, but hindlimb results may be unreliable at all three gaits. The classic is a poor gait to evaluate lameness as the torso is not really moving up and down. It is primarily only the legs moving with no upper body movement. It is like the horse is doing the Irish jig.

The Icelandic Tölt and Flying Pace

The tölt is a four-beat lateral ambling gait and will likely be measured similarly to the Saddlebred rack or

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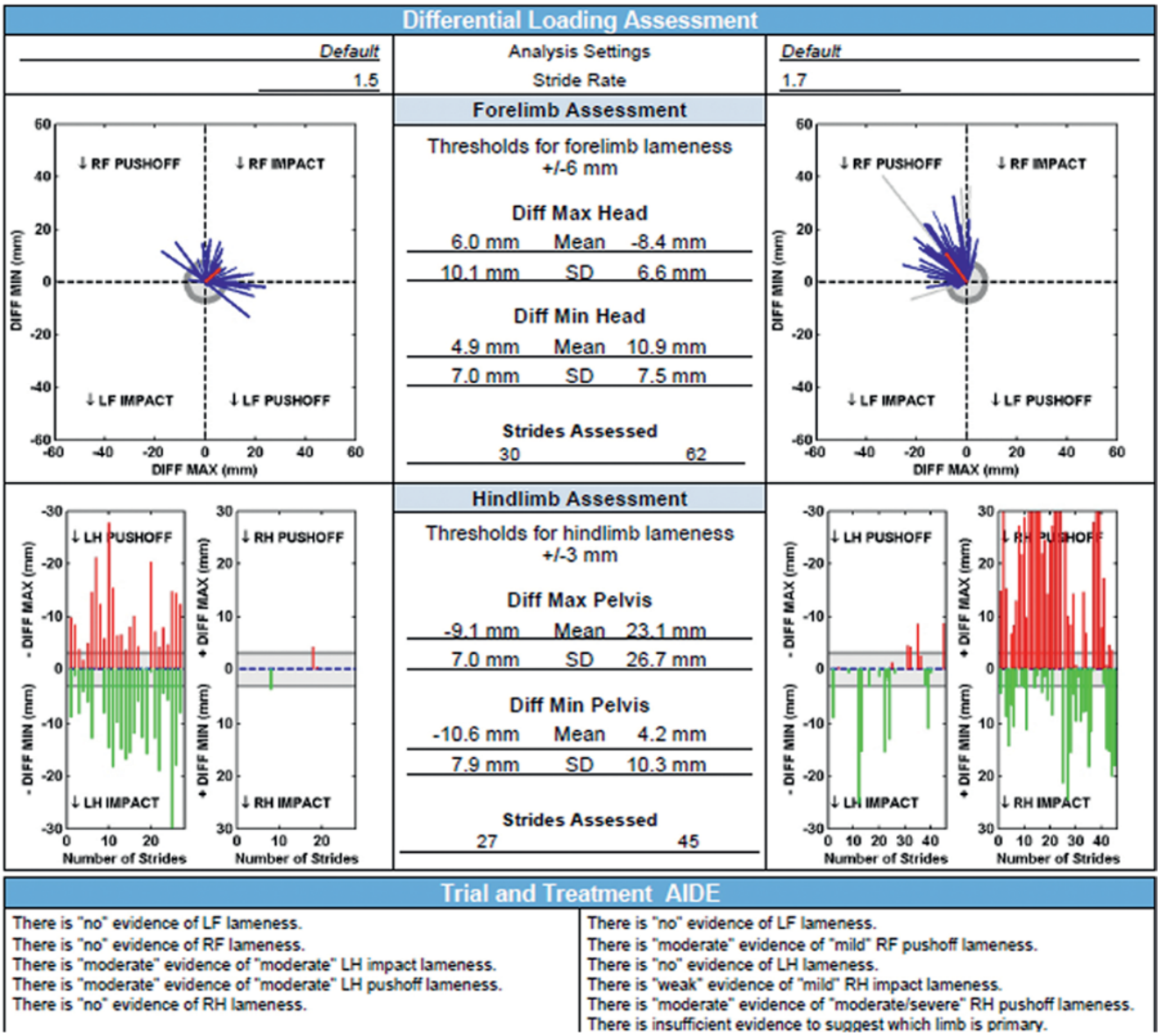


Figure 9.62. (Left) Evaluation of an American Saddlebred for lameness at the trot. A clear left hindlimb lack of pushoff and impact lameness are found with variation (standard deviations) of Diff Max and Diff Min Pelvis less than mean indicating good consistency. (Right) Evaluation of the same American Saddlebred horse at the rack. Note the right hindlimb lameness, which should be flipped to a left hindlimb lameness due to the data collection of an ipsilateral gait. Also, note the relatively high variation compared with means of Diff Max and Diff Min Pelvis, with standard deviations greater than means.

running walk of the Tennessee Walking Horse. Therefore, forelimb results will be reliable, but hindlimb results will be unreliable. The pace is a two-beat, lateralized gait with a moment of suspension between footfalls. Forelimb results will be reliable, and hindlimb results will be opposite of true.

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THE DRAFT HORSE

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INTRODUCTION

Evaluating lame draft horses presents unique challenges due to of their large size, which complicates handling, routine farriery, and the lameness evaluation. Fortunately, most draft horses are docile and amenable to restraint. Draft horses are rarely exercised at speeds greater than a trot. This can delay the onset of obvious signs of lameness because of the lower amount of athletic demand required. This section reviews the anamnesis, physical and lameness examination findings, diagnostic tests, common lameness disorders, and treatments for lameness in the draft horse.

ANAMNESIS

Knowing the type of athletic demand placed on the horse is useful in determining the most likely cause of lameness. Common uses for draft horses include pulling, farm labor, showing in a hitch (either alone or with other horses), dressage, or broodmare. Young horses (under 2 years of age) presenting with lameness should be evaluated for metabolic bone diseases such as osteochondrosis and subchondral bone cysts.

LAMENESS EXAMINATION

All draft horses should have a complete physical examination because some cases presented for lameness evaluation may have systemic disease (e.g. endocarditis). The lameness examination should begin with a visual examination of the limbs and trunk. Next, the limbs, neck, back, and pelvis should be carefully palpated for evidence of asymmetry and focal pain or swelling. Chronic forelimb lameness can contribute to foot asymmetry and muscle atrophy of the shoulder muscles. Chronic hindlimb lameness can result in gluteal muscle atrophy.

Palpation of the lower extremities is complicated by the presence of long hair (feathers) in the fetlock and pastern regions. Palpation is also hampered because of the thick nature of the skin covering the distal extremity. Picking up the foot for applying a hoof tester can be challenging because not all draft horses willingly allow the examiner to pick up the foot. If necessary, the horse can be placed in stocks and a hobble placed around the pastern of the affected limb. A rope is secured to the hobble and the foot hoisted. Hoof tester application is often not helpful because the soles and hoof walls are typically thick and hard. Nonetheless, hoof testers should be applied to fully evaluate the hoof capsule, sole, and frog.

Lameness should be evaluated at the walk and trot. Horses should be evaluated on a straight line and while trotted in a circle. If possible, competition hitch horses should be evaluated while pulling a cart or wagon. Flexion tests and diagnostic anesthesia are performed as in light breed horses. Diagnostic nerve blocks are more difficult because of the thick skin of the distal extremity. The thick skin may limit diffusion of local anesthetic; therefore, greater amounts of anesthetic solution are often needed, and observation times should be longer to allow for greater diffusion. Higher kVp and mAs are required for radiography due to the increased size and thickness of the foot and distal extremity. Ultrasonography of the lower limb (below the hock or carpus) is complicated by the thick skin and dense hair on the distal extremity, making careful skin preparation necessary.

COMMON CAUSES OF LAMENESS

Lameness of the Foot

The most common cause of lameness in the draft horse involves the foot due to poor hoof wall quality, improper or no trimming, and inadequate hoof hygiene (Figure 9.63). Tie stalls and confinement in wet, manure-contaminated stalls can be risk factors for disorders such as subsolar abscessation and proliferative pododermatitis (canker). It is difficult for owners to regularly pick up and examine the foot, and many farriers are reluctant to provide routine hoof care for draft horses because of the physical demands of holding up a large draft horse.

Farriery can be greatly aided with shoeing stocks. Some owners possess shoeing stocks and perform routine farriery on their own. Feet also can be hoisted with the help of a rope secured to a hobble placed around the pastern. Shoes for draft horses are not readily available



Figure 9.63. Hoof care can be more difficult in draft horses, predisposing to foot problems, including hoof cracks.

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because most are custom made, thus adding to the expense of routine hoof care.

Most draft horses not used for athletic use or pulling are frequently left unshod. Those left unshod and not trimmed regularly by a farrier are prone to overgrown hoof walls. Overgrown hoof walls are prone to hoof cracks, which can involve the sensitive laminae, and poor hoof care in general can contribute to chronic foot soreness and lameness (Figure 9.63).

Subsolar Abscessation

Subsolar abscessation is common in draft horses. Risk factors include inadequate hoof care and poor stall hygiene.[8,12,13,27] Hoof testers should be applied to the sole, hoof wall, and frog in an attempt to localize focal areas of pain. In some instances purulent discharge can be expressed with the hoof testers. Feet also should be carefully evaluated for foreign body penetration. Insensitivity to hoof testers does not preclude a diagnosis of subsolar abscessation. Draft horses believed to have subsolar abscessation should have a palmar digital or abaxial sesamoid block performed to confirm the diagnosis and aid in trimming of the foot and sole exploration for an abscess. If necessary, affected horses should be restrained in shoeing stocks or sedated for examination to facilitate foot examination. Inability to find an abnormal region of the sole following hoof trimming does not rule out a diagnosis of subsolar abscessation. Soaking the foot can help soften the sole and aid identification and drainage of subsolar abscessation.

Affected horses should be treated with NSAIDs as needed to facilitate patient comfort. Most abscesses resolve promptly with adequate drainage and do not require antimicrobials. It is important to remove the undermined sections of the sole involved in the abscess to ensure adequate drainage. The author recommends placing a foot bandage initially. Once cornification begins, a shoe with a treatment plate can be substituted for the bandage. The shoe can be removed following complete cornification of the sole defect, if desired, and the horse returned to use. The prognosis for routine subsolar abscessation is good.

It is a rare case (less than 5%) in which extensive subsolar abscessation leads to the development of septic osteitis of the distal phalanx in draft horses. Radiography is required to determine the amount of the distal phalanx involved. Debridement of the third phalanx is accomplished with the horse standing and is combined with perineural anesthesia of the digital nerves.

To access the distal phalanx, a portion of the sole overlying the abnormal area must be removed, and all abnormal bone should be curetted. The defect is packed with sterile gauze and a sterile bandage is placed on the foot. For large sole defects, a shoe with a treatment or hospital plate should be considered to protect the sole and facilitate foot hygiene. The prognosis for septic osteitis of the distal phalanx is fair to good as long as 25% or less of the distal phalanx is involved.[3,10]

Infectious Pododermatitis

Infectious pododermatitis (canker) is commonly diagnosed in horses housed in stalls with inadequate hygeine.[17,30] Various treatments have been described for managing infectious pododermatitis, including topical application of caustic agents such as formalin and hydrochloric acid, topical sulfa drugs and metronidazole, cryosurgery, and limited and aggressive surgical debridement. The combination of limited surgical debridement and topical application of metronidazole results in the best outcome.

Surgical debridement can be performed under general anesthesia or while standing.[11] Debridement under anesthesia is preferred because of improved visualization, lack of patient movement, and superior hemostasis. Standing surgical debridement is facilitated with intravenous sedation (detomidine hydrochloride and butorphanol tartrate) and perineural anesthesia of the palmar digital nerves. A hobble is secured around the pastern, and the leg is hoisted and secured with a rope held by an assistant or attached to stocks. A tourniquet is essential to control hemorrhage and improve visualization. All visible portions of abnormal frog and sole are resected with a sharp hoof knife or scalpel blade. Following debridement, a paste of metronidazole tablets (equivalent to a systemic dose, 15–30 mg/kg) is applied to the debrided foot. A sterile bandage is then placed on the foot. A treatment plate aids postoperative management.

Topical application of metronidazole is performed at least once daily until the debrided area of the sole and frog is completely cornified, which may take 6–8 weeks. Aftercare is time consuming and must be performed daily until the defect has completely cornified. Owners must understand the absolute requirement for good foot hygiene following treatment. If the horse is placed back into the same unhygeinic conditions, the condition will most likely recur. The prognosis following treatment is fair to good.

Sidebone

Ossification of the collateral cartilage occurs to some degree in all draft horses. Fortunately, lameness related to sidebone is rare. Sidebone can be indicative of chronic foot lameness, even though it is not the primary cause for lameness. All suspected cases of sidebone should be confirmed with diagnostic anesthesia to rule out other more common disorders of the foot.

Radiographic findings compatible with sidebone include complete to incomplete ossification of the collateral cartilages.[21,22] Separate centers of ossification are not unusual and should not be confused with a fracture of the collateral cartilage. Bone-phase nuclear scintigraphy can be useful in determining the significance of collateral cartilage ossification to lameness.[22] Corrective farriery is the treatment of choice for management of sidebone. The foot should be balanced medial to lateral, and a long-toe, low-heel conformation should be avoided. Corrective shoeing should include a rolled toe with quarter clips to stabilize the foot, or a bar shoe with clips can be considered. The prognosis for sidebone in confirmed cases is favorable. Nonresponsive horses can be considered for palmar digital neurectomy.

Distal Phalanx Osteitis

Nonseptic osteitis of the distal phalanx is not unusual. Contributing factors include heavy body weight,

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poor hoof care, thin soles, broken hoof walls, and exercise on hard surfaces. Horses with distal phalanx osteitis typically present with a history of bilateral forelimb lameness that is worse when worked on hard surfaces (e.g. gravel, pavement, dry ground). Most horses with distal phalanx osteitis have increased sensitivity to hoof testers across the toes, quarters, and sometimes the heels. When trotted in hand, affected horses have clinical signs of bilateral forelimb lameness, which may be worse when trotted in a circle. Approximately 50% of horses improve following a palmar digital nerve block, and the remaining 50% improve following a midpastern ring block or an abaxial sesamoid nerve block. The author prefers a midpastern ring block because it is more specific for the foot and pastern.

Radiographic findings associated with nonseptic osteitis include lucency along the margins of the distal phalanx and proliferative new bone formation along the margin of the distal phalanx, which can extend up the hoof wall. Treatment includes NSAIDs and corrective farriery, which should include the placement of a wide-web shoe with a rolled toe. The inside of the shoe should be filled with shock-absorbing material such as dental impression material. This type of material is very important in managing the lameness because it protects and supports the sole to minimize compression and concussion to the distal phalanx. Cases that do not respond to corrective farriery and sole support may be considered for a palmar digital neurectomy.

Hoof Cracks

Hoof cracks involving the insensitive and sensitive laminae are common in draft horses. This is in part due to the difficulty in keeping the feet trimmed but may also be secondary to previous coronary band trauma. More than 75% of hoof wall cracks in draft horses involve the insensitive laminae, and these cracks can be managed with corrective shoeing and proper trimming of the hoof wall. Grooving the hoof wall at the most proximal aspect of the crack can be useful in preventing crack propagation toward the coronary band. If desired, insensitive hoof wall cracks can be managed with hoof wall staples and epoxy/acrylic repair, with a good prognosis.

Cracks involving the sensitive laminae result in lameness and require stabilization. Acceptable methods of stabilization include staples, acrylic repair, and a bar shoe with clips on each side of the crack.[18,19] If epoxy/acrylic repair is chosen, it is very important that the crack be completely cornified prior to repair to reduce the risk of abscessation. Hoof wall cracks associated with coronary band defects may require surgical reconstruction of the coronary band defect to prevent continued cracks. The prognosis is usually good. The most common complication is infection associated with hoof crack repair.

Laminitis

Laminitis, although not diagnostically challenging, is difficult to manage in draft horses because of their heavy body weight. Common causes of laminitis in draft horses include grain overload, following gastrointestinal disease (e.g. diarrhea, postabdominal surgery), retained placenta, and contralateral weight-bearing lameness. These horses

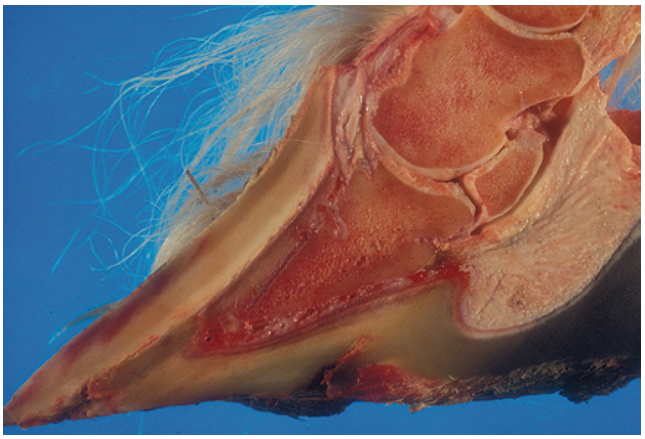


Figure 9.64. Cross section of a Clydesdale's foot demonstrating the long toe and concavity of the dorsal hoof wall that may predispose these horses to laminitis.

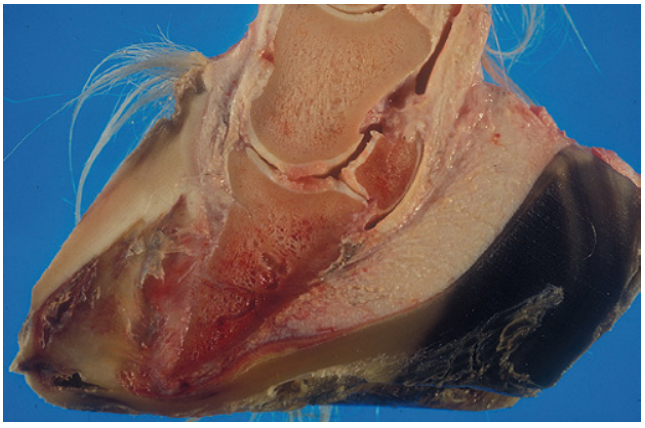


Figure 9.65. Cross section of a Clydesdale's foot with severe chronic laminitis. Significant movement of the distal phalanx is often present at the initial evaluation of draft horses, and preventing further movement can be challenging.

are also commonly shod with long toes and exercised on hard ground, which also may predispose them to laminitis (Figure 9.64). Laminitis can involve rotation, distal displacement of the distal phalanx (sinking), or a combination of rotation and distal displacement of the third phalanx.[1] The author has found no universally successful method for managing laminitis in draft horses. Laminitis is difficult to stabilize so that no further rotation or distal displacement occurs (Figure 9.65). The risk factor that most likely explains this is the large body weight.

The cornerstone of management is corrective shoeing, and working with a farrier is crucial to a successful outcome. The author prefers the application of wedge shoes with sole/frog support to relieve the pull of the deep digital flexor tendon and to support the sole and frog. Because of draft horses' large foot size, custom-made shoes are often required. Some draft horses respond well to application of heart-bar shoes with added sole and frog support. Draft horses with distal phalanx rotation greater than 15° are candidates for deep digital flexor tenotomy.[7] This should not be considered as a salvage procedure but as a useful tool in the management of laminitis. It

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can make a significant improvement, along with corrective farriery, in derotating the distal phalanx. Regardless of the treatment chosen, the prognosis for laminitis in draft horses is guarded to poor and is considered to be worse than for light breed horses.

Osteoarthritis of the Distal and Proximal Interphalangeal Joints

OA of the distal (low ringbone) and proximal interphalangeal (high ringbone) joints is not unusual. The greatest risk factor is a combination of large body size and work-related activities that pull and twist the lower limb. Radiographic evidence of ringbone is not unusual and may not be responsible for signs of lameness. The radiographic findings are similar to those found in other breeds of horses. Mild cases of ringbone can be managed successfully with conservative management consisting of corrective shoeing, NSAIDs, and intra-articular injection.

Moderate to severe cases of high ringbone that do not respond to conservative management should be considered for pastern arthrodesis. In draft horses, pastern arthrodesis should include at least two lag screws and locking plate application to ensure a strong enough construct to avoid cyclic fatique.[15,16] Pastern arthrodesis is performed less commonly in draft horses than light breed horses, primarily because of the cost as well as anesthetic concerns related to their large body size. Most owners typically opt for retirement from exercise or use these horses for breeding. The prognosis for ringbone in draft horses is favorable in mild to moderately affected horses. Severely affected horses can only be successfully managed with pastern arthrodesis, with a fair to good prognosis.

Osteoarthritis of the distal interphalangeal joint in mildly to moderately affected horses can be managed with a combination of corrective farriery and intra-articular injections.[6] Surgical arthrodesis for severely affected horses may be considered, but most owners reject this for financial reasons.[2,25] Palmar digital neurectomy also can be considered. The prognosis for distal interphalangeal OA is lower than for proximal interphalangeal joint OA.

Suprascapular Nerve Injury

Suprascapular nerve injury, or shoulder sweeny, is caused by direct trauma to the suprascapular nerve. This condition is not uncommon in draft horses because of repetitive injury to the suprascapular nerve from ill-fitting collars. Clinical signs of shoulder sweeny include muscle atrophy of the supraspinatus and infraspinatus muscles, subluxation of the shoulder joint, and lameness. Nevertheless, all affected horses should have radiographs of the shoulder to rule out traumatic injury, including fracture of the supraglenoid tubercle or proximal humerus. The clinical signs of this type of fracture and shoulder sweeny can be similar. Horses with clinical signs of suprascapular nerve injury but no radiographic abnormalities should be treated conservatively for at least 60 days before deciding on surgical decompression of the suprascapular nerve.

Conservative management consists of stall rest and NSAID therapy if needed.[5] Acute cases of shoulder sweeny can be administered corticosteroids and NSAIDs to decrease perineural inflammation. If following 60 days of conservative management no improvement in clinical signs has been observed, surgical decompression of the suprascapular nerve can be considered. The surgical technique has been described previously and is not detailed here.[23] The surgical success rate has been reported as favorable. However, muscle atrophy can persist in some cases, even though subluxation of the shoulder joint and mechanical lameness resolves. The prognosis for conservatively treated horses is fair to good, whereas the prognosis for horses treated with surgery is guarded. Horses that return to use should be monitored carefully for proper fitting of the collar to prevent recurrent suprascapular nerve injury.

Osteoarthritis of the Lower Hock Joints

OA of the tarsometatarsal or distal intertarsal joints (bone spavin) is a common cause of hindlimb lameness in competitive draft horses, especially in show horse hitches. Intra-articular anesthesia of the lower hock joints is indicated, and the author recommends using mepivacaine in both the DIT and TMT joints to confirm the diagnosis. It is not unusual for affected horses that appear lame in only one limb during baseline evaluation to become lame in the contralateral limb following successful intra-articular anesthesia of the originally lame limb. Radiography is not required but is helpful to determine the amount of radiographic change present and establish a baseline for future examinations.

Mildly affected horses can be managed with corrective farriery and NSAID therapy. Corrective farriery can include a square or rolled toe shoe with a 2°–3° heel elevation. The toe of the hoof should be trimmed short to aid break-over. Moderately affected horses also should be treated with intra-articular injections of corticosteroids. The author prefers methylprednisolone acetate for injection into the DIT and TMT joints. Ethyl alcohol also can be used for intra-articular injection to facilitate bony fusion of the lower hock joints.[26] Ethyl alcohol also is neurolytic, and injected horses frequently are more comfortable following intra-articular injection. Those that do not respond to intra-articular injections can be considered for surgical fusion of the lower hock joints. The author currently prefers to perform surgical ankylosis with intra-articular drilling alone.[31]

The prognosis for conservatively treated horses is fair to good, with the majority responding satisfactorily. In the author's experience, fewer than 5% of horses diagnosed with OA of the lower hock joints require surgery. The prognosis following surgery is good, with most horses improving.

Stringhalt

Stringhalt is caused by hyperactivity of the lateral digital extensor (LDE) muscle. Causes include trauma to the LDE tendon, peripheral neuropathy, ingestion of toxic plants, and idiopathic causes.[4,14,28] The classical clinical sign for stringhalt is hyperflexion of the hindlimb with the limb being quickly moved toward the abdomen while walking or trotting. Clinical signs alone can be used to make the diagnosis. The only conservative treatment for stringhalt is administration of phenytoin

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(15 mg/kg, PO, every 24 hours, for 14 days).[14] Phenytoin has been used to improve the gait abnormality associated with Australian stringhalt.

Horses with traumatic injury to the LDE tendon or musculotendinous junction are best treated with LDE myotendonectomy. The author prefers to remove the LDE tendon along with a 5–6 cm of the distal aspect of the LDE muscle belly, thus completely removing the musculotendinous junction. The inclusion of the musculotendinous junction seems to lessen the chance for adhesions between the stump of the resected tendon and extensor tendon sheath, which may contribute to recurrence. The prognosis following surgery is fair to good.

Shivers

Shivers affects draft horses along with other breeds, including Warmbloods.[9,29] It is an unusual problem because the horse typically only does it once it begins to walk or trot. Affected horses pick up their leg quickly, toward the abdomen, and tend to shake the limb before setting it down. It can be confused with stringhalt and upward fixation of the patella. Accompanying the flexion of the hindlimb, the tail head may elevate. The abnormal gait can be exacerbated with lack of exercise, low ambient temperatures, and increased horse anxiety. The condition can be difficult for the farrier to deal with because the unexpected flexion of the limb makes limb restraint difficult. The condition can be unilateral or bilateral and can be progressive. For some horses, the progressive nature of the condition can make it difficult to stand, especially when the condition is bilateral. Clinical signs also can involve the forelimbs.

The cause of shivers is unknown, but equine polysaccharide storage myopathy (PSSM) has been incriminated. A muscle biopsy may be obtained and submitted for evidence of PSSM. Affected horses can be improved with a high-fat, low-carbohydrate diet. No other treatment for shivers has been shown to be consistently effective, and most owners learn to live with the disorder.

DISEASES OF YOUNG DRAFT HORSES

Septic Arthritis

Septic arthritis in draft horse foals is not uncommon and is usually associated with septic omphalophlebitis secondary to a patent urachus. The most commonly affected joints are the hock and stifle, and osteomyelitis is not an uncommon sequelae. The diagnosis and treatment for septic arthritis in draft horse foals is the same as for light breed horses.[24] The prognosis for draft horse foals is similar to that for light breed horses, although the expectations for athletic demand are less.

Osteochondritis Dissecans (OCD)

OCD in draft horses mimics the condition in light breed horses except that the size of the OCD lesion in draft horses is usually larger. The most commonly affected joints are the hock and stifle.[20] Large osteochondral fragmentation of the distal intermediate ridge of the tibia and the lateral trochlear ridge is not uncommon (Figure 9.66). Likewise, for the stifle, large lesions involving the lateral trochlear ridge of the femur are not unusual. Standard arthroscopic techniques for the hock

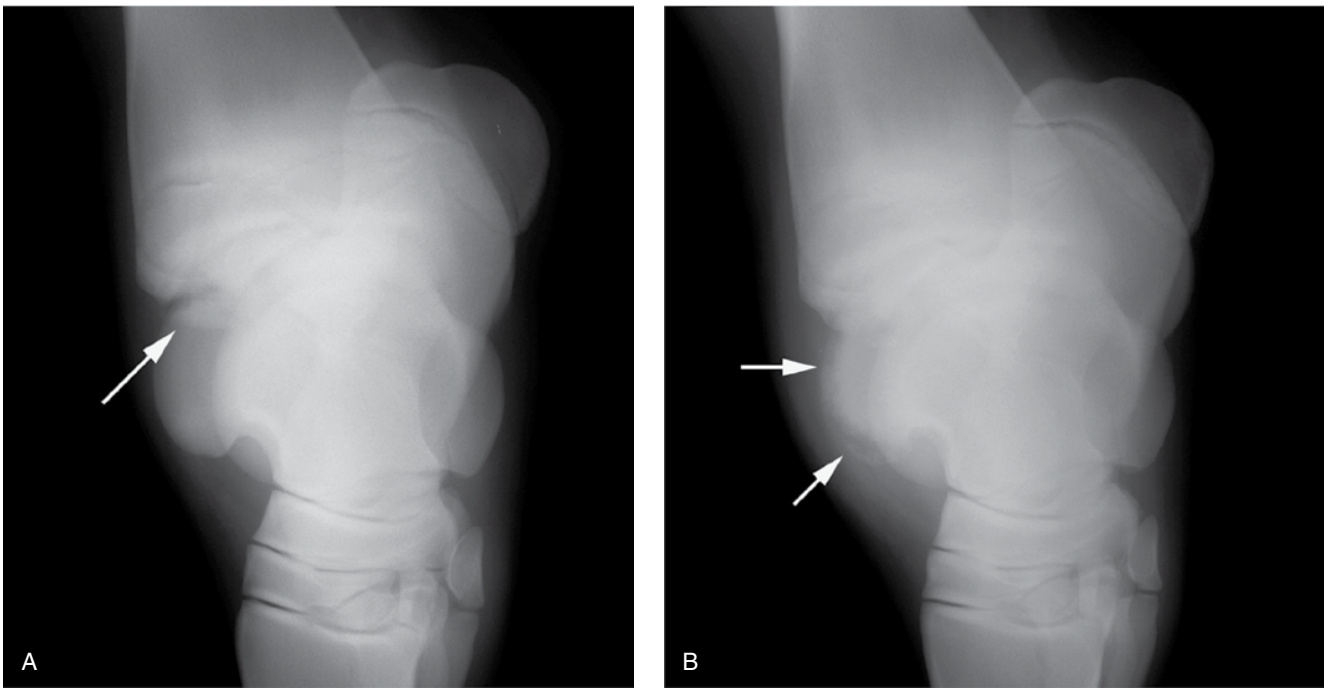


Figure 9.66. Fragmentation or osteochondral defects associated with OCD tend to be very large in draft horses and occur at more than one location within the joint. (A) A large OCD lesion of the distal intermediate ridge (arrow) and (B) an extensive subchondral defect involving most of the lateral trochlear ridge (arrows). The horse in (A) also had a large lesion of the lateral trochlear ridge in the same joint.

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Figure 9.67. Tarsocrural joint effusion is a common finding in draft horses and may persist even if the OCD lesions are removed.

and stifle are used. The prognosis following arthroscopic debridement is favorable in most instances due to the lower athletic demand placed on draft horses. However, despite arthroscopic debridement some horses will continue to have persistent joint effusion (Figure 9.67).

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CHAPTER

Lameness in the Young Horse

THE PHYSIS/PHYSEAL FRACTURES/PHYSITIS

Dane M. Tatarniuk, Troy N. Trumble, and Gary M. Baxter

THE PHYSIS

Growth and Development of the Musculoskeletal System

During fetal development, equine long bones have two main centers of ossification. The primary ossification center develops first and forms the diaphysis and metaphysis of the bone, while the secondary ossification center will form the epiphysis. In between these two centers of ossification is a specialized, highly cellular region referred to as the physis or growth plate. Most long bones have a physis at each end of the bone, whereas others have only one, such as the third metacarpal/metatarsal bone (distally) and the proximal and middle phalanges (proximally). The function of the growth plate is to increase longitudinal bone growth via endochondral ossification, a process where rapidly differentiating and maturing cartilage cells get replaced by bone.[49]

There are two types of growth plates: discoid and spherical.[4] Discoid growth plates are located in long bones between the metaphysis and the epiphysis (Figure 10.1). In addition, a type of discoid physis is present distal to an apophysis (specific type of epiphysis that is subject to tensile rather than compressive forces) on the olecranon process (Figure 10.1), calcaneal tuber, and tibial tuberosity. These physes contain greater amounts of fibrocartilage to withstand tensile forces.[38] Spherical physes are located in the small cuboidal bones of the carpus and tarsus (Figure 10.2). These growth plates develop into bones by centrifugal expansion around a central cartilage core.[4] The cartilage core begins to ossify in the center and gradually assumes the contours of the bone of an adult as bone development reaches the margins of the cartilage model (Figure 10.3).

The epiphysis forms early in life at one or both ends of long bones between the growth plate and the articular cartilage. These articular–epiphyseal complexes form in a manner similar to ossification of the cartilage models of the small cuboidal bones. This ossification mostly increases the size of the epiphyses but contributes very little to the length of the bone (Figure 10.2). The cellular events consist of vesiculation and chondrocyte death with calcification of matrix, invasion of vessels, and partial resorption and ossification.[38] Eventually, a subchondral bone plate forms protecting the articular cartilage portion that persists throughout life.

Endochondral Ossification of the Physis (Growth Plate)

The metaphyseal growth plate or physis has a characteristic cellular architecture from birth to maturity that allows for simultaneous longitudinal growth and ossification of bone. The cartilage cells of the growth plate can be divided into a number of zones, which vary in height and cell number, histological appearance, and cellular function. They include the resting/reserve, proliferative, hypertrophy, provisional calcification, and ossification zones.[49,50] On the outer periphery of the growth plate is a perichondrial ring of collagen fibers oriented vertically, obliquely, and circumferentially often referred to as the ring of La Croix.[18] It encircles the growth plate and extends from the metaphyseal periosteum to epiphysis (Figure 10.4). Its main function is to provide mechanical support for the growth plate while also acting to constraint the circumferential growth of

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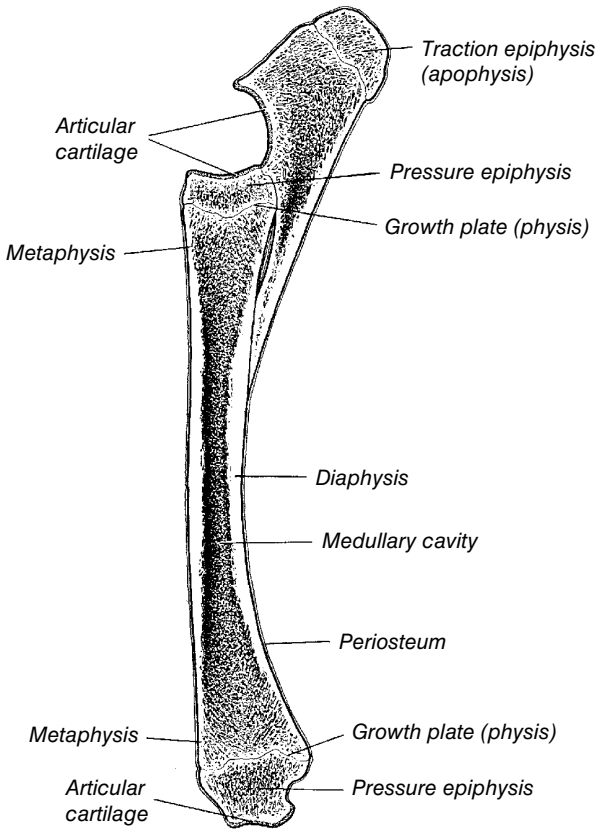


Figure 10.1. Sagittal section of the radius and ulna of a foal.

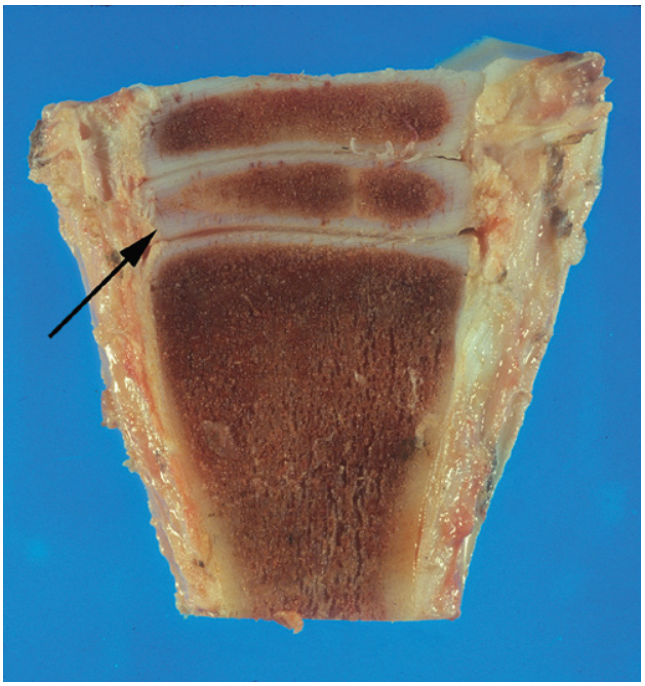
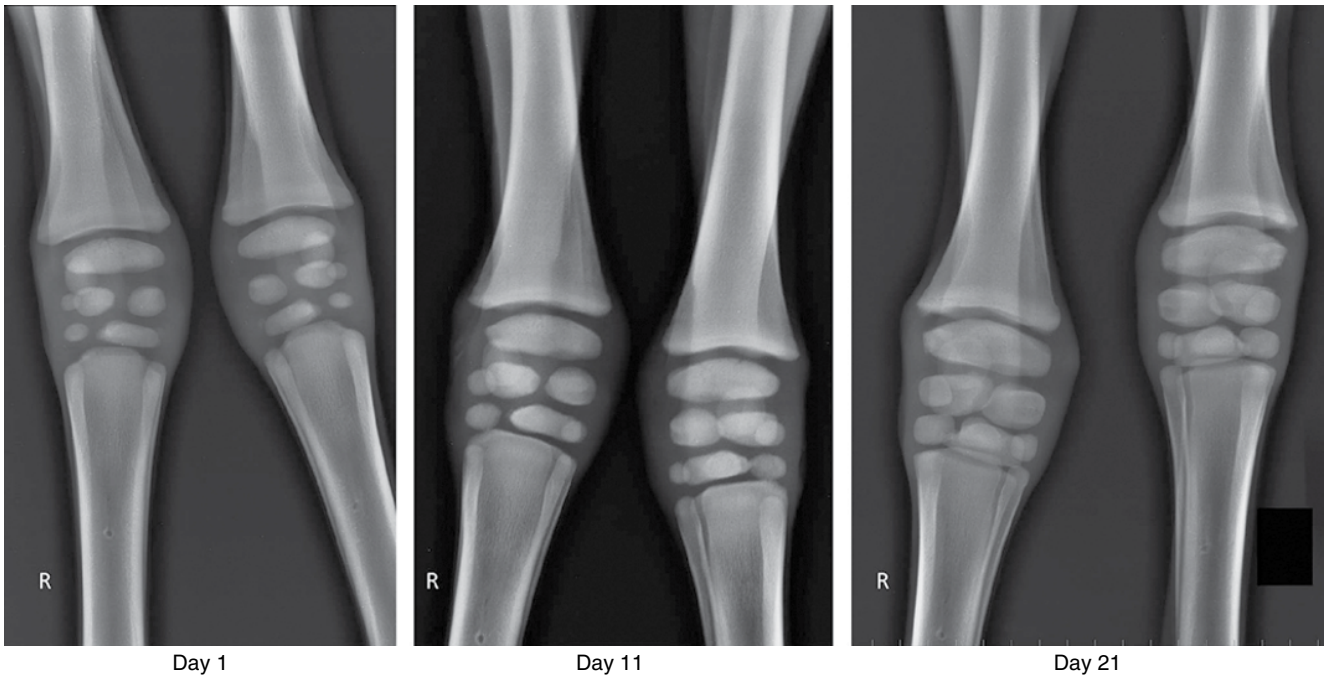


Figure 10.3. Postmortem cross section of the tarsus in a premature foal demonstrating bone surrounded by nonossified cartilage (arrow) within the cuboidal bones. These bones ossify by centrifugal expansion around a central cartilage core.



Day 1 Day 11 Day 21

Figure 10.2. Craniocaudal radiographs of both carpi from the same foal on days 1, 11, and 21 of life demonstrating the rapid centrifugal expansion of the cuboidal bones and distal radial epiphysis after birth.

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the physis.[45] Directly under this perichondrial ring is the zone of Ranvier at the level of the resting and proliferative zones (Figure 10.4).[34] This wedge-shaped zone contains undifferentiated cells, fibroblasts, and progenitor cells for osteoblasts that are supplied to the resting/reserve zone.[52] These cells function to expand the diameter of the growth plate.[52]

Resting/Reserve Zone

The zone nearest the epiphysis (Figure 10.4) contains scattered undifferentiated or resting chondrocytes (similar to those in articular cartilage).[49] These chondrocytes are highly metabolic with high cytoplasmic lipid content that suggest potential for nutrient storage.[1,50] Epiphyseal vessels (arterioles and capillaries) are closely associated with early cellular events and may provide undifferentiated cells that can add to the pool of chondrocytes.[49] Recently it has been shown that this zone contains cells that can act as precursors of the cells in the proliferative zone and it produces an orienting factor that dictates that the cells line up into parallel columns along the long axis of the bone.[1]

Proliferative Zone

The zone of growth or proliferation is where chondrocytes divide rapidly in columns of isogenous groups along the long axis of the bone (Figure 10.4).[49] This orientation directs the lengthwise growth of bone. Chondrocytes farther down the cell columns synthesize and secrete extracellular matrix (predominantly of type II collagen), aggrecan, other proteoglycan aggregates, and type X collagen.[49] Collagen that is randomly orientated in the resting zone becomes more longitudinally oriented between the columns of proliferating cells.[49] Terminal differentiation is

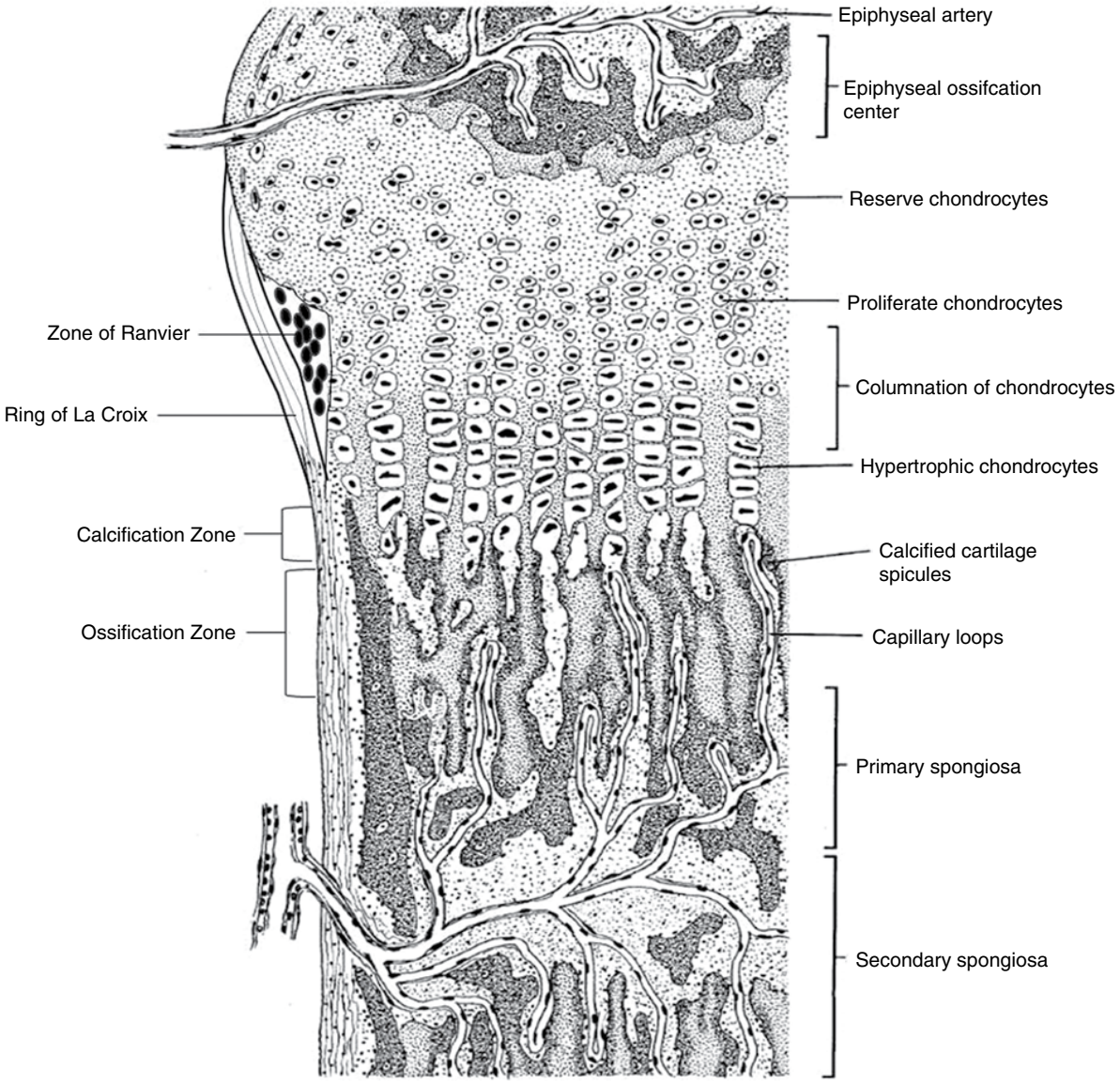


Figure 10.4. Cellular layers of the physis.

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inhibited in this zone by a morphogen produced in the resting zone.[1]

Hypertrophy Zone

After the chondrocytes finish cell division, they continue to mature and hypertrophy in the columns established in the proliferating zone. Matrix vesicles begin to form in this region. Matrix vesicles are microstructures that form in the extracellular matrix from the cytoplasmic membrane of the chondrocytes and contain products essential for calcification including alkaline phosphatase and calcium.[49] Type X collagen is also produced from the cells in this zone, which helps set up the matrix assembly for mineralization and helps increase calcium influx into matrix vesicles.[53] Within the zone, cells increase in size from the epiphyseal to metaphyseal side of the physis (Figure 10.4) due to the presence of factors such as insulin-like growth factor.[60] At the base of the hypertrophic zone, the size of the chondrocytes can be five times larger than those cells in the proliferative zone.[8] This increase in cell volume is what increases the rate of longitudinal growth of the physis.[7] The hypertrophied chondrocytes closest to the metaphysis have less metabolic activity, ultimately leading to cell degeneration and death.[9] Cell death triggers vascular invasion and calcification.[12,49] Because this zone is the transition zone between the cellular and bony region of the physis, combined with the relative lack of matrix surrounding the large chondrocytes, the zone of hypertrophy has been considered to be the structurally weak link of the physis.[4,39]

Zone of Calcification/Ossification

The zone of calcification is an ill-defined region that starts in the lowest region of the hypertrophic zone. The matrix between the cells gradually becomes mineralized due to deposition of hydroxyapatite crystals (Figure 10.4). This is thought to be initiated by the matrix vesicles, and it depends on several factors such as the availability of calcium and phosphate ions, cobalt and collagen, the pH, and the enzyme alkaline phosphatase.[49]

The zone of ossification is sometimes called the zone of angiogenesis because the terminal ends of the capillary sprouts impinge on the hypertrophic chondrocytes. Here osteogenic buds, which consist of capillary sprouts and osteoprogenitor cells, invade the columns of calcifying cartilage.[35,49] In the horse, there is approximately one capillary sprout per column of cells.[49] After the vessels come in, osteoblasts deposit osteoid, the organic part of bone, on the matrix surrounding the columns of calcified cartilage. This forms longitudinally orientated bony spicules (with a cartilaginous core inside) that make up the region called the primary spongiosa. Eventually the bone of the primary spongiosa is replaced by secondary spongiosa, which lacks remnants of the cartilage core.[55] As the bone elongates, bone at the diaphyseal end is removed by osteoclasts at the same rate that new bone is being formed on the epiphyseal side of the metaphysis. Thus, there is a continuous sequence of events, with cell division at one end, bone formation, and bone destruction, which is an important process that contributes to the final shape of the bone (Figure 10.4).[55]

Vascularity of the Physis

The blood supply to the growth plate originates from the epiphyseal, metaphyseal, and perichondral circulations.[4,38] Transphyseal vessels (those crossing the growth plate) are present in large epiphyses and may serve as a route for spread of infection from the metaphysis to the epiphysis. The metaphyseal circulation forms a series of loops that penetrate the longitudinal septa, enlarging as they return toward the diaphysis and forming a sinusoid. This produces a sluggish pattern of blood flow within the physis that predisposes this region to bacterial localization and osteomyelitis.[55]

The integrity of the blood vessels within the zone of Ranvier is important for continued appositional growth at the periphery of the epiphyseal growth plate. Disruption of the blood supply in this perichondral region can potentially cause ischemia to the physis, contributing to asynchronous growth and a subsequent angular limb deformity (ALD). In addition, lack of blood supply, particularly to the ossification zone, can prevent proper bone formation and potentially contribute to disruption of bone growth or osteochondrosis (OC).[11,40,41,66]

Biomechanical Aspects of the Physis

Although the exact mechanism is not completely understood, tension and compression on the physis are essential for continued orderly bone development and growth.[55] Each growth plate has a biologic range of both tension and compression within which it will respond. Within this range increasing tension or compression will accelerate physeal growth, while reducing tension or compression will decrease physeal growth. However, beyond the physiologic limits of tension or compression, physeal growth may be significantly decreased or even stopped; this is referred to as the Hueter–Volkmann law of physeal growth.[25,55] This law states that when mechanical compression is applied, growth will be retarded but that tensile forces will stimulate growth. This has an important practical application in the management of foals with ALDs. If we assume that a foal with an ALD of the carpus (carpus valgus) exerts an asymmetric load on the distal radial physis, then the physis will stimulate growth on the concave (tension) side of the physis and slow growth on the convex (compression) side of the physis, serving to straighten the limb without intervention. However, unrestrained exercise may cause physeal compression that is greater than the physiologic range and therefore prevent autocorrection of the ALD. In addition, foals with physitis or physeal dysplasia may have weaker metaphyseal bone than normal, which may be more susceptible to trauma.[4]

Cessation of Physeal Growth

As bone growth ceases, the physis becomes progressively thinner, and finally the epiphysis and metaphysis fuse.[38,39] The cartilaginous growth plate is replaced with

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trabecular bone, making it incapable of correcting an ALD. The timing of the physeal closure depends on the specific bone. Some close early in life and others remain open for several years (Table 10.1). However, it is important to note that functional (remaining growth potential) physeal closure occurs well before radiographic closure, and this difference has important bearing on the timing of surgery used to correct ALDs.[55] In general, the further distal on the limb the physis is located, the sooner longitudinal growth will become functionally inactive. For instance, the distal metacarpal/metatarsal physes close sooner compared with the distal radial or distal tibial physes (Table 10.1), making correction of ALDs of the fetlock more important early in life than those of the carpus or tarsus.[19] In addition, any injury to the physis such as excessive pressure, direct trauma, traction, circulatory loss, or shearing forces can lead to premature cessation of growth or asynchronous growth.[4,62] Comparatively, circumferential growth of the epiphysis continues uniformly regardless of anatomical location until the horse is fully mature.[30]

External Trauma to the Physis

When excessive force is applied to a joint and its nearby physis, an epiphyseal or physeal injury is likely to occur because the cartilaginous growth plate is weaker than the surrounding bone, ligamentous structures, and joint capsule. Physeal and epiphyseal injuries are common in foals.[16,17,42] Injuries that would normally produce a ruptured ligament or joint dislocation in an adult may produce traumatic separation of the physis in a young animal. For example, trauma to the fetlock region might cause a fetlock luxation in an adult horse, whereas a foal might sustain a distal metacarpal/tarsal physeal fracture.[55] This is partly why joint luxations appear to be rare in young horses.

CLASSIFICATION AND TREATMENT OF PHYSEAL INJURIES/FRACTURES

The most widely accepted classification of growth plate injuries is based on the Salter-Harris system, which separates the injuries into six specific types (Figure 10.5).[47] The system was designed for humans but has been applied to horses to permit equine clinicians to communicate effectively when describing such injuries.

Physeal fractures are relatively common in foals (accounting for approximately 20% of fractures) and may be considered more serious than diaphyseal fractures because of the risk of disturbed limb growth and articular involvement.[16,17,42] Type I and II physeal fractures are most common in horses. Frequent locations of physeal fractures include the distal metacarpus/metatarsus, distal and proximal radius, proximal humerus, proximal tibia, and distal and proximal femur (Figures 10.6 and 10.7).

These fractures tend to heal quickly but usually reduce the growth potential remaining in the physis via premature closure of the physis at time of injury or during fracture repair. This can lead to premature limb shortening or ALD. Certain locations (distal metacarpus/metatarsus and proximal tibia) have sparse soft tissue coverage and can lead to open fractures and increased risk of implant-associated infection with surgical repair. Concurrent injuries to the surrounding synovial, ligamentous, and tendinous structures can also influence overall prognosis. As with any fracture, the greater the degree of instability, displacement, and

Table 10.1. Range of growth plate closure in equine bone for forelimb and hindlimbs.

Forelimb			Hindlimb		
Growth plate	Age at closure (months)		Growth plate	Age at closure (months)	
	Earliest	Latest		Earliest	Latest
Cranial glenoid cavity of scapula[15]	5.0	5.0	Proximal femur/capital[54]	36.0	42.0
Supraglenoid tubercle and coracoid process[15]	12.0	24.0	Third trochanter[54]	24.0	48.0
Proximal humerus[15]	24.0	36.0	Distal femur[64]	24.0	30.0
Distal humerus[19]	13.6	14.9	Tibial tuberosity to tibial epiphysis[64]	9.0	12.0
Tuber olecranon[19]	26.6	29.7	Tibial tuberosity to tibial metaphysis[64]	30.0	36.0
Proximal radius[19]	13.6	14.0	Distal tibia[65]	17.0	24.0
Distal radius[19,56]	23.2	35.0	Malleolus[10]	3.0	3.0
Distal third metacarpus[19]	4.0	7.5	Tuber calcaneous[2,56]	16.0	27.0
First phalanx[19]	7.5	8.8	Distal third metatarsus[19]	4.0	7.5
Second phalanx[19]	7.5	7.9	First phalanx[19]	8.0	11.0

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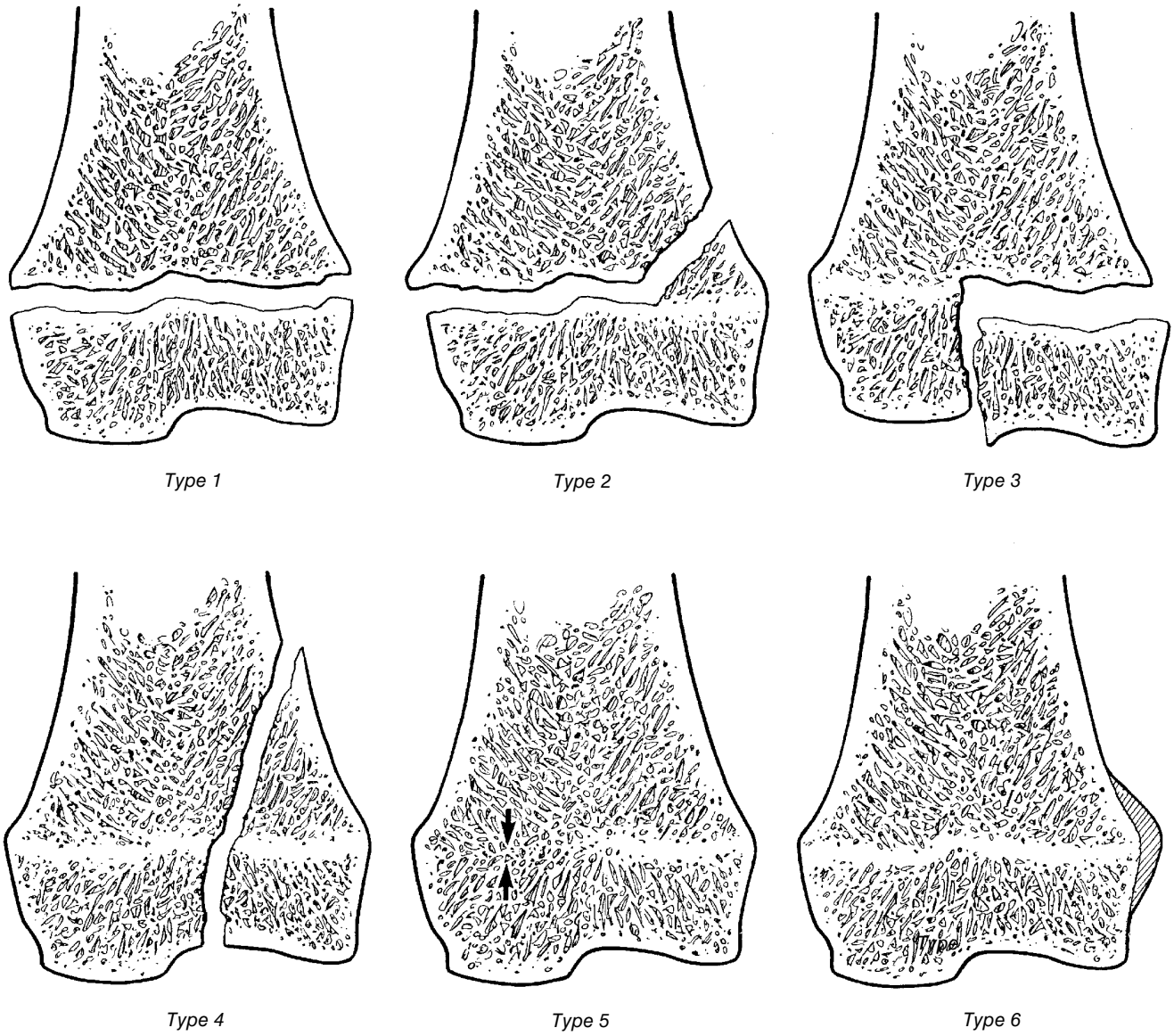


Figure 10.5. Salter–Harris classification of physeal injuries. The arrows in the type V injury indicate crushing of the physis. *Source*: Reprinted from Salter and Harris.[48] Reproduced with permission of Wolters Kluwer Health Inc.

duration until stabilization, the increased risk of damage and inadequate healing.[51] Although the prognosis can be very good, it has been shown that only 25% of foals with physeal fractures achieved complete soundness.[16,42]

Overall goals of treatment are to provide stability of the physeal fracture, allow weight-bearing function of the limb, and preserve growth plate function.[51] Stabilization can be tricky to achieve because often there is little bone to purchase, resulting in various methods of internal fixation that can be used and will differ based on individual fracture configuration (Salter–Harris subtype) and anatomical location.[33] Please see Chapters 4 and 5 for additional information on physeal fractures at specific locations on the limb.

Type I

In a type I injury, there is complete separation of the physis without fracture through the bone. This fracture usually occurs due to shearing forces across the physis causing the fracture to propagate through the zone of hypertrophy with the germinal cells of the growth plate remaining with the epiphysis (Figures 10.5 and 10.6).[39] With external trauma, this fracture configuration is common in the proximal femoral physis (slipped capital femoral epiphysis; Figure 10.6B), as well as the proximal radial physis or proximal humeral physis,[4,55] Salter-Harris type I fractures have been reported to occur as a pathological fracture secondary to severe physitis.[27] The prognosis for type I physeal fractures is generally poor.[27]

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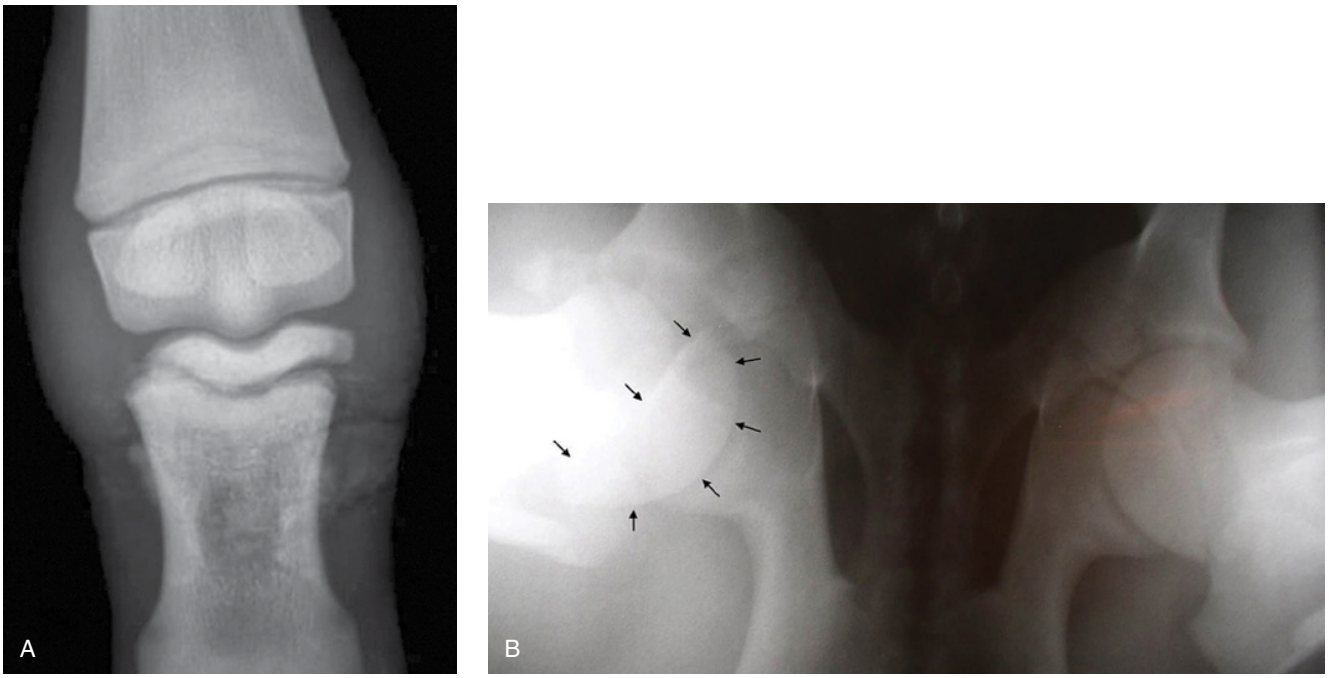


Figure 10.6. (A) Dorsopalmar radiograph of a foal with a type I fracture of the proximal first phalanx physis. (B) Ventral–dorsal radiograph of a foal's pelvis taken with the foal in dorsal recumbency while under anesthesia. There is a type I fracture of the proximal femoral physis. The arrows point out the location of the epiphysis; normal is on the right.

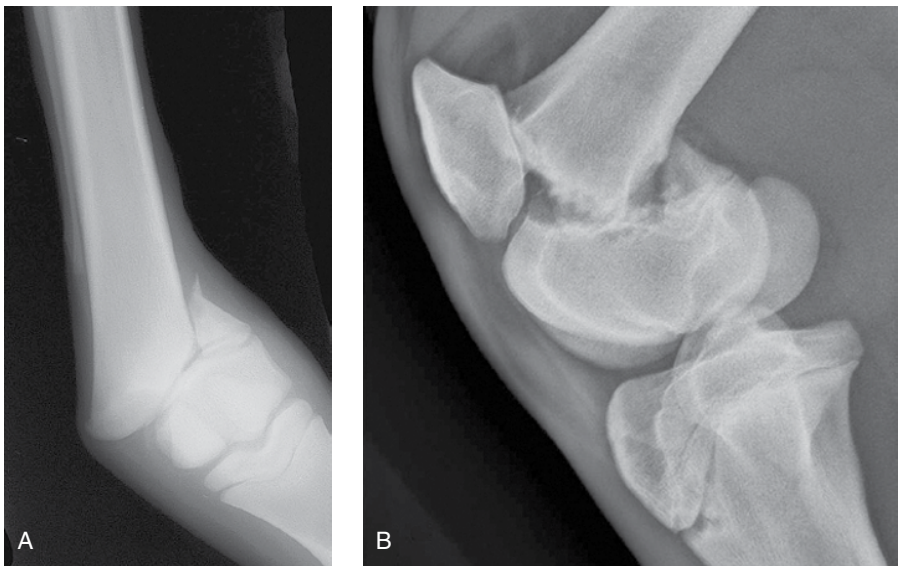


Figure 10.7. (A) Dorsoplantar radiograph of the distal metatarsus of a weanling with a type II physeal fracture. (B) Lateral radiograph of the stifle of a long weanling with a type II distal femoral physeal fracture. In most situations, these types of fractures should be repaired surgically to prevent malalignment of the limb.

Type II

Type II is the most common type of physeal injury in foals as well as in virtually all domestic animals.[55] The fracture line extends along the physis for a variable distance and then breaks out through a portion of the metaphysis, producing a triangular-shaped metaphyseal fragment (Figures 10.5 and 10.7). Similar to nearly all physeal injuries, the germinal cells remain within the physis. This type of injury is usually the result of shearing and bending forces. The distal third metacarpal/metatarsal physis commonly incurs this injury when the mare steps on the foal. The periosteum is torn on the convex side of the angulation but is intact on the concave side. Thus, the intact periosteal hinge is always on the side of the metaphyseal fragment.[47] A similar injury also occurs in the proximal tibial physis in slightly older foals. Closed reduction with casting alone may be possible in foals with metacarpal/metatarsal type II physeal injuries, but

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successful internal fixation with lag screws and casting has been described.[31] Internal fixation (medial bone plate and/or screw and wire transphyseal bridge) is usually mandatory for type II proximal tibial fractures.[42,51]

Type III

Type III fracture is intra-articular and extends from the joint surface, through the epiphysis, into the deep zone of the growth plate and then along the physis to its periphery (Figures 10.5 and 10.8).[46,47] This type of injury is uncommon in foals and is usually caused by an intra-articular shearing force or is secondary to infectious physitis. Open reduction and internal fixation are necessary to reconstruct the joint surface to prevent the development of secondary osteoarthritis (OA).

Type IV

Type IV fractures are intra-articular and extend from the joint surface through the epiphysis, across the entire thickness of the physeal plate, and through a portion of the metaphysis (Figure 10.5). This type of fracture is rare in foals. Open reduction and internal fixation are

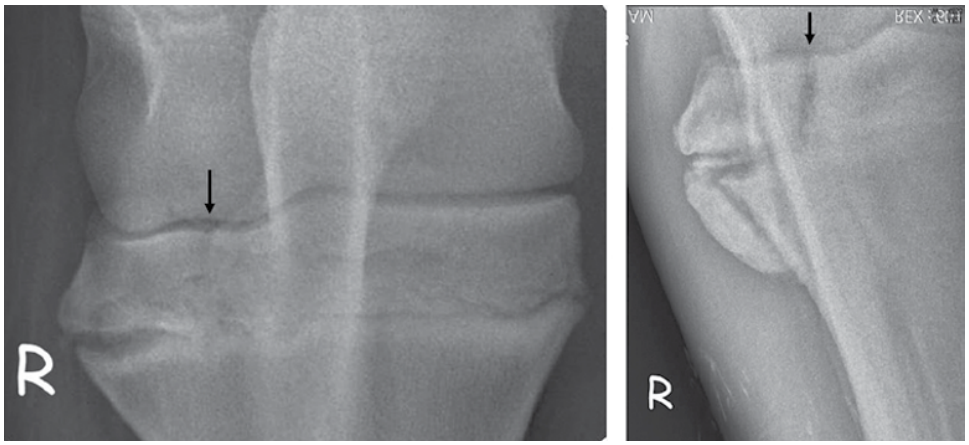


Figure 10.8. Craniocaudal radiographs of a 6-month-old horse at the time of fracture (A = day 0) and after stall rest (B = day 30). A Salter–Harris type III fracture is present that extends from the lateral articular surface (arrow) to the physis and out the lateral side of the physis. Note the extreme periosteal reaction present at day 30 with conservative treatment.



Figure 10.9. Typical swelling on the medial aspect of the distal radial physeal region (A) and radiographic changes associated with physitis (B). Arrows highlight areas of bone sclerosis and arrowhead highlights area of radiolucency at level of medial physis.

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necessary not only to restore a normal joint surface but also to obtain perfect apposition of the physeal plate. It has been observed in children that unless the fractured surfaces of the physeal plate are kept perfectly reduced, fracture healing occurs across the plate and renders further longitudinal growth impossible.[46,47]

Type V

Type V fracture is also an uncommon injury in foals. It results from a severe crushing force applied through the epiphysis to one region of the physeal plate (Figure 10.5). It may be associated with the distal third metacarpal/metatarsal physes in cases of severe varus deformity of the fetlock or the distal radial physis with severe carpal valgus. Excessive trauma to one part of the physis may be responsible for the severity of these deformities, although this is difficult to document.

Type VI

In a type VI injury, a periosteal bridge develops between the metaphysis and epiphysis (Figure 10.5). The new bone restrains growth on the affected side of the physis and has the same effect as a transphyseal staple or screws and wire. This type of injury may occur due to excessive trauma during placement or removal of staples or screws and wire, secondary to local infectious periostitis, or spontaneously from external trauma.[4,55] Removal of the periosteal bridge to help restore further growth in combination with transphyseal bridging on the opposite side of the physis is necessary to correct the problem. This approach has been reported to successfully resolve type VI lesions of the distal radial physis in two yearling horses.[21]

DEVELOPMENTAL ORTHOPEDIC DISEASES

The term developmental orthopedic disease (DOD) has historically been used to describe a comprehensive group of growth disturbances that occurs in foals and growing horses.[29,62] In recent years there has been an attempt to alter this nomenclature.[13,58] Denoix et al. proposed using the term juvenile osteochondral conditions (JOCC) to represent disorders related to the immature joint and growth plate.[13] The reasoning is that even though most joint and physeal lesions in young animals are due to orthopedic developmental issues, some are traumatic in origin based on the unique biomechanics of the developing skeleton.[13] Therefore, this terminology would involve pathology such as physitis/physeal dysplasia, ALD, OC, subchondral cystic lesions (SCL), incomplete ossification or collapse of the cuboidal bones, and juvenile arthritis. The old DOD term would still encompass all of those listed under the JOCC nomenclature, but would also include cervical vertebral malformations (CVM) and flexural deformities.[29,58] Van Weeren et al. may have summarized the nomenclature best by suggesting that the DOD term should be used for developmental disorders that relate to the entire animal, whereas JOCC should be used for pathology of articular structures in the juvenile animal.[58] Since this chapter incorporates all of the DOD conditions except for CVM, the term DOD will still be used throughout.

Regardless of the terminology used, most of these developmental conditions are attributed to alterations in bone growth or development at the metaphyseal or articular–epiphyseal growth plates. Clinical signs of these conditions vary depending on the specific disease and location, but are unique to growing horses.[29] In one survey of Thoroughbreds, 11% of young horses needed treatment for DOD conditions, and ALD and physeal dysplasia constituted 73% of treated cases.[37] Signs include variable degrees of lameness, alterations in posture or positioning of the limb, crooked legs, joint effusion, limb enlargement, and ataxia (CVM only). Although uncommon, multiple DOD conditions may develop in the same animal.

OC is a focal disturbance of endochondral ossification that results in a thickened area of articular cartilage. This may also present with fragmentation, referred to as a dissecans lesion or OC(D). The etiology is multifactorial, with no single factor accounting for all aspects of the disease.[66] The most commonly cited etiologic factors are heredity, rapid growth, anatomic conformation, trauma, and dietary imbalances; however, only heredity and anatomic conformation are well supported by the scientific literature.[49,57,66] Formation of fragile cartilage, failure of chondrocyte differentiation, subchondral bone necrosis, and failure of blood supply to the growth cartilage all have been proposed as the initial step in the pathogenesis.[41,66] High-circulating insulin levels from high-energy feeding has also been suggested to contribute to altered matrix metabolism and faulty mineralization.[28]

Although the exact cause for DOD is unknown, several risk factors predispose young horses to develop these diseases. The major risk factors appear to be nutritional imbalances, trauma, and genetic predisposition.[50] Nutritional imbalances include energy and phosphorus excesses; calcium, phosphorus, and copper (Cu) deficiencies; or any combination of these.[50] Feeding rations high in energy appears to predispose young animals to DOD abnormalities, presumably by contributing to rapid bone growth. However, in a recent study, weanling horses fed a high-starch diet had no increase in OC(D) lesions compared with those fed a medium-starch diet.[43] Defective collagen cross-linking from a deficiency in lysyl oxidase (an extracellular copper-dependent enzyme) may impair the strength of bone collagen, essentially producing a soft bone syndrome, particularly in the metaphyseal regions.[26,49] Trace mineral deficiencies, particularly copper, have been shown to cause clinical signs and joint pathology consistent with OC in foals.[26] However, copper supplementation of mares and foals had no significant effect on the frequency or severity of articular cartilage lesions in foals at 160 days of age.[22,44] The importance of copper deficiency in the pathogenesis of OC(D) is currently thought to be overemphasized.[22] In addition, trace mineral supplementation to yearling horses had no effect on growth and development, and no skeletal abnormalities were detected.[43] It has been suggested that it is likely most important to make sure the mare has adequate copper supplementation in late gestation to help protect the foal from joint pathology.[58]

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Trauma to the metaphyseal or epiphyseal growth plates may also contribute to altered growth, subchondral bone damage, avulsion of defective bone, and disruption of the physeal vasculature, all of which may predispose to DOD conditions. Weight, limb conformation, and excessive exercise may be contributing factors.[50, 57] Genetics most likely plays a role in the occurrence of these diseases, but its contribution is difficult to determine. OC is most likely a polygenic trait with a complex method of inheritance.[66] A genetic predisposition has been demonstrated in dogs, pigs, and horses.[49] Populations of risk alleles from two separate populations of OC(D) affected Standardbreds suggested that there is a true risk locus as a contributing etiology.[36] However, in most cases, the underlying cause of the DOD condition is multifactorial, usually obscure, and often never determined.[4,62] Factors that may have contributed to the disease process are often long gone by the time a veterinarian is asked to evaluate the horse. In addition, the timing at which risk factors may exert their effects on bone growth and development is currently unknown. Discussions of each specific DOD condition seen in growing horses are presented throughout this chapter.

EPIPHYSITIS/PHYSITIS/PHYSEAL DYSPLASIA

Physitis or epiphysitis is an important generalized bone disease of young growing horses characterized by inflammation and enlargement of the physeal region of certain immature long bones (Figure 10.9).[4,5] Most cases of physitis occur in young, rapidly growing horses such as weanlings, with a peak incidence between 4 and 8 months of age,[61] but even yearlings and 2-year-old horses may also develop the condition. It may affect a single or multiple growth plates, but is often bilaterally symmetrical.

Physeal dysplasia may be a more appropriate term because the condition is thought to be characterized by a disruption of endochondral ossification within the physeal growth cartilage.[5] However, in one study, physeal cartilage abnormalities and compromise of endochondral ossification were not frequently seen in Thoroughbred foals with visible bony enlargements of the distal metacarpus/metatarsus.[23] This questions the clinical significance of these physeal swellings and suggests that they may be physiological swellings associated with normal bone remodeling.[23]

ETIOLOGY

Although the exact etiology of physitis is unknown, it is most likely multifactorial and may differ from case to case.[4,5] For instance, in foals with multiple limb involvement, a nutritional problem affecting the entire animal seems most plausible. In contrast, physitis involving a single site is likely due to trauma or excessive compression of the affected physis.[4] Secondary ALDs tend to occur more frequently with trauma-induced physitis than from other causes. Foals with severe lameness in one limb may develop physitis and an ALD in the contralateral limb because of excessive weight-bearing (Figure 10.10).[3] However, in many cases, physitis appears to have a mechanical as well as a nutritional component. In young



Figure 10.10. Varus angular deformity of the left front fetlock in a foal that suffered a fracture on the opposite limb. Note the secondary physitis present with sclerosis (arrows) present in the distal medial metaphyseal region of the metacarpus.

foals, inflammation around the physis tends to occur when this newly forming bone is not able to withstand the load being placed on it.[5] This is usually because either the normally growing bone has too high of loads being placed on it as a result of being overfed for rapid growth,[4] exercise, weight, or conformation or because the bone itself is abnormal and cannot withstand normal loads.[5] The metaphysis provides much of the longitudinal growth in the foal and is therefore more prone to inflammation from repetitive loading than the epiphysis.[5] Regions of disturbed ossification within the physis from any number of these factors may predispose the underlying subchondral bone to microfractures.[61] These microfractures could lead to clinical signs of inflammation and potentially stimulate bone production and remodeling that is often seen radiographically as sclerosis in horses with physitis (Figures 10.9 and 10.10).[4,61]

The term physitis is often referred to as physeal compression, which further emphasizes the mechanical component of physitis.[55] When compression is applied to a physis, an increased thickening of the physis occurs due to retardation of provisional calcification and increased

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survival of chondrocytes.[4,55] This is an autocorrection phenomenon in which the animal corrects any minor angulation of the limb axis. However, loads on the physis are continually under challenge in the growing animal by the increase in both body weight and activity level.[6] If the compression is beyond the physiologic limits of the physis, complete arrest of endochondral ossification may occur. The end result is asynchronous physeal growth and the development of an ALD together with physitis. This is most commonly seen at the distal radial physis in horses with carpal varus (Figure 10.11).

Septic, or infectious, physitis is a separate etiology that may present similar to traditional aseptic physitis depending on severity. Septic physitis is common in younger neonatal foals (often 1 week to 4 months of age) and occurs secondary to hematogenous sepsis and/or failure of passive transfer. The abundant metaphyseal, physeal, and epiphyseal vasculature combined with slower perfusion can create an ideal environment for infection to establish. Inflammation and osteolysis of the surrounding epiphyseal/metaphyseal trabecular bone adjacent to the physis may ensue (Figure 10.12).[6,24]



Figure 10.11. Physitis of the medial aspect of the distal radius (arrow) in a 12-month-old colt, which occurred after being kicked in the area. A carpal varus deformity developed after the trauma.

CLINICAL SIGNS

The clinical signs in young foals are variable but usually include a stiff gait with a shortened cranial phase to the stride, increased amount of time lying down, and trembling when standing, potentially buckling forward at the carpus or fetlock (Figure 10.13).[5,59,61,63] In general, physitis occurs more commonly in distal versus proximal physes, as distal growth plates have less soft tissue support and are subject to increased biomechanical forces due to the long axis of the limb creating a lever-arm stress.[6] Therefore, the most common locations of physitis are the distal radius, tibia, and third metacarpal/metatarsal bones. It is usually visually apparent as an enlarged physeal region due to the metaphyseal flaring that develops secondary to physitis (Figures 10.9, 10.11, and 10.14). The enlargement is often painful to deep palpation and can therefore result in a progressive increase in muscle tension that can lead to contraction of the flexor tendons.[5,59,61]. In the case where the cause of the physitis is due to the level of exercise or the amount of weight, the physitis is symmetrical (e.g. involves medial and lateral aspect of physis), whereas for those that are due to conformation or cartilage retention, the physitis is asymmetrical (e.g. involves only part of the physis, either medial or lateral).[5]

Physitis of the distal aspect of the cannon bones often involves all four limbs (Figure 10.13), whereas the distal aspect of the radius and tibia is usually not involved concurrently.[4] In addition, foals with metacarpal/metatarsal physitis are usually younger than foals with physitis in other locations. This may be related to the activity of the physes with respect to bone growth at varying ages. Septic physitis usually occurs in young foals that are susceptible to other hematogenous infections and show similar signs with more pain on standing and deep palpation. In severe cases of physitis, secondary Salter-Harris (type I or II) fractures (Figure 10.7B) can occur.[27]

DIAGNOSIS

Radiographs help classify the severity of physitis. Early physitis, related to a transient maladaptation to increased body weight or exercise levels, will have a normal radiographic physis.[6] With chronicity, characteristic radiographic changes develop. The most common radiographic abnormality observed with physitis is paraphyseal bone production, often termed physeal lipping or metaphyseal flaring (Figure 10.14).[5] Increased radiolucency or widening of the physis, asymmetry of the metaphysis, wedging of the epiphysis, metaphyseal sclerosis adjacent to the physis, and asymmetry of cortical thickness due to altered stress on the limb can also commonly be observed (Figures 10.9, 10.10, 10.13, and 10.14).[4,61]

Radiographic abnormalities may be disseminated across the entire physis or be focal within the physis.[6] Concurrent ALDs or OC lesions also may be present. In cases of septic physitis, cystic osteomyelitis of the periphyseal trabecular bone can develop (Figure 10.12).[6,24] In addition, MRI has been shown to be a useful tool for identifying lesions that occur in the epiphysis, metaphysis, and physis with a higher sensitivity than radiographs.[20] MRI can help provide a definitive diagnosis for earlier aggressive treatment (Figure 10.15).

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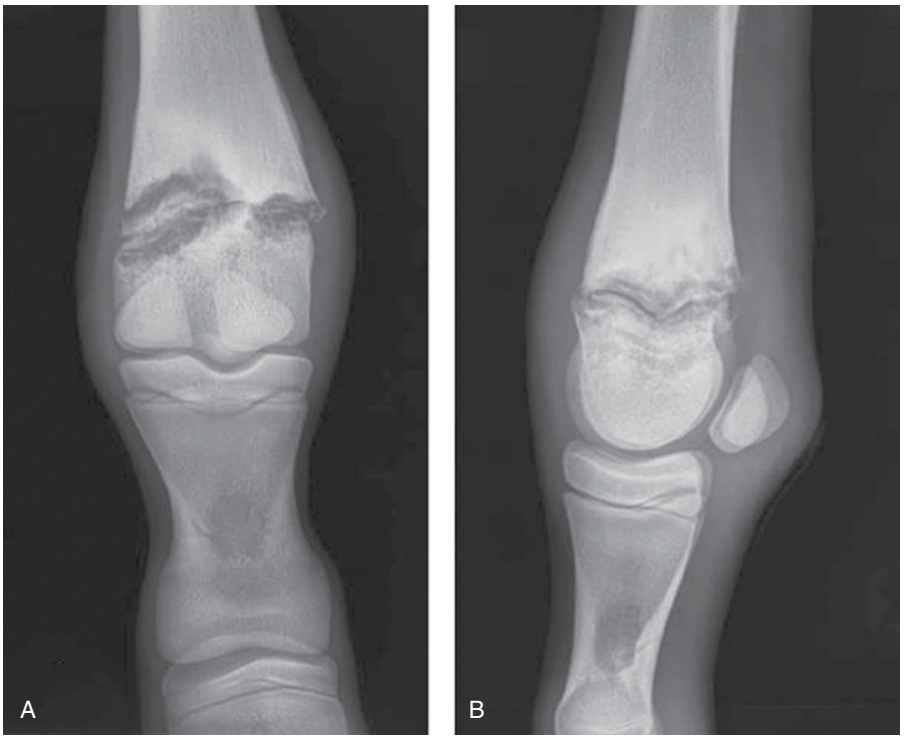


Figure 10.12. Dorsoplantar (A) and lateromedial (B) radiographs of the fetlock in a 2-week-old foal illustrating radiolucency at the distal metatarsal physis secondary to severe septic osteomyelitis.



Figure 10.13. (A) Weanling colt demonstrating typical knuckling of the fetlocks secondary to the pain associated with bilateral distal metatarsal physitis. (B) Lateral-to-medial radiograph showing typical bone sclerosis (arrows) that occurs in the metaphyseal region of horses affected bilaterally or in all four legs.

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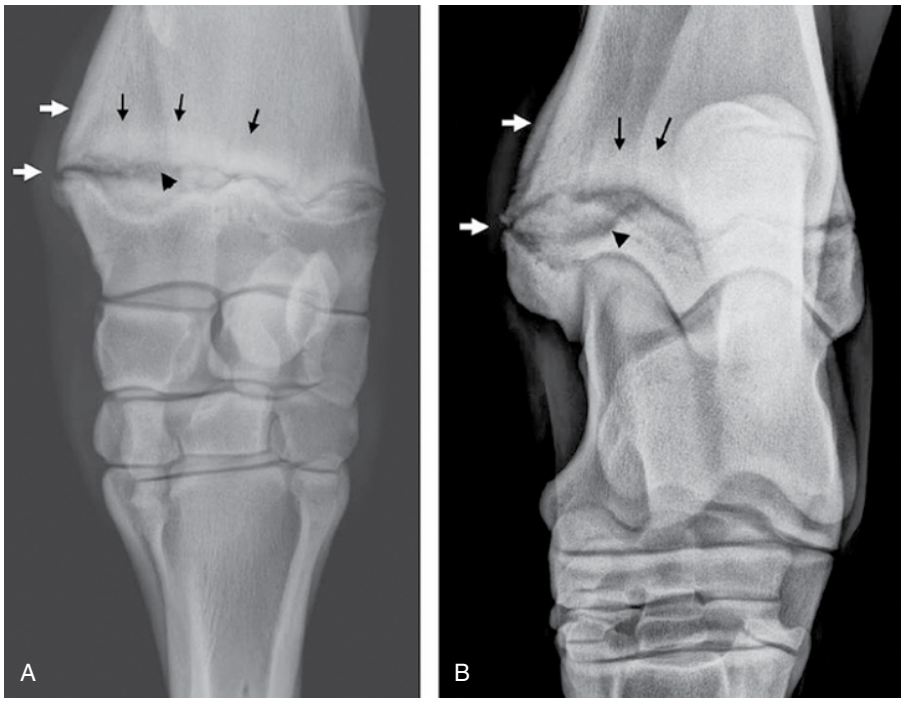


Figure 10.14. Craniocaudal radiographs of the carpus (A) and tarsus (B) of weanlings demonstrating paraphyseal lipping and metaphyseal flaring (white arrows) as well as metaphyseal sclerosis (black arrows) and radiolucency at the level of the medial physis (black arrowhead) causing widening of the physis.

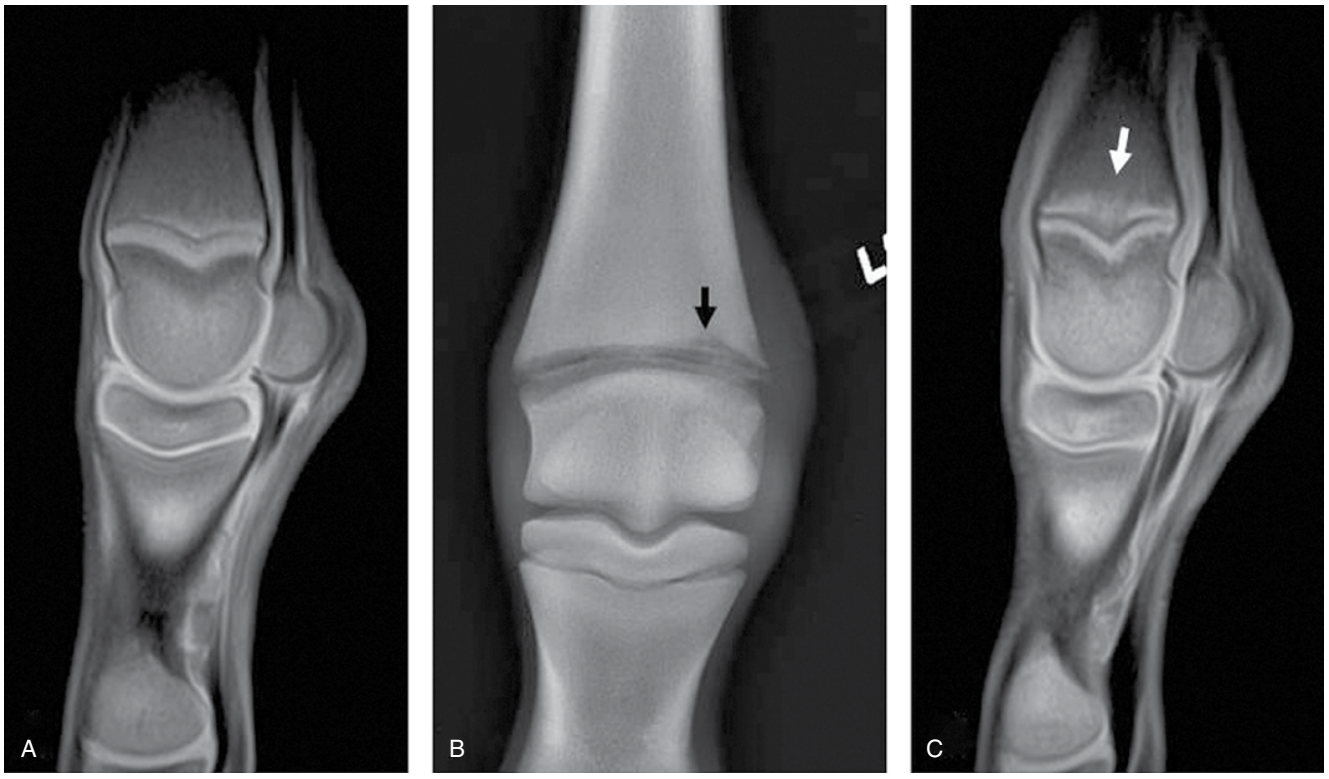


Figure 10.15. Proton density sequences (1.5T) of sagittal slices through the medial (A) and lateral (C) aspects of the metacarpophalangeal joint shown in the dorsopalmar radiograph (B) of an 8-day-old colt with early signs of osteomyelitis in the lateral metaphysis (arrows). Notice the difference between the medial (A) and lateral (B) aspects of the distal metaphysis of MCIII.

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TREATMENT

Treatment of physitis in young foals needs to incorporate treatment of the symptoms (e.g. pain) but also needs to treat the source. For most, nonsteroidal anti-inflammatory drugs (NSAIDs) are indicated to decrease physeal inflammation and improve the animal's stiff gait. NSAIDs help diminish pain and may prevent the development of flexural deformities. NSAIDS are often required for at least 2–3 weeks to completely resolve the inflammation.[6] Monitoring for gastrointestinal and/or renal complications is advisable. Phenylbutazone is a common formulation used for musculoskeletal disorders.[14] Alternatively, COX-2 selective formulations (such as firocoxib or meloxicam) may be preferable due to less risk of adverse systemic effects.[14]

If the source of the physitis is obvious, then it needs to be treated appropriately. For example, if an ALD is present, the greatest degree of physeal abnormalities usually corresponds to the direction in which the limb is deviated. For example, weanlings with significant physitis of the medial aspect of the distal radial physis often develop carpal varus (Figure 10.11). This suggests that the physitis has contributed to arrested growth on the medial aspect of the physis. Autocorrection of these deviations is unlikely, and growth retardation surgery on the opposite side of the physis is often necessary to correct the ALD.

Most of the time, the exact source of the physitis not easily identified, but the amount of exercise can be adjusted since both the intensity and duration of exercise influence physeal stress. If the foal is getting too much exercise, it should be reduced, but not eliminated to prevent further trauma to the physis.[5] This can be accomplished via confinement or by changing the exercise routine such that smaller segments of exercise are delivered multiple times per day instead of one continuous period of exercise, especially in foals and weanlings.[6] If a foal spends the first few weeks of its life recumbent, the bone needs to adapt to the loads placed on it when the foal is standing. If the exercise level increases dramatically in this time frame, adaptation of the bone will not occur quickly enough, resulting in some degree of clinical physitis.[5] Therefore, careful control of exercise is important in these particular foals. In the case of an unstable physis secondary to trauma, some protection with external coaptation may be required. However, completely protecting the physis from weight-bearing forces, as could be achieved with a cast, is contraindicated unless absolutely required, as complete disuse will lead to further weakening of the growth plate.[6]

The feed ration should always be evaluated, especially if it involves multiple locations. Often a geographic nutritional deficiency may exist, especially when multiple animals are affected. The ration should be altered accordingly, and many times it is advised to reduce the animal's body weight or growth rate. However, retardation of growth rate by feeding poor-quality hay is considered irresponsible by many.[32] The recommended approach is a carefully formulated diet that specifically restricts starch and protein while supplying National Research Council (NRC) minimum requirements of other essential nutrients.[32] In general, affected horses should be fed less grain and fewer protein supplements, and the quantity of alfalfa hay should be reduced or replaced with good-quality grass hay.[4] With nursing foals, the milk production of the mare should be decreased if possible. Specific balanced rations for growing horses to minimize the development of physitis and other DOD diseases have been developed and can be considered.

In cases of septic physitis, early recognition and aggressive management with systemic and local (intravenous or intraosseous) antimicrobials are required. In select cases, debridement of osteolytic, necrotic periphyseal bone is required. Premature closure of the growth plate with subsequent ALD or limb shortening may be complicating sequela.[6,24]

PROGNOSIS

Fortunately, the majority of physitis cases are mild and are often self-limiting. Most cases resolve when skeletal maturity is reached and growth of the affected physis ceases. In many cases, a mild amount of physitis may be part of the normal aspects of bone modeling and remodeling; as such, foals simply grow out of the problem.[5,6,23] More severe cases of physitis, such as those that develop concurrent angular or flexural limb deformities or those resultant from sepsis, may cause residual problems severe enough to limit future athletic soundness of the horse.[5,6]

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ANGULAR LIMB DEFORMITIES (ALDS) AND CUBOIDAL BONE MALFORMATIONS

Nicolas S. Ernst, Troy N. Trumble, and Gary M. Baxter

ANGULAR LIMB DEFORMITIES (ALDS)

Angular limb deformities (ALDs) are defined as conformational deviations in the frontal plane. These are axial deviations that are based on the relationship of the limb distal to a particular joint. Valgus and varus are the terms used to describe ALDs in foals. Valgus indicates an outward shift of the limb distal to the joint of origin, while varus indicates an inward shift of the distal limb. For instance, carpal valgus refers to a deformity in which the carpus is the site of the lesion and the limb distal to this joint (third metacarpal bone) is deviated away from the midline of the body (Figure 10.16).

A large number of foals examined during the first month of life can present with limb deviations, which will decrease on prevalence and severity with increasing age and exercise. Carpal valgus is the most common ALD seen in foals, followed by fetlock varus, carpal varus, and tarsal valgus.[4,5,15,18,19,22] Tarsal varus is rare, as are deviations of other parts of the limbs.[4,13,20] Foals with ALDs due to asynchronous physeal growth typically are not lame and have few physical examination abnormalities other than crooked legs. If lameness is present, physeal trauma, fracture, collapse, or sepsis of the cuboidal bones should be suspected as the cause of the limb deformity (Figure 10.17).[6]



Figure 10.16. Young filly with bilateral carpal valgus.

ETIOLOGY

Foals can be born with ALDs or can develop them over time. Proposed causes in foals born with ALDs include malpositioning *in utero*, *in utero* chemical insults such as locoweed, and hormonal or nutritional imbalances.[6,21,24,32] However, most ALDs occur over time with 73.2% of acquired deviations appearing between 3 months of age and weaning age, irrespective of the joint.[25] Acquired causes of ALDs in foals include laxity of periarticular supporting tissues, incomplete ossification of the tarsal or carpal cuboidal bones, direct trauma to the physis as with concussion or fractures, traumatic luxation or fracture of the carpal bones (Figure 10.17), and asynchronous longitudinal growth of the metaphysis and epiphysis (Figures 10.10 and 10.16).[4,6,22,32] By far the most common reason for ALDs in foals is asynchronous metaphyseal growth. For often unexplained reasons, one side of the growth plate grows faster than the other, resulting in a deviation of the normal limb axis.[18] Trauma across the growth plate is believed to be one of the main underlying causes of asynchronous growth.[18] Trauma may retard calcification of the matured and dying chondrocytes, resulting in thickening of the physeal plate with an increasing number of chondrocytes at the hypertrophied cell layer.[6] If compression is excessive, chondrocytes may undergo necrosis and the physis may prematurely close or growth is slowed. Any factor that causes asymmetric loading across a growth plate can lead to this sequence of events, resulting in an ALD.[6,15] Such factors include joint laxity, hypoplasia of the cuboidal bones, poor foot trimming, heavy muscling, excessively active foals, or lameness in the opposite limb (i.e. foals develop support limb ALDs instead of laminitis).[4,6,18]

CLINICAL SIGNS

Laxity of Periarticular Support Tissues

Foals with ligamentous laxity are often termed windswept because their limbs are very flaccid and tend to deviate in the same direction. The carpus or tarsus is usually affected, and one limb has a valgus deviation and the

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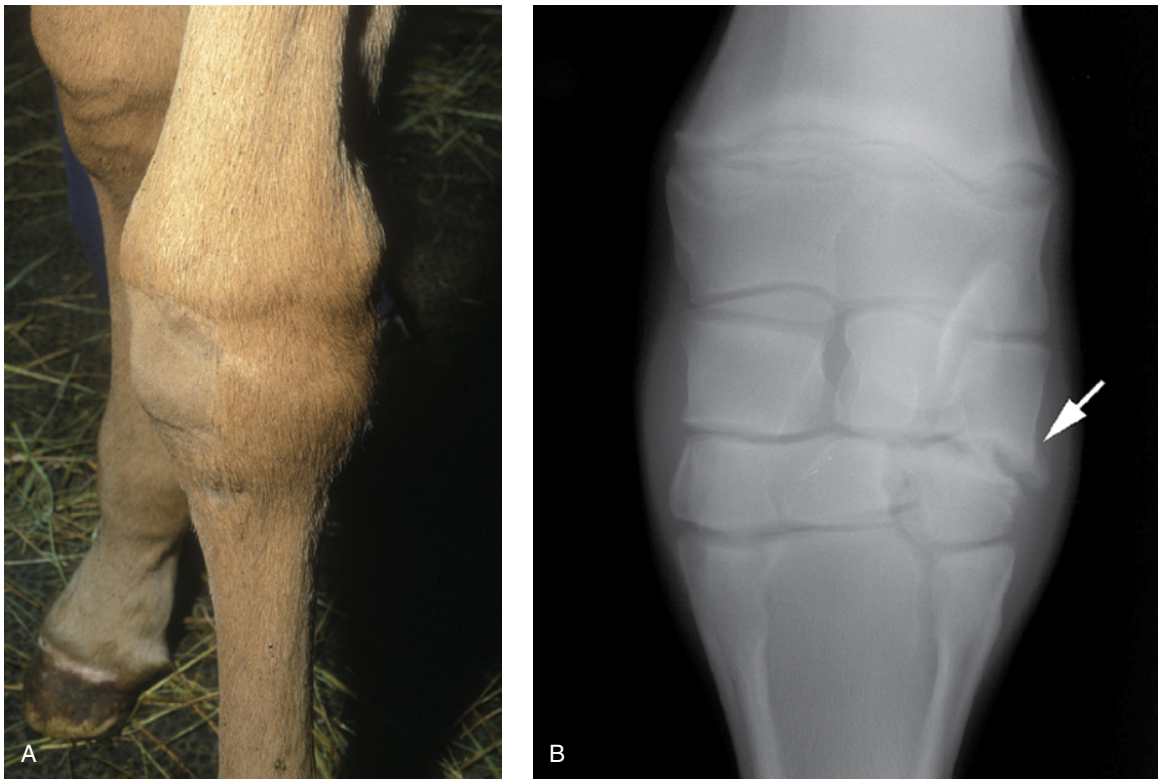


Figure 10.17. Young horse with a traumatic injury to the carpus, contributing to a carpal valgus. (A) Physical examination of the carpus revealed effusion of the middle carpal joint and pain on flexion. (B) Dorsopalmar radiograph of the carpus, which revealed a fracture of the ulna carpal bone (arrow) and collapse of the lateral aspect of the middle carpal joint.

other has a varus deformity (Figure 10.18). Usually the limbs can be manually straightened, indicating a soft tissue or cuboidal bone ossification problem and not a deviation of the bony column. Affected foals are usually within 2 weeks of birth, may be premature, and usually are not lame. At the walk, the affected joint may deviate inward or outward when weight is placed on the limb (hence the term spaghetti legs) due to the weakness of the supporting soft tissue.

Delayed Ossification of the Tarsal or Carpal Cuboidal Bones

Affected foals are usually born premature or dysmature and may have concurrent ligamentous laxity.[24] The limbs are often straight at birth and then begin to deviate within a few days or weeks due to collapsing of the cuboidal bones (Figure 10.19). Valgus deviation of the carpus and tarsus occurs most commonly.

Physeal or Articular Trauma/Fracture

The majority of foals with physeal or articular trauma/fracture have no history of limb deformities until after a traumatic incident. They may have a single limb involved, are usually lame, and often have joint effusion (Figure 10.17). Lameness and pain on manipulation of the affected joint are the most important clinical findings to suggest trauma as the cause of the ALD. Radiographs are essential to make a definitive diagnosis.

Septic Physitis of Cuboidal Bones

The physis within the cuboidal bones can become septic in young foals. This is most likely from a hematogenous route, but can also be from trauma. Any physis or cuboidal bone can be affected, but most foals only have a single limb involved. The foals are usually significantly lame and have joint effusion or pain localized to the physis. Sepsis can cause significant lysis of the physis or cuboidal bones leading to collapse that ultimately contributes to an ALD (Figure 10.20).

Asynchronous Longitudinal Growth

Foals with asynchronous physeal growth appear clinically normal except for their crooked legs. No palpable abnormalities should be present and the limbs cannot be straightened manually. Varying degrees of outward rotation of the limb often accompany foals with carpal valgus (Figure 10.16).[6,32] Multiple types of ALD may be present in the same foal, especially if it has not been treated. Carpal valgus and fetlock varus may occur concurrently, and the treatment of one deformity most likely affects the other (Figure 10.21).

DIAGNOSIS

Foals with ALD of almost any joint can usually be diagnosed by visual exam. When examining foals for ALDs, it is important to note that correct conformation

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Figure 10.18. Young foal with a windswept appearance to the tarsi due to ligamentous laxity.



Figure 10.19. Radiograph of a newborn foal that has a carpal valgus deformity due to delayed cuboidal bone ossification.

for the foal is not the same as correct conformation for the adult.[2,3,16,26] Foals will undergo changes during growth and maturation that can rapidly change the conformation. In general, examination of the foal for ALDs should start as close to birth as possible, then every week until 1 month of age as this is a critical time in which many changes can occur in response to growth and exercise demands. Examinations should then be continued on a monthly basis until 6 months of age. A complete exam entails examination of the limb of interest in a standing and a flexed position, followed by exercise and radiographs. When examining the limb standing, rotation of the limb can make interpretation of the degree of ALDs more difficult, especially those distal to the fetlock.

Rotation of the limb or torsion around the weight-bearing axis of the limb is often associated and coupled with ALDs. Therefore, when examining a limb for ALDs, it is very important to be directly lined up in front of the limb and not just in front of the foal (Figure 10.22). Being directly lined up in front of the limb will give a more accurate assessment of the ALD, especially when there is a rotational component to the limb as well. Most foals have some degree of external (toed-out) rotation that will generally improve with age as the chest broadens and pushes the elbow outward.



Figure 10.20. Septic cuboidal bones (black arrows) in a 1-month-old colt.

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Figure 10.21. Foal with a combination of carpal valgus and fetlock varus. Straightening the carpal valgus would probably make the fetlock varus worse and must be considered when developing a treatment plan.

Elevating or flexing the limb will often help alleviate some of the perceptual problems that can be encountered from rotation of the limb. Flexion of the limb around the joint of interest allows the examiner to compare the relative position of the distal limb to proximal limb by putting them in closer proximity to each other. In addition, it helps to distinguish some of the more complicated cases in which there are ALDs involving both the carpus and fetlock.[19] Picking up and manipulating the limb in a medial-to-lateral direction also allows the examiner to determine whether the limb can be manually straightened or not. If the limb can be manually straightened, it is highly likely that the deformity is centered within the joint and results from delayed ossification of the cuboidal bones or laxity of the periarticular soft tissues. If the limb cannot be straightened, the deformity likely originates at the physeal region. Watching the foal walk is often very useful in distinguishing how much of the ALD is a component of ligamentous laxity. Ligamentous laxity will tend to show more exaggerated movements around the joints of interest, making the ALD more pronounced. In these foals, it is important to make note of the age and exercise level of the foal to know what the best treatment for correcting the laxity so that a better evaluation of the ALD can be made. For example, neonates are often very lax and require more exercise, whereas older foals that have exercised rigorously need to be stall confined for a few hours for the laxity to improve.[4]

If foals are examined on a regular basis, radiographs are not necessarily required unless radical changes have occurred between examinations. However, if it is the first time the foal is examined for ALDs, especially in older foals, radiographs provide the most conclusive evidence about the location and potential cause of the ALD. For ALDs, only dorsopalmar and lateral radiographic views are required unless there is evidence of problems within the joint, and then oblique views should be added. The radiographs should be centered over the joint of interest and include the middiaphysis of the proximal and distal long bones. These views allow identification of the "pivot point" of the ALD by drawing lines down the middle of the long bones on the dorsopalmar view.[6,24,32] Where the lines bisect is where the deformity is thought to originate (Figure 5.4). In the case of asynchronous growth across a distal physis, the deviation arises from the distal metaphyseal region rather than within the joint (Figure 10.23A). Wedging of the epiphysis on the concave side of the deviation, abnormal contour of the cuboidal bones, and displacement of the splint bones often accompany limb deviations of the carpus and tarsus but usually resolve when the limb straightens (Figure 10.23B).[6,22] Epiphyseal wedging is especially prominent with varus deformities of the fetlock (Figures 10.10 and 10.24), and collapse of the lateral aspect of the third carpal bone is common with carpal valgus deformities (Figure 10.23B). However, severe abnormalities such as physeal or cuboidal bone fractures or crushing of the carpal or tarsal bones in conjunction with an ALD are a major concern and may contribute to permanent lameness problems, even if the deviation is corrected.

TREATMENT

Treatment depends on the cause, location, and severity of the ALD, as well as the age of the foal. In general, varus deformities appear to be more problematic for future soundness than valgus deformities. Varus deformities of the fetlock (pigeon-toed) are thought to contribute to pastern and fetlock lameness problems, whereas mild carpal valgus was found to be protective for carpal injuries in racing Thoroughbreds.[3] Most foals have a mild degree of carpal valgus conformation (approximately 5°) that tends to decrease as they grow older.[16,28] Foals tend to grow into the mild carpal valgus as the chest expands during growth.[16,32] If the limb is completely straight as a foal, a carpal varus may develop as the chest expands during growth. In contrast, fetlock varus deviations tend to worsen with age up to 6 months.[28] Therefore, mild carpal valgus deformities should not be treated, whereas minor varus deformities of the fetlock probably should be addressed because of the potential to worsen with age.

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Figure 10.22. Images of the right forelimb of a weanling as viewed from directly in front of the horse (A) and lined up with the externally rotated hoof wall (B). Note that the carpal valgus looks similar in both images, but the fetlock looks different (slightly valgus in A and varus in B). It is important to line up with each limb since many limbs are externally rotated in foals.

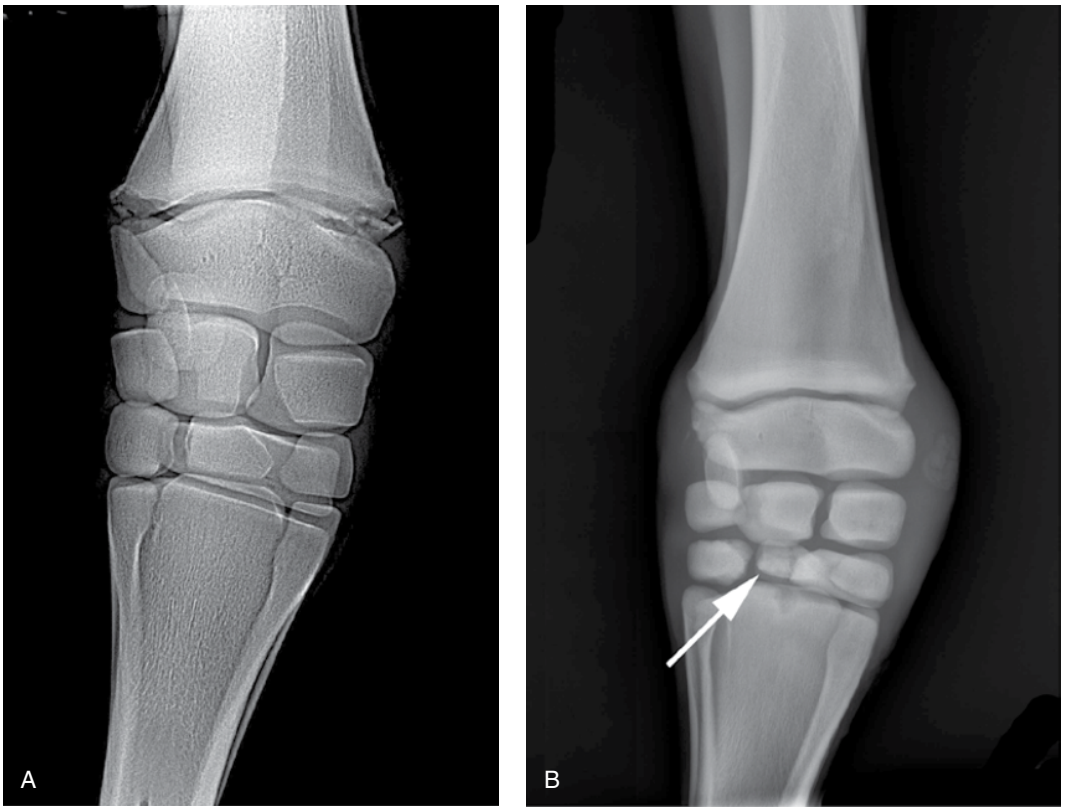


Figure 10.23. Dorsopalmar radiographs of two foals with mild carpal valgus without abnormalities of the carpal bones (A) and with wedging of the lateral aspect of the third carpal bone (arrow) (B).

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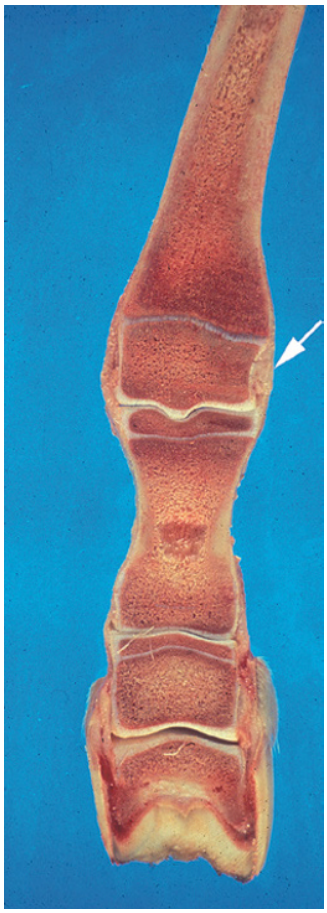


Figure 10.24. Cross section of a fetlock varus deformity in a young foal with collapse of the medial aspect of the epiphysis (arrow).

Conservative Treatment of Asynchronous Physeal Growth

Conservative treatment of asynchronous physeal growth usually involves a combination of time, exercise restriction, and foot care in foals with mild to moderate ALDs. Under normal circumstances, most foals autocorrect their deformity because the bone grows faster on the concave side and slower on the convex side (Wolff's law).[14,17,32] This only occurs under physiologic loads, so the goal of confinement is to minimize overloading of the physis. Overactivity may cause or perpetuate physeal trauma and excessive loading, possibly preventing the limb(s) from autocorrecting. Restricting the foal's exercise by confining the mare and foal to a stall and run or small paddock/pasture is usually adequate. Confinement is usually continued until the limb has straightened.

Foot trimming (or balancing) also may minimize compressive forces on the physis. In foals with varus deformities, the hoof does not wear as much on the medial side and becomes compressed on the lateral side. The opposite occurs with valgus deformities. The goal of trimming is to remove the hoof wall that is overgrown (trim the hoof on the side that the leg goes; inside hoof for varus deformities and outside hoof for valgus deformities) to make the foot flat or balanced.[24,32] This should be done with only a rasp to avoid excessive trimming. Glue-on shoes (Dalric, Advanced Equine Products, Versailles, KY) with lateral or medial extensions also may be helpful to minimize asymmetrical loading of the physis and pull the limb inward or outward. The extensions are placed on the opposite side of where the deformity goes (lateral extension for varus deformities and medial extensions for valgus deformities; Figure 10.18). Frequent reevaluations (e.g. every 2 weeks) with or without radiographic examination should be performed to monitor the progress of conservative therapy.

Confinement and minor foot trimming can correct many foals with mild to moderate ALDs and should always be considered as the initial therapy if possible. However, aggressive and prolonged trimming may lead to concurrent angular deformities elsewhere in the limb that may worsen the overall condition of the foal. For example, a secondary varus deformity of the fetlock may develop in some foals with carpal valgus treated conservatively.[4] Conservative management is usually not recommended or successful with a severe deformity of any physis in any age foal. The asymmetrical loading of the physis is usually beyond the physiologic limits in these cases, making autocorrection unlikely.[17,32] In addition, a severe ALD has a greater likelihood of causing permanent damage to the tarsal or carpal cuboidal bones if not corrected early in life.

Surgical Treatment of Asynchronous Physeal Growth

Surgical treatment of foals with ALDs is directed toward accelerating growth on the concave side or slowing growth on the convex side.[4,5,15,18,19,32] Hemicircumferential transection of the periosteum and periosteal stripping (HCTP+PS) is used to stimulate growth, and various forms of transphyseal bridging (TPB) are used to retard growth.

Physeal growth has been reported to occur by stimulation of the physis with fan-shaped stab incisions and placement of needles directly into the physis.[11] These techniques were reported as another method to treat ALDs and as an alternative to HCTP+PS.[11] The exact mechanism contributing to physeal growth stimulation is unknown but is thought to be a mechanical release of the periosteal restraint of growth.[4,5] However, there is controversy regarding the true benefit of HCTP+PS. In practices where foals can be examined on a consistent basis, clinicians found that most correct on their own, with only changes to the exercise regimen.[29] This has been confirmed by two studies. A controlled model of ALD demonstrated no benefit of HCTP+PS compared with controls treated with confinement and hoof trimming.[25] Another study in nine foals with naturally occurring carpal valgus deviation reported that correction of carpal ALD was not significantly corrected by HCTP+PS.[31] These studies and observations have questioned whether HCTP+PS is a necessary surgical procedure in foals with ALDs. However, many veterinarians remain convinced that it is beneficial because foals with ALDs treated conservatively improve after HCTP+PS is performed.[4,11,18]

TPB is effective and is currently the most reliable surgical technique to correct limb deformities. However,

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the advantages of HCTP + PS over TPB techniques are that the procedure is easy to perform, has minimal complications, and is less expensive than TPB procedures and the limb does not overcorrect. In addition, the majority of horses can perform well after surgery, although racing performance has been shown to be reduced if HCTP+PS is performed at two or more anatomic locations.[23] Despite these benefits, HCTP+PS appears to be most useful for mild to moderate cases of ALD that have not responded to conservative treatment. Foals with severe ALD at any location are best treated with TPB.

Retardation of endochondral ossification on the convex side of the deformity can be accomplished with various TPB procedures.[10,13,18,20,22,33] The bridges may consist of staples,[10,18] screws and wires,[4,22] a small plate spanning the growth plate,[18] or a transphyseal lag or positional screw placed across one side of the physis[20,33] (Figure 10.25). A positional screw is currently recommended to avoid screw breakage and difficulties in screw removal. The transphyseal screw technique is especially useful in the tarsus because of the ease of screw placement (the epiphysis is narrow and angled), but can also be used primarily in the fetlock (Figure 10.25B).[20,29,33]

The implants create a static compression across one side of the physis so that bone growth is retarded.[17,32] Slowed growth on one side of the physis and continued growth on the opposite equalize the relative length of the medial and lateral aspects of the distal metaphysis, thereby straightening the limb. Once the limb is straight, the implants must be removed or the limb will over correct. Limb growth usually returns to normal following implant removal. However, continued growth retardation has been seen following removal of the transphyseal screw, and this should be considered when deciding when and where to use this technique. Other considerations for a transphyseal screw are the potential for the development of a physitis that results in metaphyseal collapse (creates an angular deformity in the opposite direction) when used for carpal ALDs.[9] Due to this and the fact that use of a transphyseal screw increased the risk of physitis that required further treatment, surgeons should carefully weigh the pros and cons of using a single screw in the carpus.[9] The advantages of the TPB procedures are that severe deformities can be corrected quickly and most surgeons feel that the results are more reliable than HCTP + PS. The disadvantages include that the procedure is more difficult to perform, there are more wound healing complications (can cause a cosmetic blemish), a second surgical procedure is needed to remove the implants, and the limb can overcorrect if the owners fail to monitor the limb or the implants damage the physis. For these reasons, most surgeons utilize TPB in severe deformities that will not correct with HCTP + PS alone or in older foals/weanlings where the growth potential of the physis may be limited.

All improvements should be made within the time frame for maximal growth for each particular growth plate so that the bone can still respond. The maximal

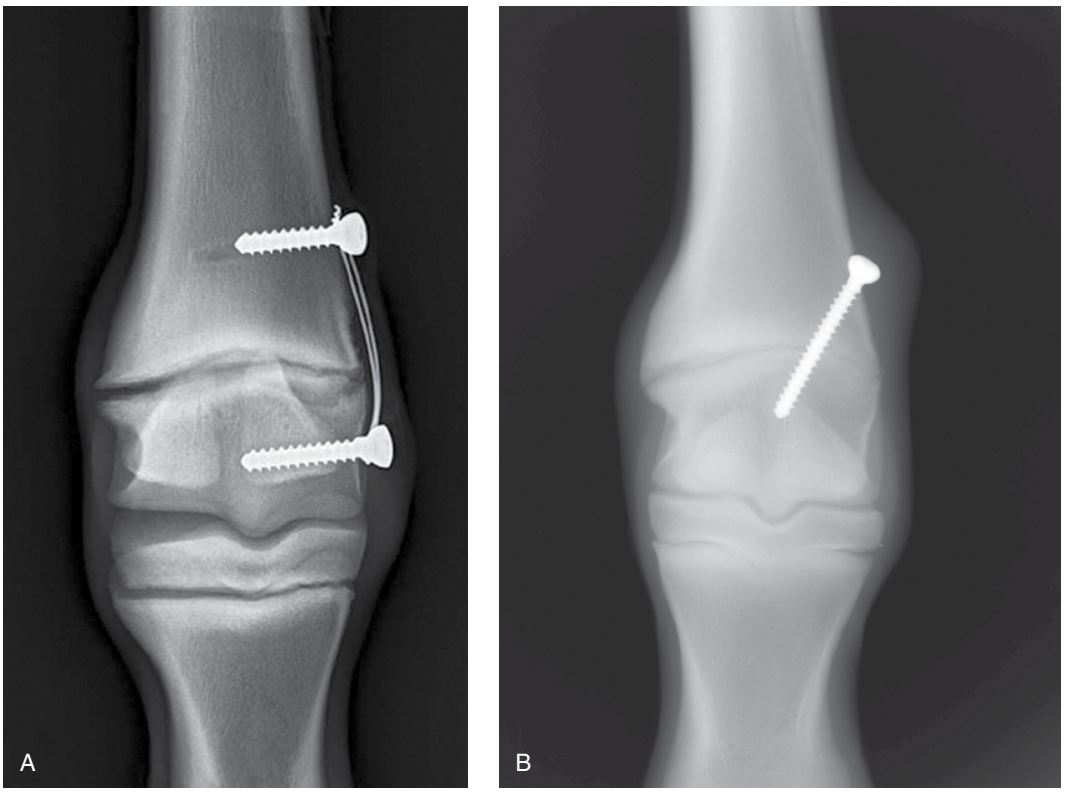


Figure 10.25. Dorsopalmar radiographs demonstrating transphyseal bridging of the distal physis of the third metacarpal bone using (A) screws and wire and (B) a transphyseal screw.

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growth in the distal physes is over after 2 months for the metacarpal/metatarsal bones, 4 months for the tibia, and 6 months for the carpus.[4] In most cases, ALDs of the fetlock or phalanges should be treated surgically within 45–60 days of age, or sooner if they are worsening.[4,32] Hunt recommends that HCTP+PS be performed by 1–2 months and TPB by 3–4 months to achieve correction of ALDs at the fetlock.[18] However, some minor limb deviations of the fetlock may be corrected with TPB after 4–6 months of age. The distal radial and tibial physes close at a later age, and therefore conservative therapy can be continued longer (up to 4–5 months). However, if the deformity is severe (more than 15°) or if limb deviation worsens with time, then surgery should be performed earlier.

In most cases of carpal and tarsal deformities, surgery is indicated if the limbs have not corrected by 4–5 months of age.[18,20,33] Carpal valgus deformities of less than 10° in foals less than 4–5 months of age may benefit from HCTP+PS alone.[4,5,18]

The HCTP + PS technique should be performed by 45 days of age to correct fetlock deformities and in one study was most beneficial for tarsal valgus deformities if performed by 60 days of age.[13] TPB techniques are usually recommended for carpal deformities greater than 10°, especially in foals older than 4–5 months, and for most fetlock deformities in foals older than 2 months of age. The transphyseal screw is the recommended TPB technique for the tarsus because it is much easier to place than staples or screws and wires. The mean age of foals in two different studies with fetlock, tarsal, or carpal deformities that were treated with transphyseal screws was 94, 220, and 377 days, respectively.[20,33] These average ages were greater than what is currently recommended for TPB techniques, but several older horses were treated in these studies, which contributed to the greater mean ages. Depending on the severity of the deformity and to ensure correction, the upper age limit for using TPB techniques should be approximately 6 months for the tarsus and carpus and 3 months for the fetlock. However, horses older than these upper limits have been treated successfully with TPB techniques, depending on the severity. TPB can be performed successfully up to 14–16 months of age in the carpus, up to 12 months of age in the tarsus, and up to 7 months of age in the fetlock.[18,33]

Many veterinarians' philosophy about managing foals with ALDs appears to have changed over the last decade. Mild carpal valgus is considered normal, and there is less desire to obtain perfectly straight front limbs at an early age. Furthermore, more foals are being treated conservatively and for longer periods of time before surgery is performed. It also appears that correction of ALDs can be accomplished with TPB techniques in foals and weanlings older than what has been previously thought. All of these findings have led to a more conservative approach to manage foals with ALDs, relying more on normal bone remodeling and growth for correction. However, severe deformities at any location and varus deformities should still be considered problematic, less responsive to conservative treatment, and often an indication for surgical intervention.

PROGNOSIS

Foals with mild and moderate ALDs of any region usually have an excellent prognosis for correction and future athletic soundness. In general, varus deformities appear to be more problematic for future soundness than valgus deformities. As such, even mild varus deviations (1°–8° deviation in the distal aspect of radius) are usually treated surgically with a favorable outcome for sales and racing performance.[30] Varus deformities of the fetlock (pigeon-toed) are thought to contribute to pastern and fetlock lameness problems, whereas mild carpal valgus may be protective for carpal injuries. Severe ALDs have the potential to cause asymmetrical loading of the carpal and tarsal cuboidal bones, contributing to collapse, articular cartilage damage, and future lameness. Early correction of severe ALDs usually helps prevent these problems and improves the long-term prognosis in affected foals.

CUBOIDAL BONE MALFORMATION/INCOMPLETE OSSIFICATION

Incomplete ossification of the cuboidal bones of the carpus or tarsus occurs most commonly in premature, twin, or underdeveloped newborn foals.[6,32] At birth, the cuboidal bones have not ossified sufficiently to withstand the forces of normal weight-bearing, predisposing to variable degrees of carpal or tarsal bone wedging or collapse.[12] In one study of foals with tarsal valgus deformities, 56% had concurrent incomplete ossification of the tarsal cuboidal bones.[13] Incomplete ossification without collapse is not readily apparent clinically in young foals and may go unrecognized until the horse has matured. These horses often develop osteoarthritis (OA) of the distal tarsal joints at an early age and become lame when put into work.[7,8] This condition has been termed juvenile arthritis or spavin.

ETIOLOGY

Cuboidal bone ossification normally occurs during the last 2–3 weeks of gestation.[32] Foals born prematurely or immature may simply have a delay in the normal ossification of the cuboidal bones (Figure 10.26). Others foals with incomplete ossification may have similar congenital or developmental abnormalities that are associated with ALDs and OCD. For example, juvenile arthritis is thought to occur because of a defect in endochondral ossification of the cuboidal bones, similar to OCD. The articular–epiphyseal physes are affected with OCD and the spherical physes of the tarsal cuboidal bones with juvenile arthritis. Cuboidal bones can become crushed or collapse over time with normal biomechanical forces if there is incomplete ossification present. This can result in ALDs and/or accelerate the development of juvenile arthritis.

CLINICAL SIGNS

Clinical signs associated with delayed cuboidal bone ossification may be evident in the newborn foal as an ALD of the carpus or tarsus (Figure 10.19). Concurrent

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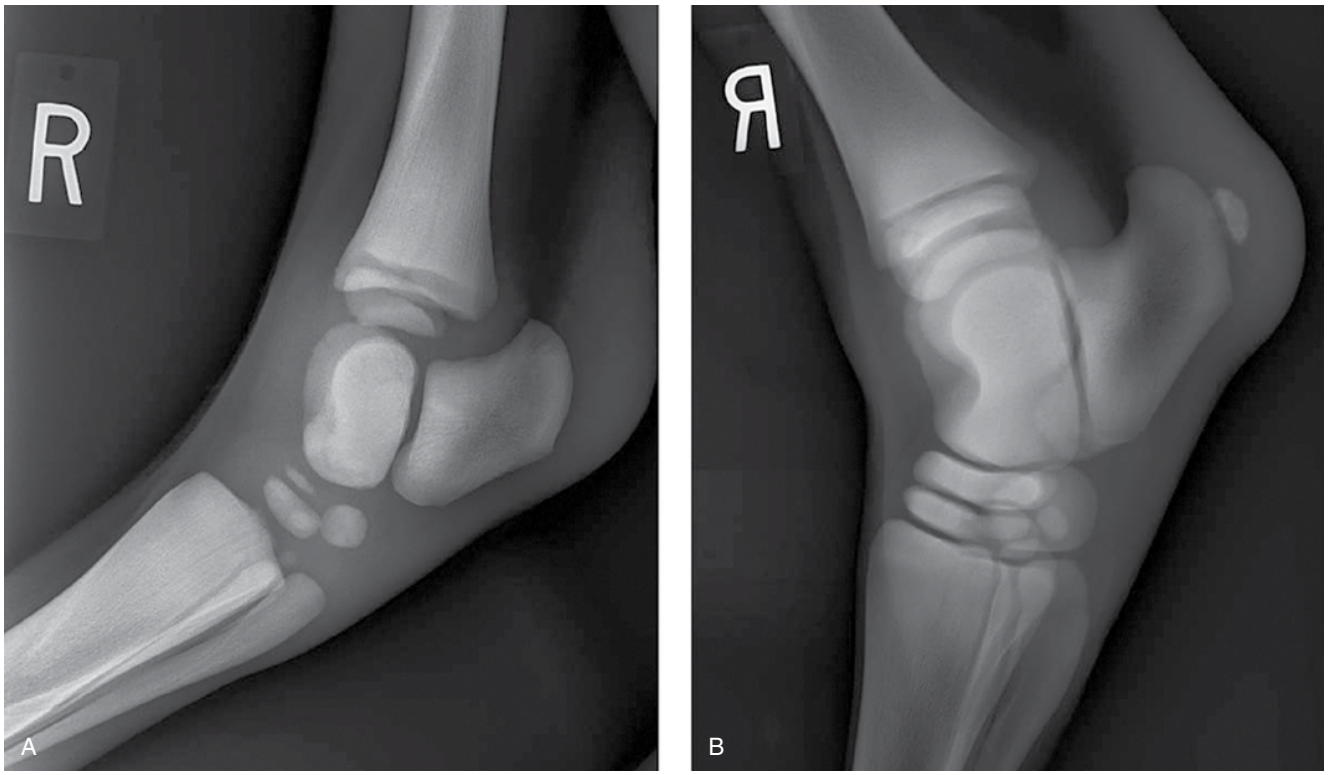


Figure 10.26. Radiographs of the tarsus of two newborn foals on their first day of life. (A) Premature foal. (B) Foal with a normal gestation period.

laxity of soft tissue structures is often present.[24] The limb can usually be straightened manually but often bows inward or outward during weight-bearing. It is usually bilaterally symmetrical, and both the carpi and tarsi may be affected. When collapse or crushing occurs, this tends to affect the tarsal bones more commonly than the carpal bones (Figure 10.27).[12,13] Tarsal collapse is often associated with a sickle or cow-hocked conformation because the tarsus is often deviated cranially (reduced angle to the hock).[1] The tarsus may appear like it has a curb. There is usually no lameness initially, but lameness is inevitable if the cuboidal bone collapse went unrecognized and was not clinically apparent until later in life (Figure 10.27).

Incomplete ossification and mild collapse of the tarsal bones are also thought to predispose to juvenile spavin in young horses (Figure 10.28).[6–8] These horses tend to develop hind lameness and moderate to severe radiographic signs of distal tarsal OA at a young age without a history of exercise.[6] Incongruencies or minor malformations of the central and third tarsal bones are thought to contribute to the development of OA at such an early age (Figure 10.28B).[8] For unknown reasons, the distal intertarsal joint appears to be affected most commonly in horses with juvenile arthritis, with changes in the tarsometatarsal joint being more subtle (Figure 10.28A).

DIAGNOSIS

The diagnosis is based on the history, characteristic limb conformation, and radiographs indicating incomplete ossification of the cuboidal bones. Radiographically, incomplete ossification is observed as wide joint spaces with small, rounded cuboidal bones, with or without compression or crushing (Figures 10.26 and 10.27).[24] Serial radiographs should be performed on a weekly basis to monitor the progression of ossification (Figure 10.2). Ultrasound has also been reported to help diagnose and monitor progression of ossification of the cuboidal bones.[27] In the tarsus, collapse is usually located on the cranial aspect of the distal tarsus, contributing to the cranial deviation of the limb.[6,12] The lesions in the tarsus have been described as incomplete ossification with less than 30% collapse of the affected bones (type I) and more than 30% collapse with pinching or fragmentation of the affected bones (type II).[12] Type II lesions are more common and are associated with a worse outcome than type I lesions (Figure 10.27).[12] In addition, a skeletal ossification index has been proposed to classify osseous immaturity in the carpal bones, but it is not used clinically by the authors.[1]

Milder forms of incomplete ossification may not become clinically apparent until later in life. However, young horses (under 2 years of age) with hindlimb lameness localized to the distal tarsus should be suspected of having malformation/incongruency of the tarsal cuboidal bones (Figure 10.28).[8] Some of these horses also may have a tarsal valgus or sickle-hocked tarsal conformation. The diagnosis is confirmed by radiographs, which may indicate mild cuboidal bone collapse, wedging, or malformation, and signs of OA.[7,8]

TREATMENT

Treatment depends on the age of the foal, severity of the incomplete ossification, and the limbs affected. If diagnosed early, the goal of treatment is to prevent cuboi-

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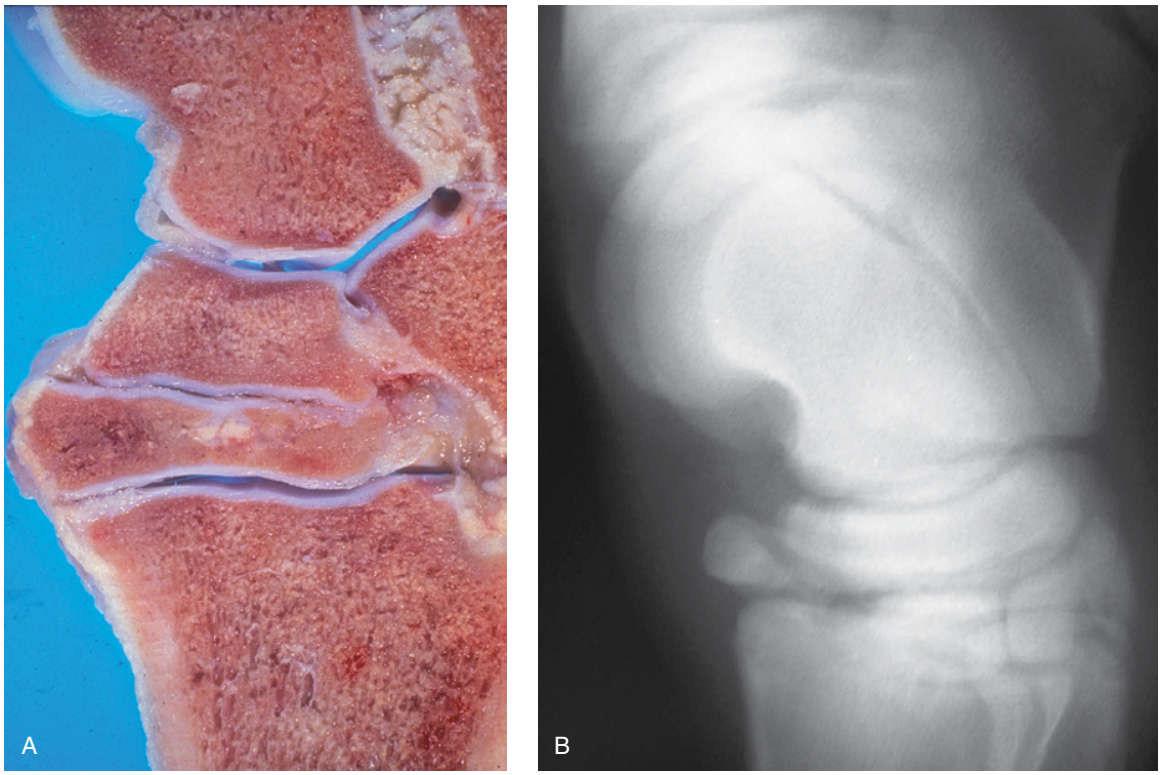


Figure 10.27. (A) Cross section of the tarsus in a young horse with partial collapse of the third tarsal bone. (B) Lateral radiograph of 4-month filly with lameness due to complete collapse of the third tarsal bone.

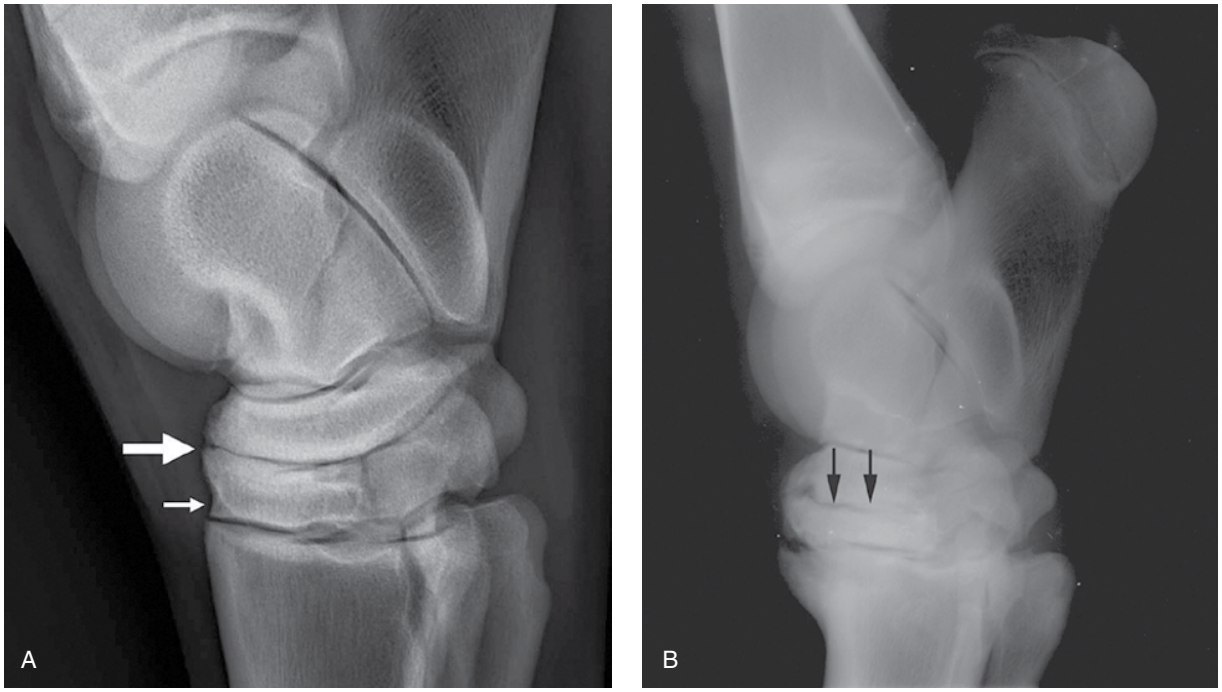


Figure 10.28. (A) Lateral radiograph of the tarsus in an 8-month-old yearling with hindlimb lameness. Subchondral bone lysis of the distal intertarsal joint (large arrow) and osteophytosis formation in the tarsometatarsal joint (small arrow) can be seen. (B) Lateral radiograph of a 2-year-old demonstrating an undulating distal intertarsal joint surface with narrowing of the third tarsal bone (black arrows) and severe osteophyte formation consistent with distal tarsal OA.

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dal bone collapse and maintain axial alignment until bones are fully ossified. This usually involves minimizing compressive (weight-bearing) forces on the bones until they have time to ossify completely. Confinement, sleeve casts, bandages alone, bandage casts, or bandages and splints may be used, depending on the severity.[6,24,32] However, bilateral sleeve casts or bandages can be difficult to manage in newborn foals, and the foals may have difficulty getting up and down. The decision whether to use external coaptation is made on a case-by-case basis. In addition, it is important to remember that the more rigid the coaptation used, the more lax the ligamentous structures will become in newborn foals. Therefore, the type of coaptation and amount of exercise should be reassessed on a weekly basis via the use of serial radiographs to monitor the progression of ossification. If cuboidal bone collapse has already occurred, any form of treatment is unlikely to be of benefit and abnormalities within the joints are inevitable.

PROGNOSIS

Foals that can be managed successfully to prevent cuboidal bone collapse can do well. Foals with only minor tarsal bone collapse (type I) were able to perform as intended, whereas foals with more severe tarsal bone collapse and fragmentation (type II) could not be used for their intended purposes.[12] No study has looked at the prognosis of foals with carpal bone collapse, but it is logical to assume that these foals have a more guarded prognosis than those with tarsal bone collapse.

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FLEXURAL DEFORMITIES

Nicolas S. Ernst, Troy N. Trumble, and Gary M. Baxter

Flexural deformities, or contracted tendons, of limbs of young horses are classified as congenital (apparent at or near the time of birth) or acquired (develop during the growth period).[2,23] Limb deformities secondary to trauma also may be considered as acquired flexural deformities.[13] The term contracted tendons is often used synonymously with flexural deformities, but the potential for contraction of dense tendinous tissue is limited and the primary defect is usually not in the tendon itself.[23] For this reason, as well as the fact that other structures such as the suspensory ligaments, distal sesamoidean ligaments, joint capsule, and fascia can be involved,[18] the contracted tendon terminology should be obsolete. The mechanism for development is not well understood,[10] but in many instances the result is an effective functional length of the musculotendinous unit(s) that is less than what is necessary for normal limb alignment. These abnormalities are most accurately discussed relative to the joint(s) around which the deformity is centered rather than the probable affected tendons and ligaments because more than one structure may be involved.

CONGENITAL FLEXURAL DEFORMITIES

Etiology

Several factors have been incriminated as causes of congenital flexural deformities in neonates. This condition primarily has been attributed to uterine malpositioning or overcrowding, but more complex influences have been implicated, such as genetic factors, equine goiter, and teratogenic insults from toxic and infectious agents during the embryonic stage of pregnancy or from malnutrition of the mare.[10,12,23,25] Arthrogryposis, as a cause of congenital flexural deformities, has been well documented in calves but is less well defined in horses.[23]

Clinical Signs

Generally, congenital flexural deformities may affect one or more limbs with the superficial and deep digital flexor musculotendinous units being most commonly affected. Therefore, the carpus and/or fetlock cannot be completely straightened. The carpus is most commonly affected. The fetlock can also be affected with the carpus or on its own (Figure 10.29). With fetlock flexural deformities the foals may be able to stand but knuckle over at the fetlock. In newborns, the fetlock flexural deformity may be combined with laxity at the level of the distal interphalangeal (DIP) joint (Figure 10.30); if the flexural deformity of the fetlock is not corrected, subluxation of the DIP can occur.[12] In severe instances, the foals walk on the dorsal surface of the fetlock. Involvement of the deep digital flexor tendon (DDFT) alone may manifest as a flexural deformity of the DIP joint. Congenital flexural deformities of the pastern and tarsus occur, but much less frequently.[10,23] Tarsal flexural deformities are rarely seen because the deformity often causes a dystocia, leading to stillbirth, but, when present, are often combined with multiple other congenital deformities (Figure 10.31).

Rupture of the common digital extensor (CDE) tendon may occur concurrently with carpal or fetlock flexural deformities (Figure 10.32).[23,38] Occasionally, flexural deformities may not be noted despite evidence of a ruptured extensor tendon. The extensor tendon rupture may occur secondary to flexor tendon contracture, or it may predispose to the flexural deformity. Affected foals may have other associated birth defects including prognathism, underdeveloped pectoral muscles, and incomplete ossification of the carpal/tarsal bones.[23]

Diagnosis

The diagnosis is usually based on the history of being born with the problem and physical examination of the foal. Affected limb(s) cannot be straightened manually, and the foal may have difficulty standing. It is important to try to get the foal to stand, if possible, as the deformity is usually not as bad as it seems when trying to straighten the limb while they are recumbent (Figure 10.33). In more mild cases it may be possible to palpate which tissues tighten while trying to extend the limb, but this becomes increasingly difficult in more severe cases and those with contracture of the carpal fascia and/or palmar ligaments. This contributes to an arthrogryposis-like position of the carpus with minimal flexion or extension capabilities (Figure 10.29A). Radiographs or ultrasound can be performed but are usually not necessary for the diagnosis. Radiographs can be useful though for identifying any underlying abnormality of the cuboidal bones when the carpus is affected.[12] Radiographs should be considered more often in the distal limb since fetlock flexural deformities can lead to subluxation of the DIP joint (Figure 10.30B). In addition, rare pastern flexural deformities are hard to diagnose early in life without the benefit of radiographs.

Concurrent extensor tendon rupture is characterized by the presence of unilateral or bilateral swelling over the dorsolateral aspect of the carpus (Figures 5.8 and 10.32).[38] It may or may not be possible to palpate the enlarged ends of the ruptured tendon. Commonly, the ruptured CDE tendon is not recognized, and the problem is diagnosed as a carpal flexural deformity.

Treatment

Medical treatment usually consists of NSAIDs and IV oxytetracycline and is often combined with bandaging and splinting. Treatment of congenital flexural deformities should be initiated soon after recognition of the problem, with the severity of the deformity dictating how quickly and aggressively treatment should proceed.[10,31] Foals with minor congenital flexural deformities (particularly of the

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Figure 10.29. (A) Congenital flexural deformities of both carpi and front fetlocks. Neither limb could be straightened manually and the foal had difficulty walking with splinting. (B) More typical flexural deformities of both carpi that cause the carpus to knuckle forward.



Figure 10.30. (A) Visual appearance and (B) lateral radiograph of the left hind distal limb in a 3-week premature foal demonstrating a flexural deformity at the level of the fetlock combined with laxity at the level of the distal interphalangeal joint.

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Figure 10.31. Foal with a tarsal and fetlock flexural deformity of the left hindlimb as well as bilateral carpal and forelimb fetlock flexural deformities.



Figure 10.32. Fluid swelling at the lateral aspect of the left carpus (arrow) in a 3-day-old foal that is characteristic of a rupture of the common digital extensor tendon. This condition can be unilateral as in this foal or bilateral.

carpus) may improve spontaneously and do not require treatment (Figures 10.29B and 5.6). In addition, most fetlock flexural deformities usually respond well to bandaging and splinting.[2,10,23] DIP joint deformities may respond to glue-on toe extensions that stretch the DDFT and also prevent excessive wear of the toe. However, these should be used with caution because they may produce excessive tension on the laminae.[10,27]

While NSAID treatment in young foals is not ideal, it can often be necessary to help offset the pain that is produced from the additional therapies such as splinting. It is important to have the foals be comfortable enough to stand as they will improve quicker than if then remain recumbent due to pain (Figure 10.33). Please refer to Chapter 8 for more information on NSAID therapy. Generally, it is recommended to use gastroprotectants while foals are being administered NSAIDs.

Oxytetracycline remains a common treatment for foals with congenital flexural deformities.[1,2,10,19,23] Clinically, it appears to be more effective on congenital than acquired flexural deformities, but can be used for both types. The use of oxytetracycline was first described in 1985 by Lokai and again in 1992 by the same author.[19,20] All foals in the latter study (n = 123) were treated either once or twice with 3 g of oxytetracycline intravenously (undiluted), and no external splinting was used. A 94% overall success rate was reported with the forelimbs responding better (98%) than the hindlimbs (75%).[19] Oxytetracycline's mechanism of action has been hypothesized to result from chelation of calcium in muscle or to decrease the expression of the contractile protein α-smooth muscle actin.[14,19] A recent study found that myofibroblasts made up most of the cells in the distal check ligament and the DDFT in normal foals, supporting this rationale.[14] Oxytetracycline was also found to inhibit normal collagen organization in equine myofibroblasts through a matrix metalloproteinase-1 mechanism.[5] However it works, oxytetracycline appears to be an effective method to obtain a short-term moderate decrease in metacarpophalangeal (MCP) joint angle in newborn foals. Two controlled studies have documented changes in MCP joint angles following treatment with oxytetracycline.[16,21] A significant decrease in mean MCP joint angles at rest was observed in foals 24 hours after treatment, and these angles returned to pretreatment value by 96 hours.[21] In another study, maximum MCP joint angles that occurred during the stance phase of the stride at a walk and range of joint motion were significantly increased in treated foals compared with controls.[16]

Currently, 1–3 g (or 44 mg/kg) of oxytetracycline diluted in a small amount of saline IV is recommended for the average-sized foal.[12,18] The 44 mg/kg IV dose was used in one experimental study with good effects and no alterations in blood chemistry values.[21] It is generally ideal to perform this treatment within the first few weeks of life. One or two repeat treatments may be given, but the risk of renal problems should be considered with multiple dosing or overdosing.[10,37] The authors usually combine splinting with IV oxytetracycline treatment, especially in moderate to severe cases of congenital flexural deformities.

Splints may be made from PVC pipe tubing or fiberglass cast material. The PVC pipe can be bent with heat to whatever conformation is desired while the cast material can be molded over a padded bandage. In addition, IV simplex tubing has also been used,[18] acting as a rubber band over the dorsal aspect of the limb. It is often easiest to apply bandages and splints with the foal sedated and positioned in lateral recumbency. Regardless of the method, splinting increases tensional forces to the flexor units during weight-bearing, which induces flexor relaxation through the inverse myotatic

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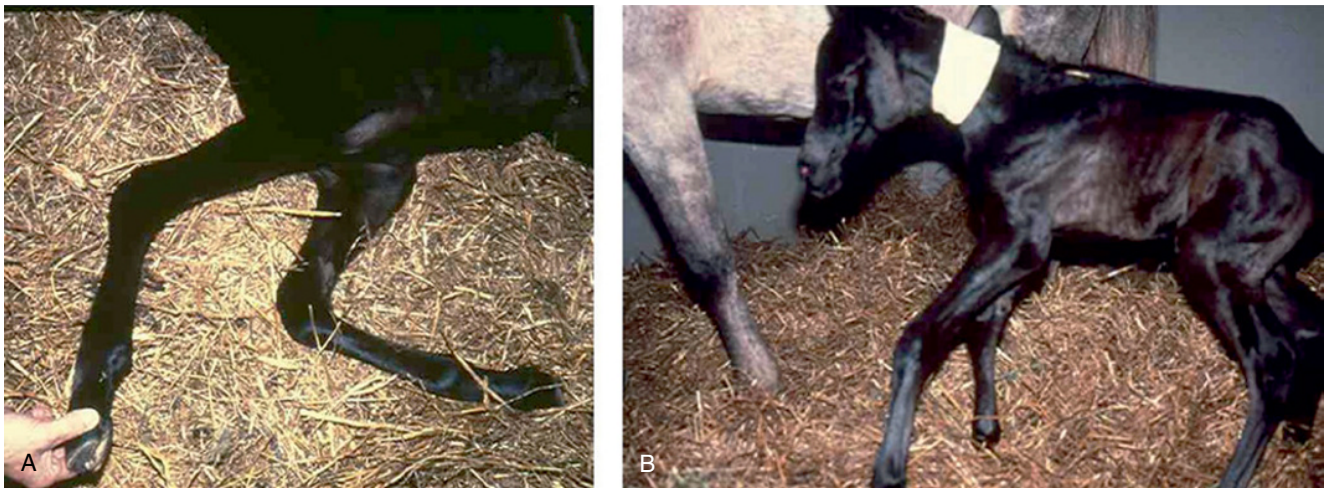


Figure 10.33. Images of a newborn foal with a carpal flexural deformity. (A) The clinician is attempting to straighten the left forelimb while the foal is recumbent. (B) Notice that the foal can straighten the left forelimb more when standing than with manual force being applied while recumbent.

reflex (stretching the musculotendinous unit leads to relaxation).[23] All splinting devices require strategic placement of padding, constant evaluation, and changing of the splints to prevent complications, as the skin can be easily irritated in foals (Figure 10.34). To help prevent decubital ulceration of the skin, many clinicians will keep the splints on for 6, 12, or 24 hours, followed by an equal amount of time out of the splint (bandage only).[12] In more severe cases where the splint needs to remain on, the angle of the splint will often need to be changed every 1–2 days as the foal starts to improve to prevent rotation and unequal pressure sores and to continue to provide constant tension to the limb.

Concurrent rupture of the extensor tendon usually responds spontaneously with stall rest, but bandaging and splinting may be needed to prevent knuckling over at the fetlock.[23,38] There is no need for surgical intervention in these foals as the ends will reappose naturally over about 6 months, with minimal swelling.[11,26] Exercise is usually restricted initially because the foals often have difficulty walking, but exercise may actually increase strength and tone in the flexor muscles.[10]

Surgical treatment of congenital tendon contractures is usually the last resort and often unnecessary. Flexor tendon tenotomies and carpal (inferior) or radial (superior) check ligament desmotomies have been used successfully but are not commonly indicated.[10,23] Transection of the superior check ligament may benefit some foals that are chronically over at the knees because the carpus becomes more hyperextended following transection.[3] Transection of the palmar carpal ligament in the palmar carpal joint capsule has been used to treat foals with severe carpal flexural deformities.[35] Tenotomy of the ulnaris lateralis and flexor carpi ulnaris 2 cm proximal to the accessory carpal bone has been used to treat less severe carpal flexural deformities with a very favorable outcome.[2,9] It is important to know that if tenotomies of the superficial or deep digital flexor tendons are performed in severe cases, then the surgeon usually cannot manually straighten the limb at surgery. This should not dictate cutting more tendons or ligaments as many will straighten within a week after surgery. Over correction is possible. Arthrodesis of the fetlock joint has also been used to correct severe flexural deformities of the fetlock in a donkey and miniature horse and may be used as a salvage procedure in some cases.[2,36] For most flexural deformities of the pastern, surgical realignment and arthrodesis of the pastern joint are usually performed with a fair prognosis for soundness.[22] Desmotomy of the peroneus tertius has been reported to improve a foal with a tarsal flexural deformity.[30]



Figure 10.34. Image of a distal limb of a foal that had multiple palmar splints applied to help straighten a fetlock flexural deformity. Notice the decubital ulceration of the skin due to pressure from the splinting.

ACQUIRED FLEXURAL DEFORMITIES

Acquired flexural deformities can be unilateral or bilateral and usually affect the DIP joint (club foot) or MCP joint (dorsal knuckling of the fetlock). Flexural deformities tend to occur in fast-growing horses or in those following a growth spurt. Flexural deformities of the DIP joint typically occur in foals and weanlings, and flexural deformities at the MCP joint are more typical of 1- to 2-year-old animals (Figures 10.35 and 10.36).[24,32,33,35]

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Figure 10.35. Weanling Quarter horses with typical club foot appearances. The dorsal hoof wall was vertical to the ground (A) with a concavity of the hoof wall (B). The deformities were corrected in both with an inferior check ligament desmotomy.



Figure 10.36. Visual (A) and radiographic (B) appearance of a yearling Quarter horse with a flexural deformity of the left front fetlock. The deformity did not respond to a previous inferior check ligament desmotomy.

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However, the relationship between age and the type of flexural deformity is not absolute.

Etiology

The cause of acquired flexural deformities is multifactorial. Historically, acquired flexural deformities have been grouped within the DOD complex because the potential causes and risk factors (overfeeding, nutritional imbalances, trauma, and genetics) are similar.[23] In addition, other types of DOD conditions such as physitis and OCD can often be seen concurrently in these animals and may actually contribute to the development of flexural deformities (Figure 10.37). This is because pain in the limb for any reason may initiate a flexion withdrawal reflex causing flexor muscle contraction and an altered position of the joint.[23] Chronic unweighting of the leg also may contribute to foot contracture, high heels, and the development of a club foot. Lack of exercise has been suggested to prevent proper stretching of the tendons and ligaments, contributing to limb contracture.[23] Severe trauma to a flexor tendon or its associated muscles is also known to cause tendon contracture due to fibrous tissue deposited during the reparative process.[13] The authors have seen flexural deformities of both the DIP and MCP joints develop subsequent to trauma to the elbow region.

The mechanism by which the suggested risk factors produce flexural deformities is still uncertain. Suggested hypotheses include the failure of tendons and ligaments to develop at the same rate as bone lengthening and a discrepancy between bone growth and the capacity for lengthening of the check ligaments.[13,23] Flexor muscles are stronger than extensor muscles, and the foal consequently develops a flexural deformity. These theories are not entirely compatible with our knowledge of bone growth since the majority of bone growth occurs early in life (first few months) and many flexural deformities develop later than that time period.

Clinical Signs

Flexural deformity of the DDFT centers around the DIP joint and results in a raised heel with a "club foot" appearance (Figure 10.35). The heel may be completely off the ground in severe cases (walking on the toe; Figure 10.38), but usually the heels maintain contact with the ground and grow excessively long (Figure 10.35). Most foals should not be lame, and a pronounced



Figure 10.37. This yearling Quarter horse filly had flexural deformities of both front fetlocks and subchondral cystic lesions in both stifles.

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Figure 10.38. Young foal with a flexural deformity of the DIP joint, in which the hoof wall is beyond vertical and the heels are completely off the ground. Surgical treatment is recommended for these types of deformities. An inferior check ligament desmotomy was performed in this foal.

lameness should cause wariness of other problems in the limb. Dishing or concavity of the dorsal hoof wall may occur in chronic cases (Figure 10.35B), and tearing of the dorsal laminae may lead to seedy toe and toe abscesses.

The severity of the deformity may be subdivided into stages I and II based on the visual position of the dorsal hoof wall.[23,24] Stage I contracture is when the dorsal hoof wall does not go beyond vertical (Figure 10.35A), and stage II is when the dorsal surface of the hoof passes beyond vertical (Figure 10.38). The more severe the deformity, the more shortening of the musculotendinous unit, and the more aggressive treatment should be. With severe contracture, pathologic changes may develop in the joint capsule and other tissues of the coffin joint, making permanent correction less likely.[13,23] With chronicity, more changes can happen to the hoof capsule due to the pressure being placed on the toe. The degree of concavity at the toe and discrepancy in growth rings from dorsal to palmar have been categorized into different stages.[8,27]

Flexural deformity of the MCP joint has been classically referred to as contracture of the superficial digital flexor tendon (SDFT). It is characterized by dorsal knuckling of the fetlock with the hoof itself remaining in normal alignment (Figures 10.36 and 10.37). The term SDFT contracture is a gross oversimplification because the SDFT, DDFT, both the SDFT and DDFT, and the suspensory ligament in chronic cases may the involved with the deformity.[2] Early in the condition, the fetlock and pastern may begin to appear more upright with the fetlock angle approaching 180° measured from the dorsal surface (the normal angle is approximately 140°).[2] In more severe cases, the fetlock knuckles forward with every step, and the horse may stand with a dorsal fetlock angle of more than 180° (Figure 10.39).

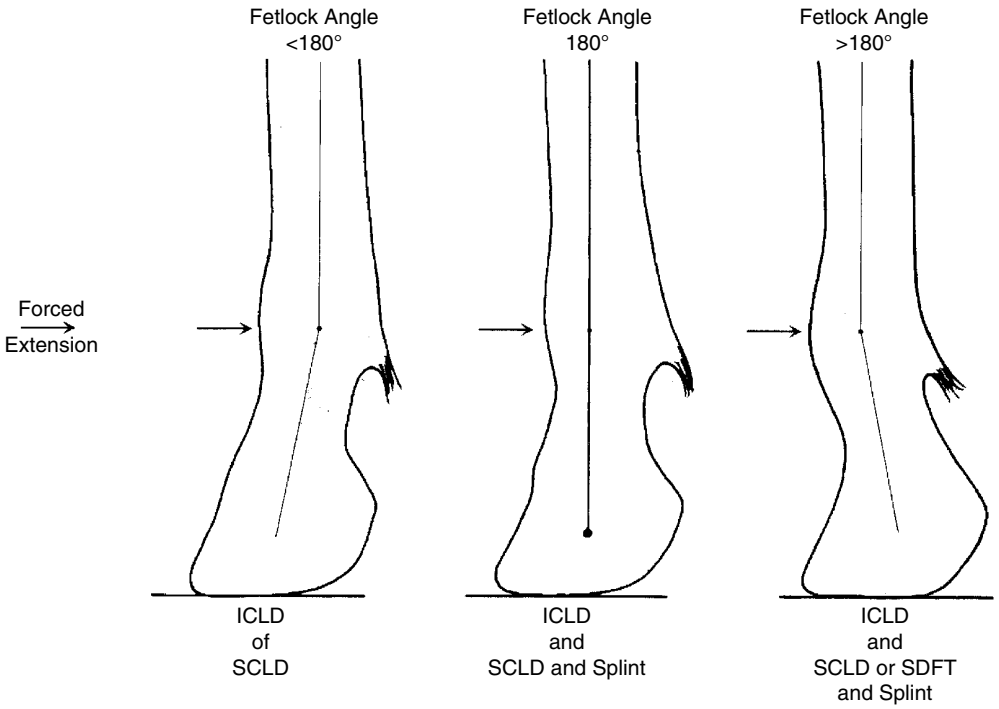


Figure 10.39. Treatment guidelines based on the angle of the fetlock for horses with flexural deformities of the fetlock joint. *Source*: Reprinted with permission from Adams and Santschi.[1] Reproduced with permission of American Association of Equine Practitioners.

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As with flexural deformities of the DIP joint, when the deformity goes beyond vertical (constant dorsal knuckling of the fetlock), more aggressive treatment is usually required. In chronic cases, the suspensory ligament becomes involved, fibrosis of the joint capsule may occur, and osteoarthritic changes may develop in the fetlock joint.[23]

Diagnosis

A tentative diagnosis of flexural deformities of the DIP or MCP joints usually can be made based on the characteristic foot and limb conformation. Flexural deformities of the DIP joint only involve the DDFT. Determining which soft tissue structure(s) is involved with deformities of the MCP joint is more difficult. Since these acquired deformities can developed secondary to a painful focus, such as physitis, it is important to identify any potential sources of pain, as without eliminating those, it will be very difficult to treat the flexural deformity.

The horse should be examined from a distance in its natural environment and then on flat ground to determine how much the limb can straighten while moving or standing flat. Standing palpation should then be performed to determine the presence of any asymmetries that could be related to physitis or joint effusion (possible OCD). Similar to congenital cases, the clinician can attempt to manually straighten the limb while simultaneously palpating the flexor surface of the limb to see if any specific structure becomes more taught. For instance, the DDFT, SDFT, and the suspensory ligament may all contribute to an MCP deformity.[2] Careful palpation of the limbs in both the standing and flexed positions may suggest that the DDFT or SDFT is more taut on palpation. However, if this cannot be determined, it is probably best to assume that both the DDFT and SDFT are involved to avoid failure in treatment. It is also the authors' opinion that younger horses with MCP joint deformities usually have primary DDFT involvement (Figure 10.40), whereas older horses often have both SDFT and DDFT involvement (Figure 10.36). This decision is very important when selecting surgical treatment.

Radiographs can be used to confirm the diagnosis and assess any changes in the joints involved or those affected by the abnormal stance. Lateral-to-medial and 60° dorsopalmar views of the foot should be performed in foals with DIP joint deformities and at least two views of the phalanges/fetlock for MCP deformities. The degree of DIP joint subluxation, angle of the dorsal hoof wall, and abnormalities at the apex of the distal phalanx should be observed. Varying degrees of osteolysis in the distal part of the distal phalanx is not uncommon, and the foals with the most pronounced clinical signs usually display the most prominent radiologic changes (Figure 10.41A).[4] Radiographs of the phalanges/fetlock in horses with MCP deformities usually reveal dorsal knuckling of the fetlock with no other bone abnormalities of that joint, but can have varying degrees of DIP joint subluxation (Figures 10.36B and 10.41B).



Figure 10.40. This 6-month-old Quarter horse filly with flexural deformities of both front fetlocks responded well to bilateral inferior check ligament desmotomies.

Treatment

Nonsurgical Treatment

Nonsurgical treatment may consist of changes in diet and exercise, corrective trimming and shoeing, splinting for MCP deformities, IV oxytetracycline, and the use of NSAIDs if pain is considered to be a contributing factor.[1,2,23] Mild deformities or those in the early stages of the disease are the most appropriate candidates for conservative treatment. Foals with DIP joint deformities that cannot touch the heel to the ground or MCP joint deformities that constantly knuckle forward at the fetlock are not candidates for nonsurgical treatment (Figures 10.38 and 10.39).

Animals with flexural deformities of the DIP joint should have the heels trimmed to increase tension on the palmar flexor tendons (primarily the DDFT). Trimming of the heel combined with a toe extension or elevation also may be used. Extension of the toe may be accomplished with a steel shoe, glue-on shoe, or acrylic applied to the bottom of the foot, depending on the age of the foal.[8,27] In general, toe extensions are not as well tolerated in foals as they are in older individuals.[27] Application of acrylic at the toe of foals helps maintain the health of the toe as well as the concavity of the foot while shifting

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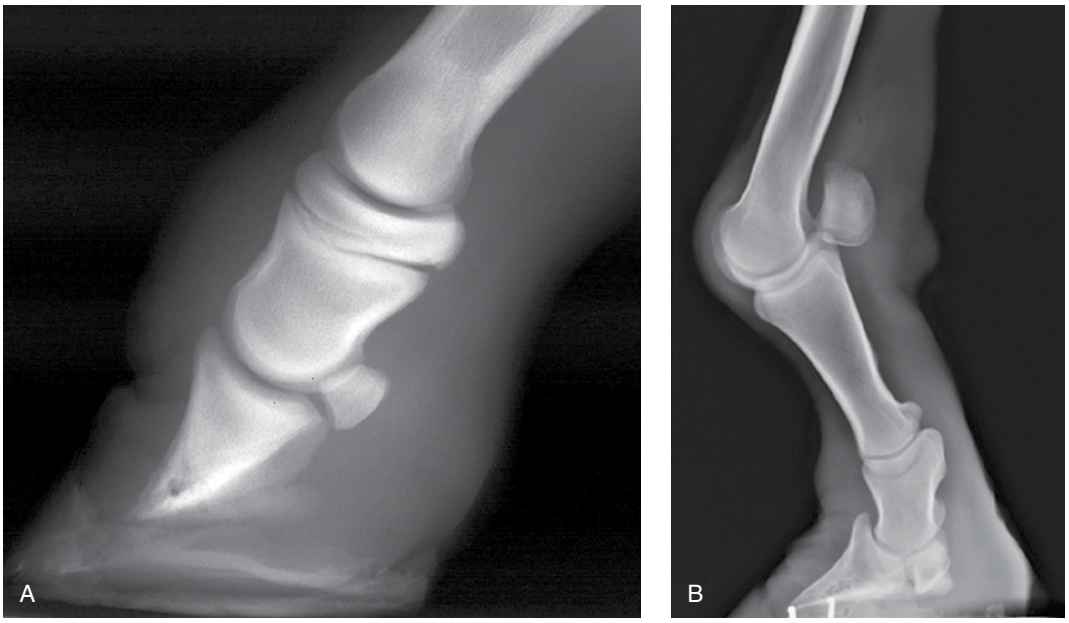


Figure 10.41. (A) Lateral radiograph of a foal with a club foot. Remodeling and lysis of the distal aspect of the distal phalanx was present, and the foal had a concurrent abscess in the toe. (B) Lateral radiograph of the distal phalanges on a yearling with a chronic MCP flexural deformity. Note that even though the fetlock joint has no changes, the DIP joint is chronically subluxated.



Figure 10.42. Rocker shoe placed on a 2-year-old horse after trimming its heel so that it could rest it forward if painful but would transfer weight to the palmar structures when walking. Walking on flat surfaces is crucial for this type of shoe to be effective.

the force palmarly.[27] As the heel is trimmed, the horse may experience pain related to stretching the DDFT and may require NSAID administration. In older individuals, another way to help counteract this pain is to use a rocker shoe that can serves to slowly stretch the DDFT (Figure 10.42) after the trim, but the horse must be walked on a firm surface to ensure that downward force goes through the heel.

It is generally recommended to raise the heels with corrective shoeing to treat horses with MCP joint deformities. This is thought to relax the DDFT and cause the fetlock to drop.[23,32] However, each case should be evaluated individually by placing wedges under the foot to either elevate the heel or elevate the toe to observe which scenario improves the angle of the fetlock (Figure 10.43). Whether heel elevation reduces strain on the SDFT and suspensory ligament is debatable, but most clinical cases respond best to heel elevation in contrast to lowering the heels.[2,23] Heel elevation (2–3 cm) combined with a 1- to 2-cm toe extension also has been advocated.[2] Corrective shoeing is often combined with splinting of the MCP joint to prevent dorsal knuckling of the fetlock. The PVC splint is applied to the palmar aspect of the distal limb to pull the fetlock in a palmar direction. Conforming the PVC splint (using heat) to the palmar aspect of the limb or use of fiberglass cast material may improve the effectiveness of the splinting.[2]

Corrective trimming and shoeing, and splinting if used, may contribute to lameness in some horses by increasing tension on the palmar soft tissues. In addition, horses must bear full weight on the limb(s) to achieve the maximum benefit of nonsurgical treatment. Therefore, low doses of oral NSAIDs are recommended for 1 to 2 weeks to help stretch the musculotendinous unit. Oxytetracycline IV can be used as an adjunct treatment similar to that in foals with congenital flexural deformities, but appears to be less effective in older animals.

Surgical Treatment

The decision for surgical treatment of DIP and MCP joint flexural deformities depends on the severity of the deformity, age of the horse, and response to conservative treatment. Surgical intervention is the only alternative in cases that do not respond to conservative methods.

Surgical options for foals with deformities of the DIP joint include an inferior check ligament desmotomy or DDFT tenotomy at the level of the pastern or mid-metacarpus.[1,2,15,29,33] Foals treated at a younger age (younger than 6–8 months) with an inferior check ligament

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Figure 10.43. Elevation of the heel vs. the toe with a wedge in horses with flexural deformities of the fetlock can help guide both surgical and conservative treatment. In most horses, heel elevation causes the fetlock to drop and improves the angle of the fetlock.

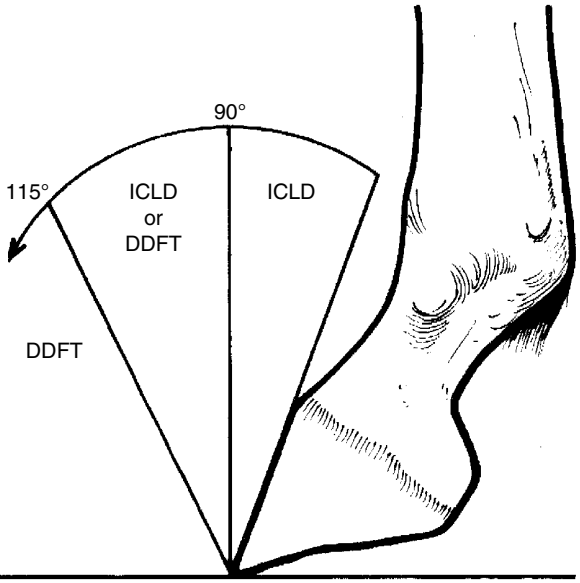


Figure 10.44. Treatment guidelines for foals with flexural deformities of the DIP joint based on the angle of the dorsal hoof wall relative to the ground. *Source*: Reprinted with permission from Adams and Santschi.[1] Reproduced with permission of American Association of Equine Practitioners.

desmotomy have an improved prognosis for normal hoof conformation and for athletic performance.[29,35]

Surgical options for horses with deformities of the MCP joint include an inferior check ligament desmotomy, a superior check ligament desmotomy, both inferior and superior check ligament desmotomies, and SDFT tenotomy in the mid-metacarpus.[1,2,17,32] There is scarce information regarding the prognosis of horses with MCP flexural deformities following surgery. However, in the authors' experience, lack of response to surgery and recurrence of the flexural deformity is much more common with MCP joint deformities than with DIP joint deformities. In addition, multiple surgeries may be required since the source of the deformity is harder to initially identify. There should be at least 2 weeks between surgeries, since some horses will improve more a few days after surgery than they did immediately post-op.

The majority of DIP deformities can be corrected with an inferior check ligament desmotomy. This includes all type I and many type II deformities if the surgery is performed at a young age (younger than 6–8 months) and if combined with corrective shoeing such as a toe extension and/or toe elevation.[23,35] An inferior check ligament desmotomy is recommended in foals with hoof-ground angles of 90° or less and in most foals with deformities between 90° and 115° (Figure 10.44).[2] An inferior check ligament desmotomy is highly recommended in foals and those older than 1 year of age that have not responded to conservative therapy, with hoof deformities approaching or beyond vertical, with osteolysis of the distal phalanx, and that cannot touch the heel to the ground.

The surgery can be performed with the traditional open approach in the proximal metacarpus or with ultrasound guidance through a 1- to 2-cm incision.[15,23,24,29,33] Trimming the heel and using a shoe with a toe extension, toe elevation, or rocker shoe is recommended following surgery in most cases (especially older horses) to achieve a normal hoof conformation as quickly as possible. Normal limb alignment may be obtained almost immediately following surgery, but relaxation may progress for 7–10 days postoperatively.[23] Normal hoof conformation should be achieved within 2 weeks of surgery in most cases. Several studies have documented that the majority of foals treated with an inferior check ligament desmotomy develop a normal hoof conformation and can be used for their intended purpose.[24,29,33,35] Experimental studies also have documented that transection of the inferior check ligament causes minimal changes in locomotion, joint angles, or strains to the palmar soft tissue structures in normal horses.[6,7] The surgery itself should have minimal effects on future performance.

Tenotomy of the DDFT is usually reserved for older horses (older than 1 year) with severe deformities of the DIP joint. A DDFT tenotomy is recommended if the hoof-ground angle is more than 115° (Figure 10.44).[2] Foals with deformities between 90° and 115° may respond to an inferior check ligament desmotomy, and this should be attempted initially. However, the response to surgery is less predictable in these cases.[2,23] The DDFT tenotomy may be performed in the mid-metacarpus or

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the palmar aspect of the pastern. Greater release of the DDFT and correction of the DIP joint deformity can usually be obtained with transection in the pastern, but the wider separation of the tendon ends is more of a concern with later function. Fewer complications and a better functional outcome are usually achieved with DDFT tenotomy in the mid-metacarpus compared with the pastern region.[2] Tenotomy of the DDFT should be considered a salvage procedure; however, many of these horses can be used for light riding.[1,2]

The results of surgical treatment of MCP joint deformities are less predictable than those of deformities of the DIP joint. The recommended surgery also is less defined and is often based on the clinical experience of the veterinarian and the physical examination findings of the horse. Surgical guidelines have been recommended based on the angle of the MCP joint (Figure 10.39).[2] Mild deformities have a fetlock joint angle of less than 180°, and an inferior or superior check ligament desmotomy is recommended. Moderate deformities are those with a fetlock angle of 180° (straight upright), and performing both an inferior and superior check ligament desmotomy is recommended. Severe deformities have a fetlock angle of more than 180° (knuckled forward), and an inferior check ligament desmotomy combined with a superior check ligament desmotomy or SDFT tenotomy is recommended (Figure 10.39). Splinting is recommended after surgery for both moderate and severe MCP joint deformities.

Often the decision of which desmotomy (or both) to perform is based on which tendon feels more taut on palpation. If the SDFT is most taut, then a superior check ligament desmotomy may be appropriate, or an SDFT tenotomy if the deformity is severe. If the DDFT is most taut, then an inferior check ligament desmotomy is indicated. However, this approach is very subjective, and little information is available regarding its appropriateness. In one report of 15 cases of MCP joint flexural deformities, mild deformities were treated nonsurgically, moderate deformities were treated with an inferior check ligament desmotomy, and severe deformities were treated with both inferior and superior check ligament desmotomies.[23] In a more recent report of using tenoscopy to transect the superior check ligament, both desmotomy procedures were performed on horses with MCP flexural deformities.[17]

One author has found that younger horses (younger than 1 year) with MCP joint deformities respond better to inferior check ligament desmotomy alone than older horses. A combination of inferior and superior check ligament desmotomy is usually recommended by the author in yearlings or 2-year-olds with MCP joint flexural deformities. However, successful results have been reported with inferior check ligament desmotomy alone by some authors.[23,32] A tenoscopic approach is currently recommended for superior check ligament desmotomy to minimize the complications seen with the open approach.[17,28]

Horses with severe MCP deformities have a poor prognosis and often do not improve with either or both check ligament desmotomy surgeries.[32] Transection of the SDFT in the mid-metacarpus in the standing sedated horse is often the quickest, least expensive, and most beneficial procedure in these cases (Figure 10.45). However, an SDFT tenotomy combined with an inferior check ligament desmotomy has been recommended in these cases (Figure 10.39).[2] Suspensory ligament/branch desmotomy also may be considered when the MCP joint deformity is refractory to other surgical treatments, but is often unsuccessful due to fibrosis and scarring of the soft tissues around the fetlock that often accompany chronic cases.[23] In addition, it has been one author's experience that once the suspensory ligament is transected, there is a risk of the fetlock dropping several weeks after surgery.



Figure 10.45. Before (A) and after (B) images of a 2-year-old with severe flexural deformities of the front fetlocks that had the SDFT transected in the mid-metacarpal region. This horse had been treated previously with an inferior check ligament desmotomy.

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Prognosis

In general, the prognosis is much better for foals with DIP joint flexural deformities than older horses with MCP joint deformities. Most reports indicate that young (under 5–6 months) horses with a DIP joint deformity treated surgically have a better prognosis for future athletic use than older horses.[29,35] In one report, 33 of 40 horses treated with an inferior check desmotomy were used for their intended purpose.[33] In addition, foals treated surgically with an inferior check desmotomy had an improved prognosis for racing than those treated conservatively.[29] In another report, Thoroughbreds with a mean age of 151 days that underwent inferior check ligament desmotomy were less likely to start in a race compared with their maternal siblings. However, for those who raced, the longevity of career and earnings per race were not different than controls.[34] In contrast, the response to surgery is often unpredictable in horses with MCP joint deformities, and these horses often have a guarded to poor prognosis for soundness. In one report, horses with mild and moderate MCP joint deformities returned to useful work, but none of the horses with a severe flexural deformity became athletes.[32] Similar to DIP joint deformities, it is the author's opinion that MCP joint deformities respond much better to either nonsurgical or surgical treatments in younger horses. The sooner treatment can be initiated in either type of deformity, the better the prognosis for correction and future athletic use.

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OSTEOCHONDROSIS

C. Wayne McIlwraith

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INTRODUCTION

Osteochondritis dissecans (OCD) is arguably the most important entity within the developmental orthopedic disease (DOD) complex. It is a frequent cause of lameness in young athletic horses and is the most frequent condition of the complex requiring surgical intervention. OCD has been classically considered as a manifestation of osteochondrosis.[10] Rejno and Stromberg[22] described the first stages of osteochondrosis as a disturbance of cellular differentiation in the growing cartilage and the second as involving the process of basal forces within the joint, giving rise to fissures in the damaged cartilage. The terms osteochondrosis, osteochondritis dissecans, and osteochondrosis dissecans have been regularly used as synonyms, but this is misleading. Poulos[18] distinguished them as follows: osteochondrosis is the disease, osteochondritis is the inflammatory response to the disease, and OCD is the condition in which a flap can be demonstrated. This is a simple but fairly appropriate representation.

Subchondral bone cysts (SBC) or subchondral cystic lesions (SCLs) were also proposed by Rejno and Stromberg[26] as a manifestation of osteochondrosis. The author considers that SCLs showing up clinically in the first 2 years of life are indeed manifestations of osteochondrosis. However, they represent quite a different disease than OCD. Examination of the lining of enucleated SCLs in the medial femoral condyle showed that they produce increased levels of PGE_2, neutral metalloproteinases, and nitric oxide (NO) and that there was enhanced osteoclastic resorption activity attributable to the tissue.[20] *In situ* hybridization of sections of fibrous tissue of SCLs showed that mRNA of IL-1β was upregulated at the periphery of the cystic lesion and IL-6 was upregulated in the fibrin tissue of the center.[21] Other work showed that SCLs could be produced after 5-mm-diameter, 3-mm deep defects were created in the subchondral bone plate at the central weight-bearing of the medial femoral condyle,[19] leading to an alternative pathogenesis for clinical cases in older horses.

Other diseases in the DOD complex have sometimes been referred to as osteochondrosis, but the important clinical manifestations of osteochondrosis (and the primary ones that fit this pathogenesis) are OCD and SCLs, and discussion will be limited to those conditions. Osteochondrosis also can occur in the physis, but it is uncommon and rare for it to cause a clinical problem. Figure 10.46 illustrates the proposed pathways of osteochondrosis leading to both OCD and SCLs. More details on the osteochondrosis process can be found elsewhere.[28]

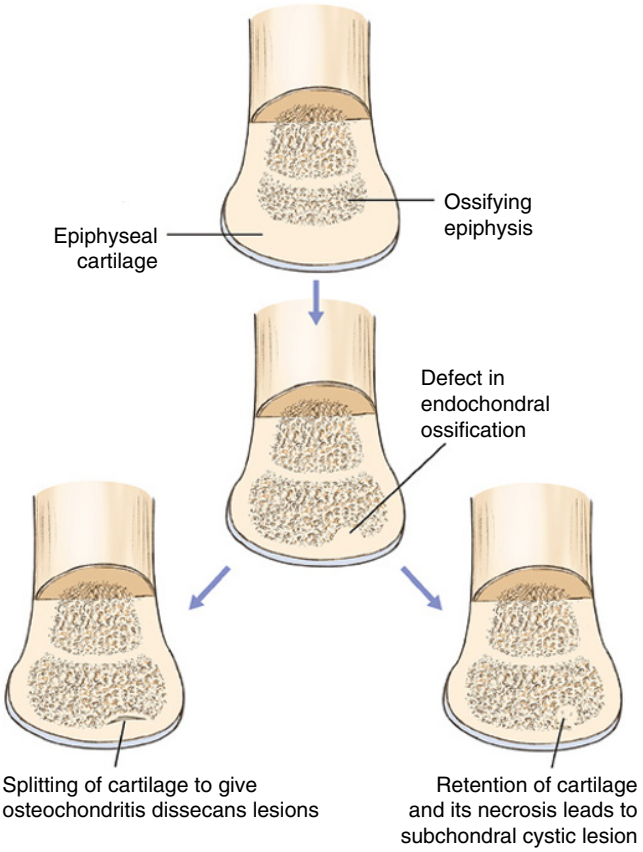


Figure 10.46. The proposed pathways of osteochondrosis leading to both OCD and SCLs.

OSTEOCHONDRITIS DISSECANS (OCD)

Disease Manifestation

Three categories of OCD lesions are recognized:

1. Those showing clinical and radiographic signs.
2. Those showing clinical without radiographic (but arthroscopic) signs.
3. Those showing radiographic, but no clinical signs.

Data from the first two categories of disease have been tabulated from the most commonly selected joints from the author's surgical case reports.[9] The relative incidence of clinical signs vs. radiographic lesions also has been documented in the femoropatellar joint by McIntosh and McIlwraith.[15] The third category of radiographic lesions has become increasingly important because of the common use of presale radiographs of yearlings.[6,7]

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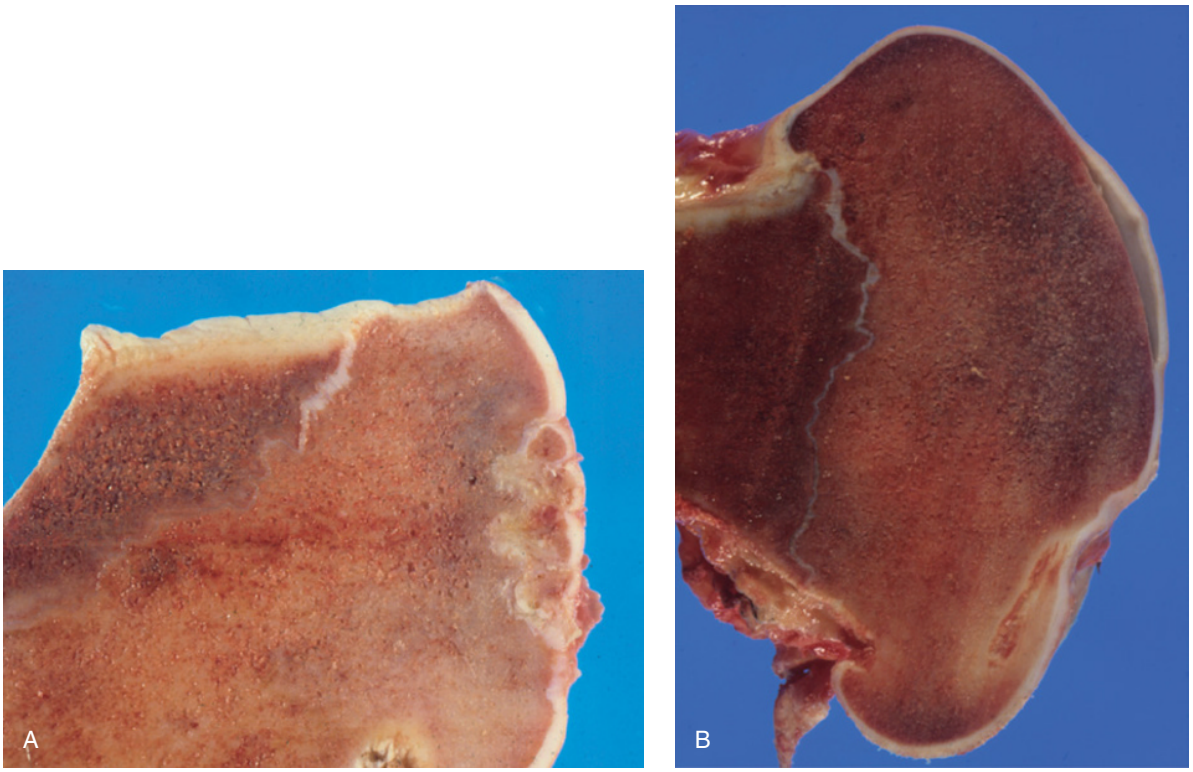


Figure 10.47. Variation in depth with which OCD lesions can extend on the lateral trochlear ridge of the femur. (A) This is a typical lesion with fragments within pathologic cartilage in a clear defect in the subchondral bone (this will be easily discernible radiographically). (B) Less commonly (and usually on the medial trochlear ridge of the femur), there can be elevated cartilage, which has normal fragments with normal bone contour and no evidence of an endochondral ossification defect.

Osteochondrosis affects the articular (joint) cartilage and often involves the subchondral bone beneath the cartilage surface (Figure 10.47A). Generally, a dissecting lesion develops that involves cartilage, or cartilage and bone, and the defection plane ultimately reaches the joint surface. It is theorized that clinical signs develop when the joint surface is reached by the dissecting lesions. Sometimes the OCD fragments can detach and become a loose body or joint mouse. In most instances, however, the fragments remain loosely attached to the bone of origin, but the debris that is released into the joint from the flap results in synovitis or joint inflammation and the clinical signs of pain and lameness that are seen with the disease.

In some instances there is no defect in the subchondral bone, but merely separation of cartilage (normal thickness) from a normal subchondral bone contour (Figure 10.47B). These manifestations not only lead to questioning of the classical defects in the endochondral ossification concept but also provide diagnostic challenges because there is no defect identified on radiographs.

OCD can affect a number of joints, but the most commonly involved are the femoropatellar, tarsocrural, and fetlock joints. Shoulder joints also are affected, but less frequently. Each of these entities is discussed separately.

Osteochondritis Dissecans of the Femoropatellar Joint

The femoropatellar joint is one of the principal joints affected with OCD. Although stifle OCD can be diagnosed in almost any breed, it seems to be more common in Thoroughbreds than in other breeds (Table 10.2).[4] Approximately 60% of affected horses are 1 year of age or younger when the condition becomes symptomatic, and younger animals that develop clinical signs often have more severe damage within the joint (Table 10.3).[4] However, incidental radiographic lesions are sometimes

Table 10.2. Breed distribution of 161 horses presented for femoropatellar OCD.

Breed	Number	Percentage
Thoroughbred	82	50.9
Quarter horse	39	24.2
Arabian	16	9.9
Warmblood	9	5.6
Crossbred	5	3.1
Paint horse	3	1.9
Appaloosa	3	1.9
Other	4	2.5

Source: Foland et al.[4] Reproduced with permission of Equine Veterinary Journal.

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Table 10.3. Age distribution of 161 horses presented for femoropatellar OCD.

Age (year)	Number	Percentage
<1	22	13.7
1	68	42.2
2	36	22.4
3	21	13
>4	14	8.7

Source: Foland et al.[4] Reproduced with permission of Equine Veterinary Journal.

identified in older horses in which no clinical signs have ever been observed.

Clinical and Radiographic Signs

Animals usually present with a sudden onset of joint swelling and lameness. A recent increase in the level of exercise is sometimes part of the history. Lameness may be very mild, with a stiff action and shortened stride observed, rather than the horse having a prominent lameness. Some more severely affected horses have a bunny hop action behind, and severe cases can sometimes be confused with a neurologic problem due to an inability to flex the stifle and may appear to be circumducting the limb. Joint distention, however, is the most consistent clinical sign seen in horses with OCD of the stifle (Figures 2.90 and 5.110). Careful palpation of the joint may identify free bodies or the surface irregularity associated with the damage within the joint. Bilateral involvement is common in the femoropatellar joint, so both joints should be examined carefully. Flexion of the limb usually exacerbates the lameness, and anesthetic placed into the joint improves or eliminates the lameness. However, intra-articular anesthesia is usually not necessary to confirm a diagnosis.

Lateral-to-medial radiographs provide the most useful information regarding specific lesion location and size (Figure 10.48). The most commonly identified defect is a variably sized irregularity or flattening of the lateral trochlear ridge of the femur. The area of the ridge that comes in contact with the distal aspect of the patella is most commonly involved. Partial mineralization of the tissue within the defect or fragment formation is often seen, and free bodies are also occasionally identified. It is rare to see OCD primarily affecting the patella, but secondary radiographic changes in the patella resulting from the trochlear ridge damage are often seen at arthroscopy (Figure 5.113). The medial ridge of the femur is less commonly involved.

Generally, the extent of damage to the joint identified at surgery is more extensive than would be predicted from radiographs.[25] Although other joints may be involved concurrently, this is uncommon. In one study of 161 horses with stifle OCD, 5 also had OCD affecting the rear fetlocks, 4 had hock OCD, and 1 had OCD of a shoulder joint.[4]

Treatment

It is generally accepted that surgical debridement of the lesions using arthroscopic surgery is the treatment of choice.[4,11,15] However, smaller lesions in younger horses may respond to rest and resolve radiographically.[15] It has also been shown in a study taking radiographs once a month for the first year of life in a group of Warmblood foals that femoropatellar OCD lesions can resolve.[3] They can develop into obvious lesions radiographically, but then completely heal. In the case of the femoropatellar joint, healing is completed (if they are going to heal) by 8 months of age. These are generally lesions that are not causing severe clinical signs. If lameness and swelling are prominent and the horse is older than 8 months, arthroscopic surgery is indicated.

As for all joint surgery, the joint is thoroughly explored, and suspicious lesions are probed. Loose or detached tissue is elevated and removed (Figure 10.49), and loose bodies are removed. The defect site is then debrided down to healthy tissue. Care must be taken to not be overly aggressive with bone debridement in young animals that have soft subchondral bone. Salvage and reattachment of OCD cartilage flaps in the stifle have been reported.[15,17] While not common, an OCD cartilage flap that is relatively smooth and is not detached on its entire perimeter can be elevated and the underlying necrotic cartilage and marrow fibrosis debrided. The flap can then be replaced and secured with polydioxanone (PDS) pins (OrthoSorb, Ethicon, Johnson and Johnson) or PLLA tacks (Chondral Dart, Arthrex, Naples, FL).

Prognosis

In one study of 252 stifle joints in 161 horses with follow-up information available for 134 horses, 64% returned to their previous use, 7% were in training, 16% were unsuccessful, and 13% were unsuccessful due to reasons unrelated to the stifle.[4] The success rate was higher in horses with smaller lesions and in older horses. However, the age factor was considered to be due to the fact that the most severe lesions were generally identified in younger horses.

Osteochondritis Dissecans of the Tarsocrural (Hock) Joint

Tarsal OCD most commonly involves the intermediate ridge of the tibia.[13] However, lesions can also develop on the trochlear ridges (the lateral ridge is much more common than medial ridge) and the medial malleolus of the tibia.

Clinical and Radiographic Signs

The most common clinical sign of tarsal OCD is effusion of the tarsocrural joint. Lameness also can be seen but it is uncommon and rarely prominent. Racehorses usually present as 2-year-olds, but nonracehorses usually present as yearlings prior to beginning training.

On radiographs, lesions, in order of incidence, can occur on the distal intermediate ridge of the tibia (DIRT) (Figure 10.50), followed by the lateral trochlear ridge of

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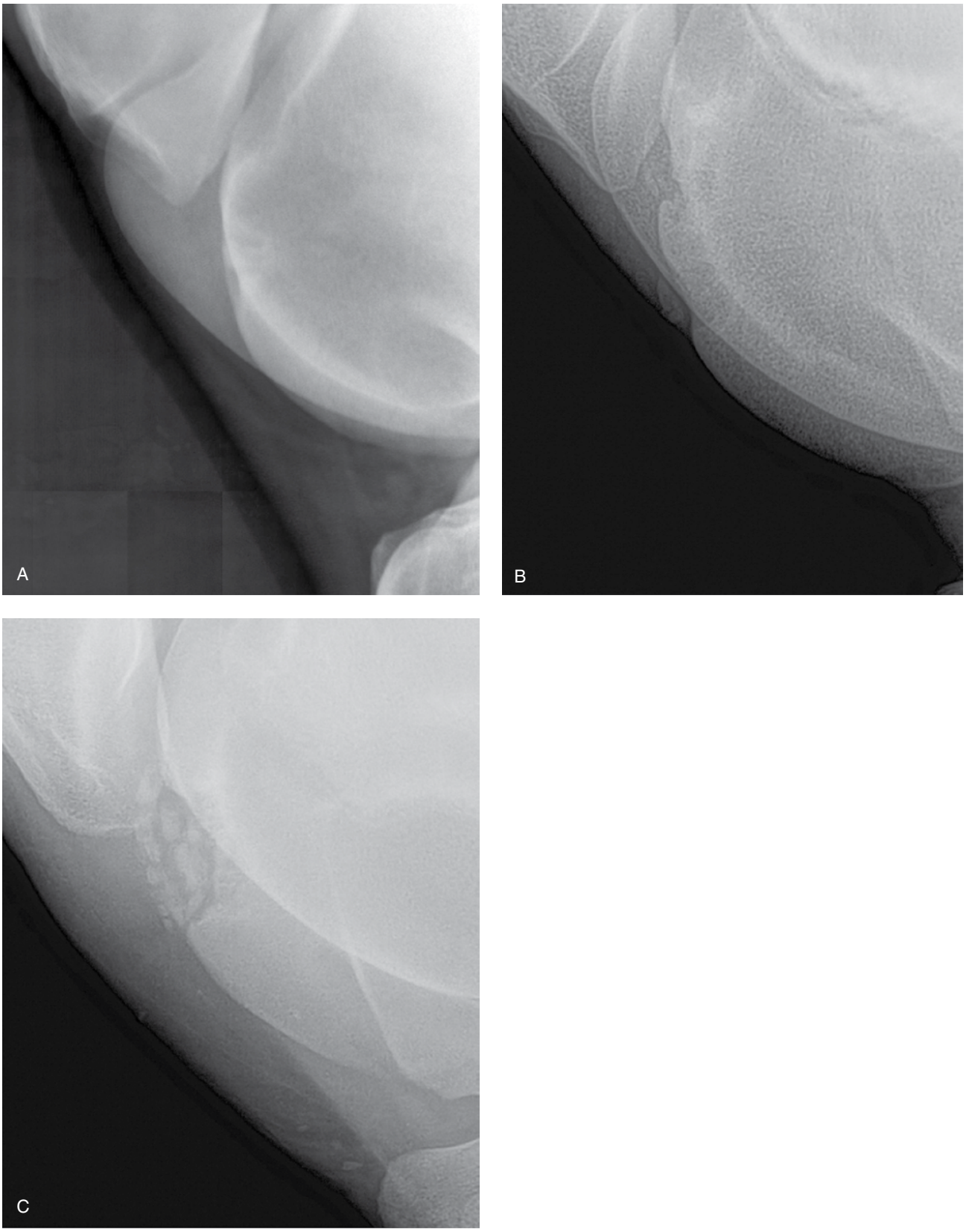


Figure 10.48. Radiographs of three cases of OCD of the lateral trochlear ridge of the femur in the femoropatellar joint. (A) A small defect in the lateral trochlear ridge without obvious fragmentation within the defect (treated conservatively). (B) A fragment within a defect. (C) Multiple fragments in a more severe lesion.

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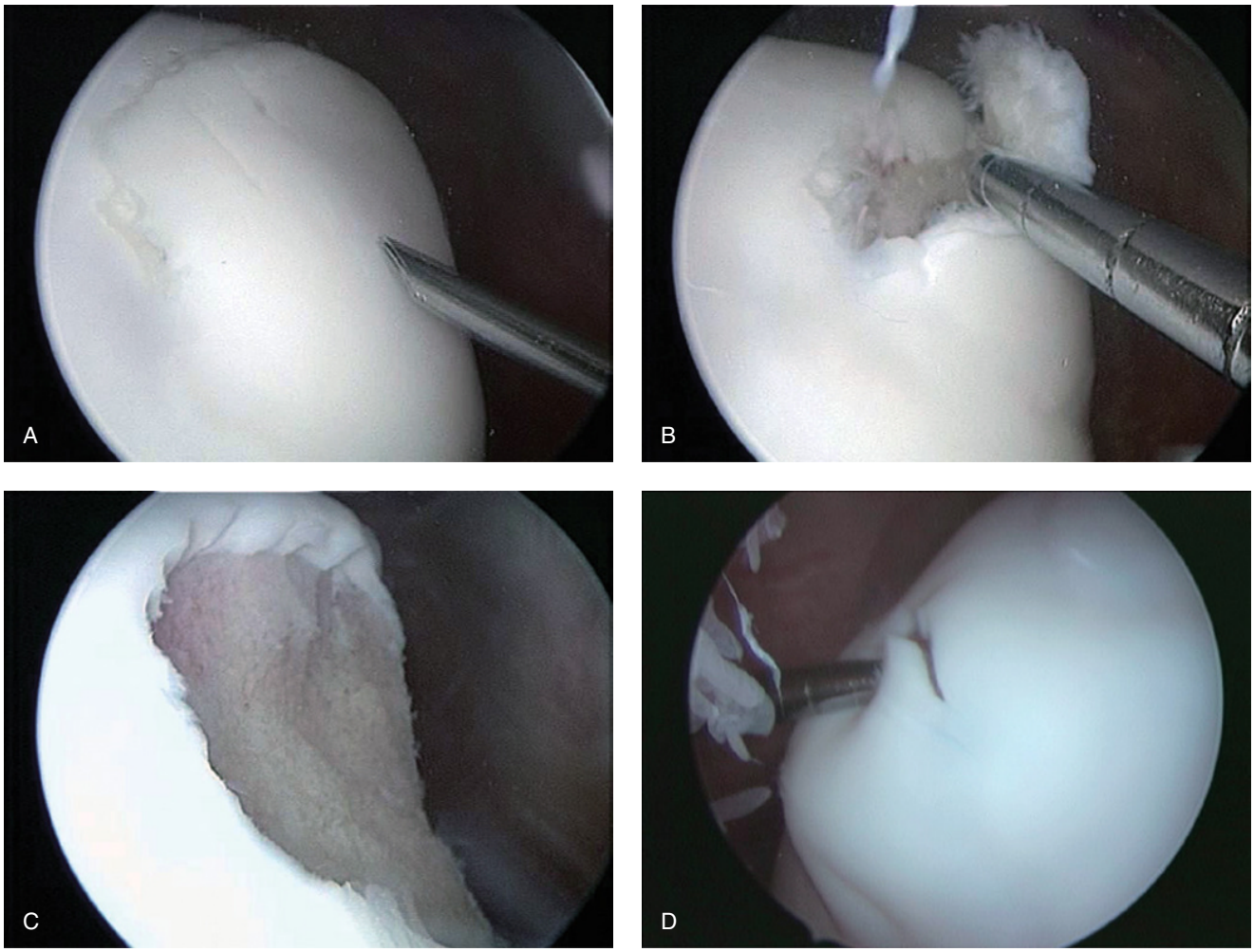


Figure 10.49. Arthroscopic views of OCD of the lateral trochlear ridge of the femur prior to probing (A), during elevation of the OCD flap (B), and after debridement of osteochondritic tissue (C). (D) A lesion on the medial trochlear ridge of the femur manifesting as elevated cartilage of normal thickness without a subchondral defect beneath.

the talus (Figure 10.51) and then the medial malleolus of the tibia (Table 10.4). Lesions are identified as defects alone or defects containing fragmentation. The radiographic appearance often underestimates the extent of damage identified at surgery, particularly for lateral trochlear ridge lesions. The hock is also a joint in which radiographically silent lesions (those identified at surgery and in which no abnormality was seen on radiographs) occur more commonly than in other joints.[13]

Treatment

Although lameness is usually minimal with hock OCD, surgery is the recommended treatment. Lameness may only be a problem at racing speeds, or at upper levels of performance, and this cannot be determined during a clinical examination. Resolution of the effusion cannot be expected without removal of the abnormal tissue. However, not all horses need surgery. Those with small lesions, minimal effusion, no lameness, and a potential career as a pleasure horse or light use horse may not require surgery. Sequential radiographs of Dutch Warmblood tarsocrural joints showed development (and resolution in most cases) of lesions of the DIRT and lateral trochlear ridge of the talus over the first 5 months of life.[3] Surgery should be considered early enough in the course of the disease (but after 5 months of age) so that the joint capsule is not unduly stretched, which makes resolution of the joint effusion less likely. Arthroscopic surgery with removal of fragments and debridement of defective tissue is recommended.

Prognosis

In a study involving 183 horses, 76% raced successfully or performed at their intended use after surgery.[13] Only 11% were unsuccessful because of a persistent hock problem. If degenerative changes were identified at surgery in the cartilage remote from the OCD lesion (wear lines on the medial trochlear ridge of the talus), the prognosis was less favorable. Complete resolution of effusion was inferior for lesions involving the lateral trochlear ridge and medial malleolus compared with the intermediate ridge of the tibia; however, this seemed to have no

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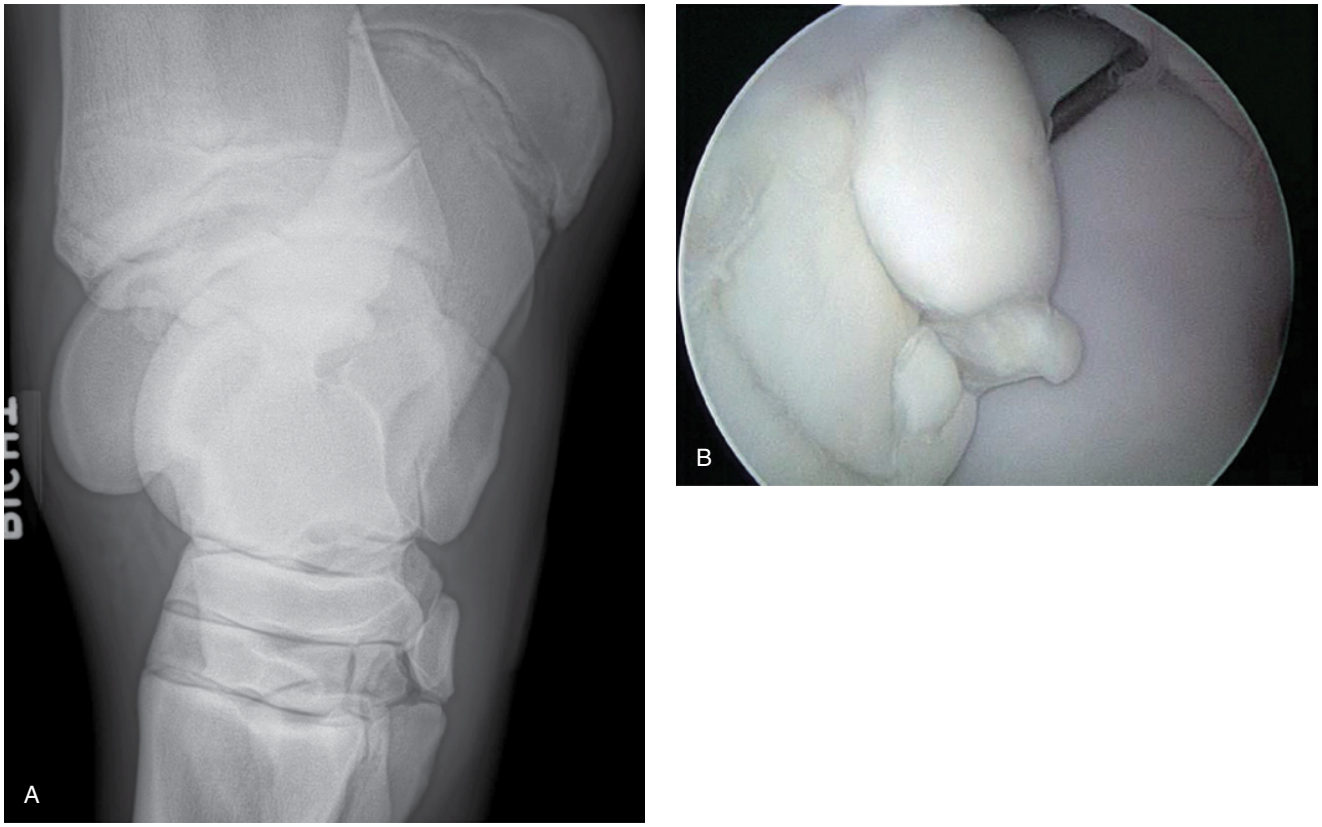


Figure 10.50. Radiographic view (A) and arthroscopic view (B) of OCD of the distal intermediate ridge of the tibia (DIRT) in the tarsocrural joint.

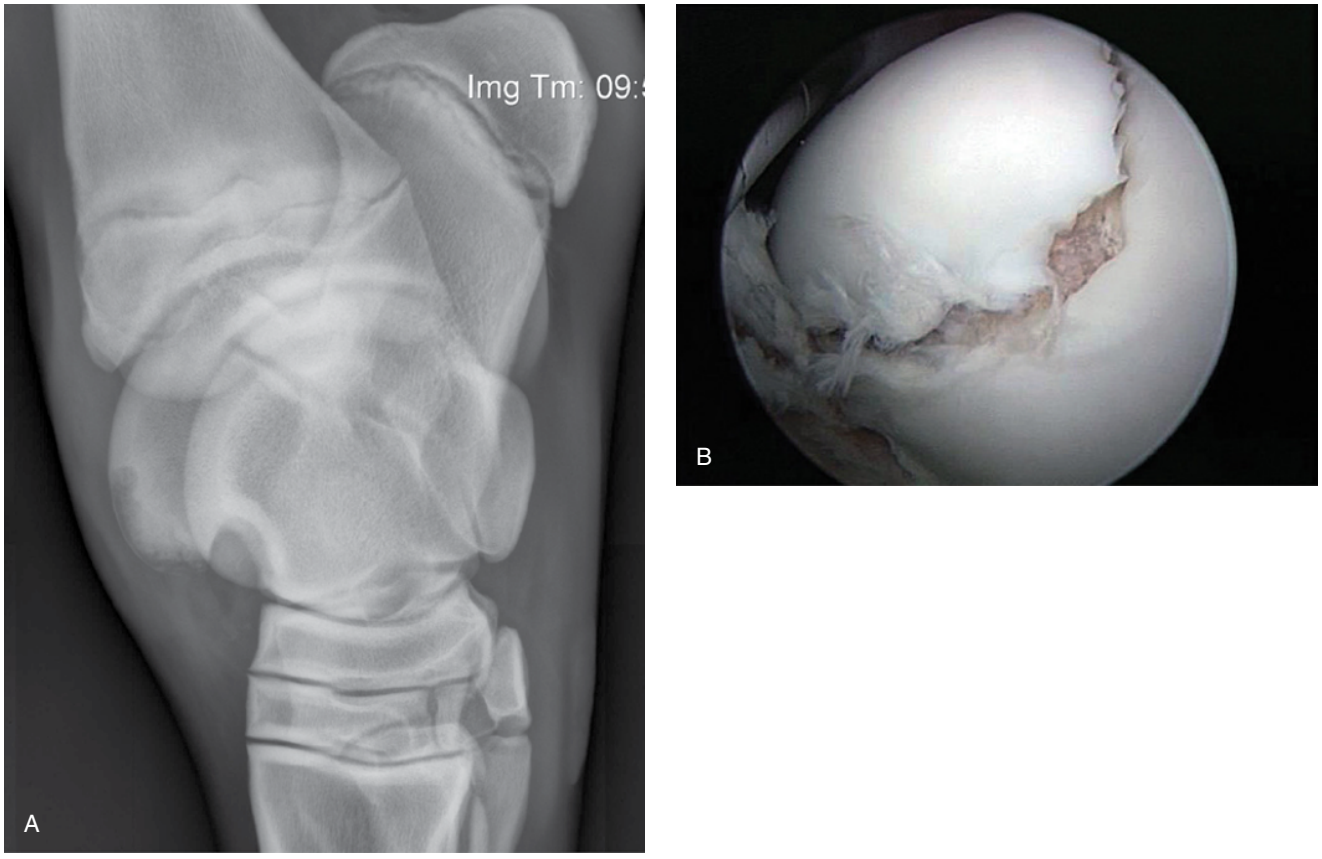


Figure 10.51. Radiographic view (A) and arthroscopic view (B) of OCD of the lateral trochlear ridge of the talus within the tarsocrural joint.

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Table 10.4. Location of OCD lesions in 318 tarsocrural joints.

Number of joints	Location
244	Intermediate ridge (dorsal aspect) of distal tibia
37	Lateral trochlear ridge of talus
12	Medial malleolus (dorsal aspect) of tibia
11	Intermediate ridge of tibia plus lateral trochlear ridge of talus
4	Intermediate ridge plus medial malleolus of tibia
3	Intermediate ridge plus medial trochlear ridge of talus
3	Medial trochlear ridge of talus
3	Lateral trochlear ridge of talus plus medial malleolus of tibia
1	Lateral and medial trochlear ridge of talus
318	Total

Source: McIlwraith et al.[13] Reproduced with permission of Equine Veterinary Journal.

effect on subsequent performance (but can be a cosmetic issue for show horse owners). Further studies have confirmed the success rate with arthroscopic surgery.[1,8]

Osteochondritis Dissecans of the Fetlock Joint

The most common manifestation of OCD in the fetlock joint is fragmentation and irregularity that occurs on the dorsal aspect of the sagittal ridge and the condyles of the metacarpus or metatarsus (cannon bone). A second manifestation involving the fetlock is fragmentation of the dorsal aspect of the proximal phalanx. A third manifestation that has been considered by some to be osteochondrosis related is plantar/palmar fragments of the proximal phalanx. However, the consensus now is that these are avulsion fragments.

Osteochondritis Dissecans of the Dorsal Aspect of the Distal Metacarpus/Metatarsus

Clinical and Radiographic Signs

Joint swelling (effusion) is the most common clinical sign, with lameness variable in both appearance and severity. Fetlock flexion tests are usually positive. It is not unusual for all four fetlocks to be involved, and bilateral forelimb or hindlimb involvement is quite common.

The diagnosis is confirmed on radiographs, and clinically silent lesions (no effusion or baseline lameness) are often identified along with the lesions causing clinical signs. Lameness can sometimes be induced by flexion in these clinically silent joints. A variety of radiographic presentations are seen with fetlock OCD. Some joints show only flattening or a defect in the sagittal ridge (type I OCD), others have a fragment in place within the area of flattening (type II OCD), and others have flattening with or without a fragment in place, but also have free or loose bodies within the joint (type III OCD).[15,30]

Treatment

A conservative approach is initially recommended for type I lesions. Many of these cases have resolution of clinical signs, as well as improvement or disappearance of radiographic signs[12,15]; however, surgery is eventually necessary in a small number of these cases. Surgical debridement is recommended for type II and III lesions, where fragmentation or loose bodies are present.[12]

Prognosis

The prognosis is quite favorable for type I lesions, but more guarded for type II and type III lesions. In one study involving 42 horses, the success rate was approximately 60%.[12] Horses with other signs of articular cartilage erosion or wear lines within the joint had a less favorable prognosis. If the lesion extended onto the condyle of the metacarpus/metatarsus from the sagittal ridge, the prognosis was also less favorable. It was determined that clinical signs would persist in approximately 25% of cases.

OCD Fragments of Proximal Dorsal Aspect of Proximal Phalanx

Joint swelling (effusion) is the most common clinical sign, with lameness variable in both appearance and severity. Quite often these fragments are identified on survey radiographs and are presented for removal. The fragments are usually rounded in appearance and are off the proximal medial eminence of the proximal phalanx. Arthroscopically they show a typical OCD appearance with separated cartilage and defective cartilage underneath.

Osteochondritis Dissecans of the Shoulder Joint

OCD involving the shoulder joint is the most debilitating form of OCD affecting horses. Generally, large areas of the joint surfaces are involved, and secondary joint disease is common. However, it is unusual to have free or loose bodies develop. OCD of the shoulder is less common than for the other joints described and seems to affect Quarter horses and Thoroughbreds with a similar incidence.

Clinical and Radiographic Signs

Most horses with shoulder OCD present at 1 year of age or younger, with a history of forelimb lameness of variable severity. Many of these horses have prominent lameness, and if lameness has been present for many weeks, shoulder muscle atrophy is also seen. Because of the altered gait and use of the limb, many cases develop an upright or club-footed appearance to the foot, and the foot may appear smaller on the affected limb. Deep pressure over the shoulder joint often causes discomfort,

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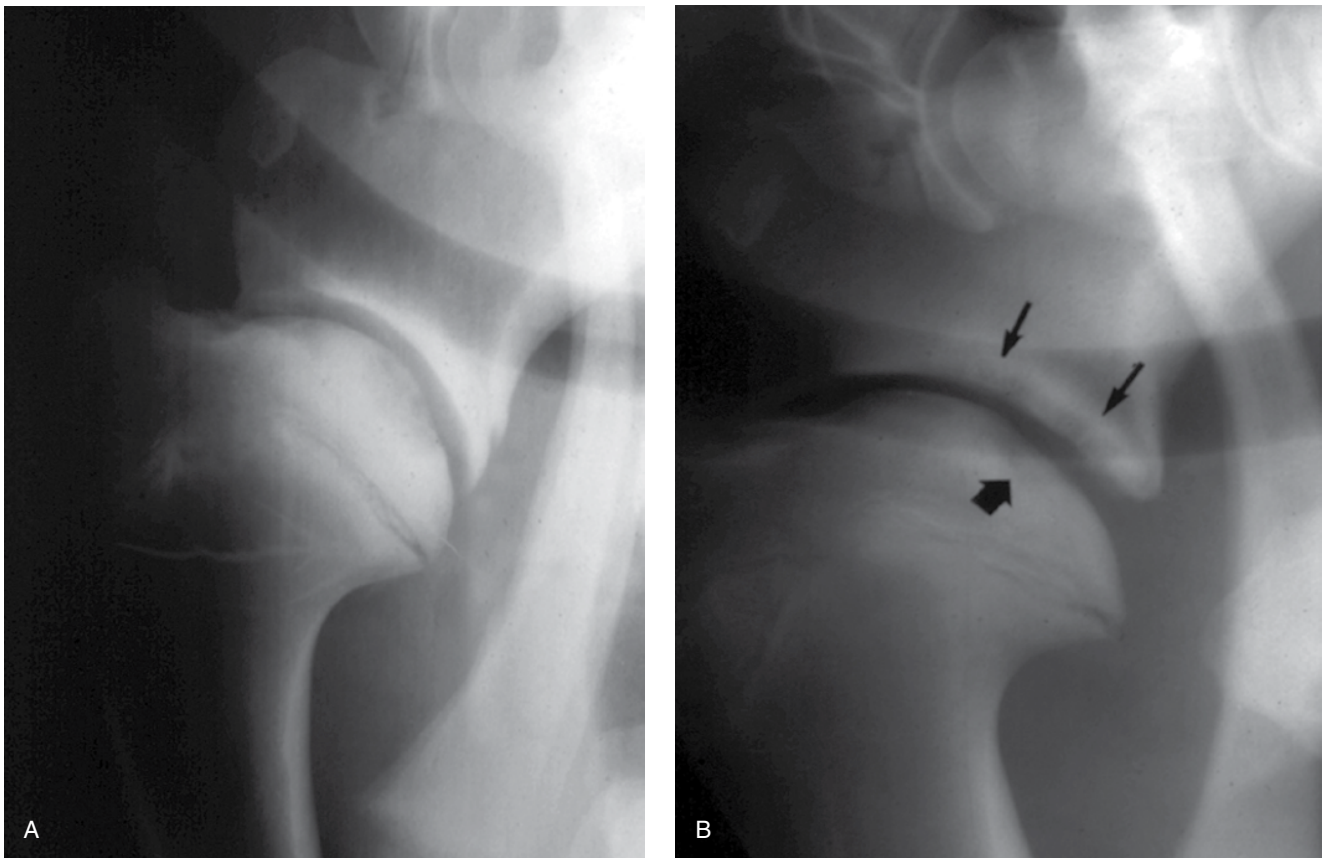


Figure 10.52. Radiographic manifestations of OCD of the shoulder. (A) This defect in the humeral head is the only lesion. (B) Defects in the humeral head (large black arrow) and glenoid of the scapula (2 black arrows).

and forced flexion/extension of the limb sometimes accentuates the lameness that is seen. Intra-articular anesthesia improves or eliminates the lameness.

On radiographs, the most common sign is flattening or indentation of the humeral head (Figure 10.52). Often, defects or cystic-type lesions are also identified in the glenoid cavity of the scapula. Productive remodeling changes are commonly identified along the caudal border of the glenoid cavity.[2]

Treatment

Historically, the author had advocated that conservative treatment is never associated with a successful outcome. More recently it has been recognized that in selected cases the radiographic obvious lesion can resolve without secondary osteoarthritis (OA) with conservative management.[15] The typical case will be localized to the glenoid, which is recognized early, and follow-up radiographs taken regularly to ensure that resolution is occurring. In the majority of cases arthroscopic surgery is necessary to achieve athletic activity, and dramatic improvement in the clinical signs in most affected cases is the norm.[2,15,16] If extensive degenerative arthritic change is present on radiographs at the time of the initial examination, the prognosis for an athletic career is unfavorable, and surgery should only be considered for relative improvement in the degree of lameness. For more localized lesions, the prognosis is favorable for a successful outcome.

The shoulder is probably the most difficult joint on which to perform arthroscopic surgery, due to the depth of the joint below the muscles in the area, and there is a learning curve.[15] Surgery is easier on younger animals due to their relative muscle mass. Problems encountered in the shoulder are inaccessibility of lesions due to their location within the joint and extravasation or leakage of fluid outside the joint, which impairs visibility within the joint.

Prognosis

A large series of cases having surgery for shoulder OCD has not yet been reported, although preliminary results from 80 cases compiled at Colorado State University suggest that the overall prognosis is approximately 50%. The prognosis seems to be less favorable if lesions are present on both the humeral head and the glenoid cavity. In unsuccessful cases, further deterioration of the joint surfaces on subsequent radiographs is common.

SUBCHONDRAL CYSTIC LESIONS

SCLs have been mentioned previously as a manifestation of osteochondrosis, at least in young horses. The most common location for clinically significant SCLs is within the medial condyle of the femur in the medial femorotibial joint (Figures 10.53 and 7.17). Other areas

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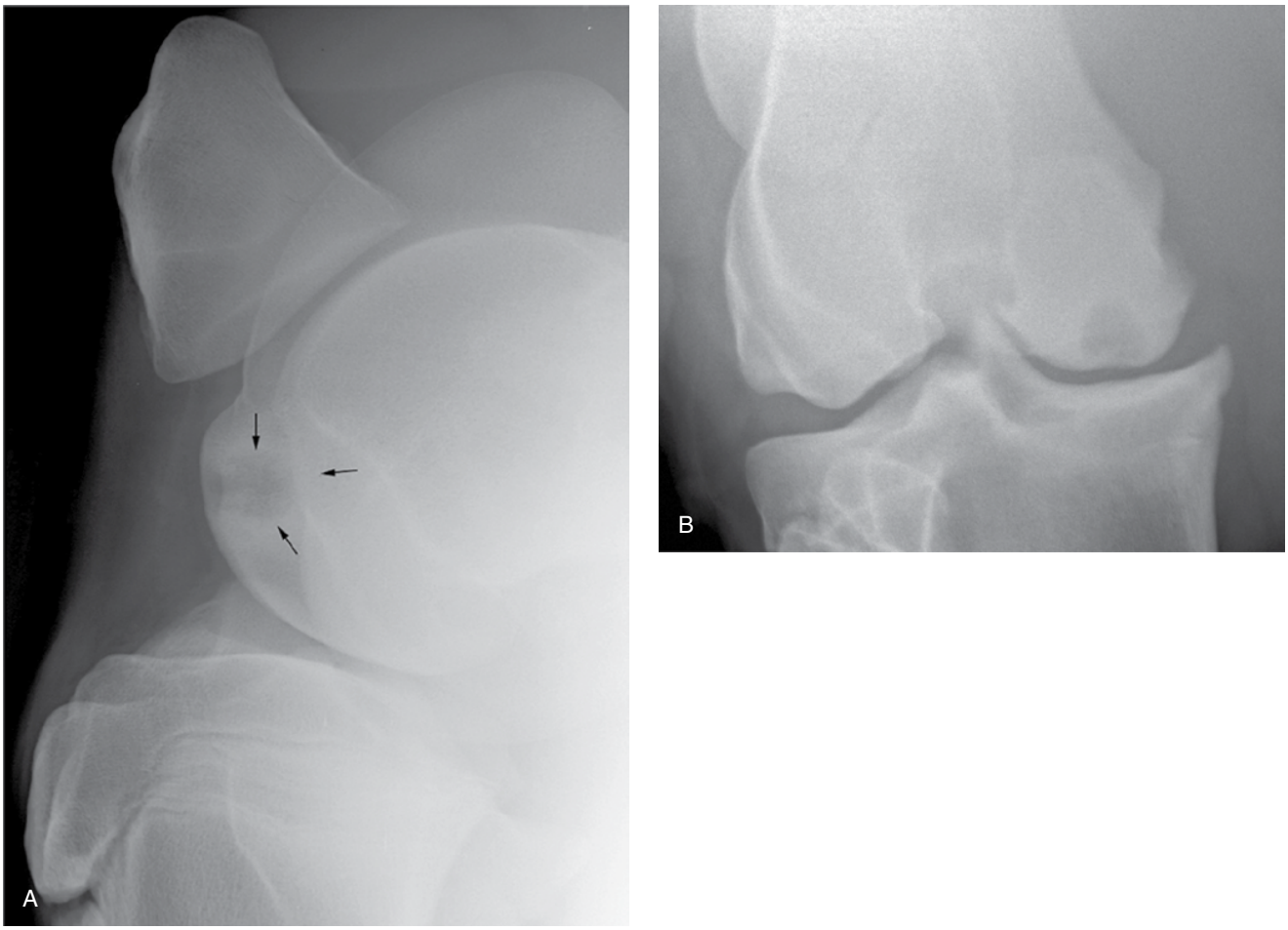


Figure 10.53. (A) Flexed lateral–medial radiograph and (B) caudal–cranial radiograph of subchondral cystic lesions (arrows in A) of the medial femoral condyle of the femur.

of importance are the distal metacarpus or metatarsus in the fetlock joint, the distal proximal phalanx in the proximal interphalangeal joint, and shoulder, elbow, carpus, distal phalanx, and talus. Principles of diagnosis and treatment will be restricted here to the most common entity, the medial condyle of the femur.

With regard to radiographic appearance and classification of SCLs of the MFC, there has been an evolution. Initially there were two types: type 1 was a radiographically domed-shaped lucent area that was confluent with a flattened joint surface, and type 2 had a circular lucent area within the condyle with a thinner radiolucent track connecting the cyst to the articular surface of the condyle (the typical manifestation as seen in Figure 10.53).[29] This early classification evolved into type 1, 10 mm or less in depth, type 2 more than 10 mm in depth, and type 3 flattened or irregular contours of the subchondral bone. More recently five different types were described by Wallace et al. in 2008 (Figure 5.127).[27] Type 1 lesions were defined as being concave and smaller than 10 mm in depth and were usually dome shaped. Types 2A and 2B were typical cystic lesions over 10 mm in depth, and the width of the surface communication was the only difference. Type 3 lesions (noted incidentally on survey radiographs of yearlings) were defined as condylar flattening or small defects in the subchondral bone usually noted in the limb contralateral to that of the clinically significant SCL, and type 4 lesions were defined as those that had a lucency in the condyle with or without an articular defect but no radiographic evidence of an opening in the subchondral bone plate (less common). Digital radiographs and surveys have led to increased scrutiny of radiographs, and type 1 lesions have now evolved into two sizes of concave defects as well as including flattening of the condyle.

The decision for surgery based on the authors' experience is based on the presence or absence of clinical signs. Most cases with clinical signs referable to the medial femoral tibia joint when there is an SCL present are typical SCLs (type 2 under the Wallace classification). Type 1 lesions are occasional associated with clinical signs and treated with arthroscopic surgery as well. Historically, SCL lesions in the medial femoral condyle have been treated with curettage,[5,24,29] but this technique is quite rarely done now. Based on recent agreement to a treatment algorithm,[14] it had arthroscopic surgery. If the SCL is found to have stable margins, the treatment of choice is intralesional injection of triamcinolone acetonide into the contents of the cystic lesion.[27] This technique was developed based on work previously cited by von Rechenberg et al.,[20] in which the lining of SCLs produced increased levels of PGE_2, MMPs, and NO, as well

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as enhanced osteoclastic resorption activity. Recently published results have shown a 90% success rate with unilateral SCLs and 67% success rate with bilateral SCLs. Some instances of SCLs (other than type 2) have been treated with debridement, but intralesional therapy with triamcinolone acetonide is attempted in the first instance and any debridement is limited to a loose flap associated with the SCL defect.

Most recently a technique of lag screw fixation across the cystic lesion has been described.[23] Promising results have been reported. The author has done a number of cases with this technique after encouraging results were reported in this paper and favorable results have been experienced by other surgeons including the author.

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LAMENESS IN FOALS

Robert J. Hunt

Lameness in foals is common and may be recognized shortly after birth or at any time during development. The economic impact of foal lameness in the equine industry is tremendous. With proper treatment the outcome is usually favorable, but with improper management, the consequences may be disastrous and result in permanent debilitation, compromised economic value, or loss of the animal. Even with proper clinical management, some diseases are destined to clinical failure; it is important for the clinician to recognize these to avoid prolonged and unnecessary suffering. This chapter focuses on foals up to approximately 6 months of age.

By nature of their physical makeup, behavior, and environment, foals are particularly vulnerable to events resulting in lameness. There are multiple causes of lameness, including infectious and noninfectious etiologies. Several clinical entities are unique to foals and will be addressed. Because of the often rapid clinical progression of lameness with potentially life-threatening consequences, timely recognition along with accurate diagnosis and treatment is critical for a favorable outcome.

DIAGNOSIS

The correct diagnosis of lameness in foals begins with an accurate history, signalment including age, sex, and breed of the foal, and knowledge of the housing and husbandry practices of the farm. Also relevant is an understanding of the turnout schedules and environment, in addition to recent changes in the paddock size or the duration of turnout. Observation of the behavior of the mare and foal and herd size and temperament is important for assessing risk factors for injury to the foal. The medical history of the lame foal as well as others on the farm is vital for determining the possibility of exposure to pathogens. Information should be obtained about the mare's general condition, the presence of any medical or reproductive problems during pregnancy or parturition, and possible insufficient immunoglobulin transfer to the foal. The presence or absence of any febrile episodes and any alterations in the hemogram are important, and evidence of any infectious disease on the premises and equine traffic on the farm should also be noted.

There are specific age ranges in which the foal is most susceptible to certain disorders. For example, lameness in the post-foaling period is commonly associated with trauma during foaling or *in utero* sepsis, resulting in early-onset septic arthritis/osteomyelitis. Excessive traction during delivery of the foal may result in musculoskeletal injury involving bone or soft tissue of the extremity or injury to the ribs. Other examples are illustrated throughout the chapter in their respective areas.

The physical evaluation is the primary means of isolating lameness. A comprehensive evaluation of the entire foal should be performed to determine its overall condition and development compared with other foals on the premises. Visual observation, physical evaluation, and locomotor assessment are utilized in locating, defining, and isolating lameness and obtaining a preliminary diagnosis. Observing the foal at rest and unrestrained in the stall will allow assessment of the demeanor of the foal and allows manifestation of any subtle postural changes and gate alterations. Observation of the restrained foal may be difficult if the foal is not acclimated to a handler and it may be necessary to assess locomotion following the mare. Most present-day breeding operations handle foals at an early age to allow assessment of the foal walking while restrained. With the foal walking in a straight line, careful attention should be given to stride length, foot and limb flight pattern and placement, head and neck carriage, height of the shoulders and elbows, and symmetry across the hips as well as any tail deviation. All articular structures and all regions of the limbs and the trunk should be carefully inspected for swelling or alterations in contour.

Routine palpation of the foal should begin with gaining familiarity with the foal by rubbing and allowing contact with the foal so it is relaxed for the examination. If the rectal temperature has not yet been obtained, it should be performed. Limb palpation should commence with a normal limb to avoid confusion between a behavioral and a pain response. Unless there is an obvious area of pathology, a complete palpation should begin at the foot and extend proximally. Inspection of the foot includes observing for changes in the shape and integrity of the capsule, the presence of wall cracks, wall separation, or sole cracks. The digital pulse should be carefully assessed for intensity and compared to the opposite limb. The coronary band should be palpated thoroughly for any swelling or regions of sensitivity possibly indicating sepsis. Circumferential swelling of the coronary band is commonly associated with advanced sepsis of the distal interphalangeal joint that often requires prompt treatment. Palpation of the hoof wall includes detection of focal regions of temperature elevation or reduction. Hoof compression with a hand placing pressure across the heels will detect heel pain commonly observed with wing fractures of the distal phalanx. Commercial hoof testers may also be used here and on the rest of the foot. A painful response at the toe is commonly associated with bruising, sepsis, or fracture. In foals less the 1 month of age, abscess of the toe along with a type VI distal phalangeal fracture is common, especially in the hindfeet.

Advancing proximally the fetlock should be assessed for articular or periarticular swelling and surface temperature changes or areas of increased sensitivity to

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digital compression and flexion or manipulation of the joint. The surfaces of the proximal sesamoid bones during extension and flexion are palpated for sensitive areas. Sequential and methodical palpation of the entire limb including surfaces of the articular as well as nonarticular structures should be routinely performed. Most articular structures affected with sepsis yield a response of the foal with digital compression of the joint margins and should raise suspicions of the clinician. Likewise, focal areas of injury typically produce a pain response to digital compression and warrant further investigation with imaging procedures. Deep palpation of the proximal forelimb, vertebral column, proximal hindlimbs, and pelvis should be a routine part of the examination. Each compartment of the stifle should be isolated and palpated for effusion, pain, and temperature alteration. The entire rib cage should be carefully examined especially in the young foal that may have sustained rib fracture with subsequent forelimb lameness.

Isolation of the lame limb and the specific origin of pain may be challenging if subtle, but in general foals readily display locomotor evidence of the location of pain. Most foals attempt to avoid the painful component of the gait and usually display more exaggerated lameness reactions than adults. Observation of the young foal at rest and unrestrained in the company of the mare may be useful in detecting mild gait alterations or postural changes. Special attention should be given to the stance of the foal, particularly any trends in abnormal posture. Young, untrained foals may require gait evaluation while unrestrained and following the mare. The gait should be assessed at a slow walk in a straight line and in turns in both directions. Placement of the foot as well as limb and body carriage during ambulation is important in localizing lameness. Although there are some common findings with certain conditions (see below), there are few absolutes regarding gait characteristics and location of lameness in foals. Conventional lameness diagnostic anesthesia is often necessary to isolate the location of the source of lameness.

NONINFECTIOUS CAUSES OF LAMENESS

There are a multitude of events that may affect any part of the musculoskeletal system resulting in lameness; several disorders are unique to foals and occur routinely. Categorically lameness may be divided into infectious or noninfectious causes. Noninfectious causes include trauma, metabolic (DOD), or afflictions of vascular or neurogenic origin. For discussion purposes disorders causing lameness will be mentioned by anatomical region involved.

Virtually all components of the musculoskeletal system are susceptible to injury and lameness. Intrinsic and external sources of trauma commonly result in structural compromise to tissue of the musculoskeletal system. Other conditions resulting in lameness include those associated with DOD and disorders of vascular or neurogenic origin. Several disorders specific to foals occur routinely and are worth mention.

Foot Lameness

Distal phalangeal fractures in horses have been classified into seven types.[10,40] Although any of these may occur in foals, type VII fractures are unique to foals and occur very commonly on Thoroughbred breeding operations. There is a reported prevalence of 19%–74% that are recognized predominantly between 2 weeks and 5 months of age.[5,13,48] Fractures involve the nonarticular margin dorsal to the palmar/plantar process. They have been described as ossicles or osseous bodies but are commonly referred to as "wing" fractures.[25] The lateral palmar process is almost always involved, but occasionally they occur biaxial or bilateral; seldom is only the medial wing involved. Although factors such as hard or soft surfaces, changes in turnout schedules, and hoof conformation and application of external hoof acrylics are incriminated, no single factor has been substantiated as causative.[13,14] Because many type VII fractures are recognized as incidental findings, they have been considered separate centers of ossification; however this has not been documented.[40]

The degree of lameness associated with type VII fractures varies from non-apparent to non-weight-bearing but generally display a 3/5 lameness acutely that improves in a matter of days. While ambulating in a straight line, the foal will display a characteristic abduction of the limb during contact with the ground, presumably to reduce loading on the painful region. The lameness is more exaggerated on turns as well.

There is usually a palpable increase in the intensity of the digital pulse, but only a mild increase in the temperature of the foot. The most consistent clinical finding is an increase in reaction to medial-to-lateral compression of the hoof wall at the quarters and heels (Figure 10.54). Palmar digital anesthesia of the involved side will eliminate the lameness. The diagnosis is confirmed radiographically using the 45° dorsal–ventral



Figure 10.54. Manipulation of the foot to locate the focus of pain in a lameness examination in a foal.

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Figure 10.55. Forty five degrees dorsoproximal palmarodistal radiograph showing a type VII fracture of the coffin bone.



Figure 10.56. Forty five degrees dorsoproximal palmarodistal radiograph showing septic osteitis of the third phalanx. There is a radiolucent area on the medial aspect of the solar margin (arrow).

proximolateral to distal–medial oblique projections, although fractures may be seen on the lateral–medial, horizontal dorsal–palmar, and 45° dorsal–ventral projections (Figure 10.55).

Treatment for type VII fractures of the coffin bone is conservative and consists of confinement to a small area on soft, uniform bedding for 2–3 weeks or until the horse is comfortable enough for paddock turnout.[48] Rigid coaptation of the foot with a shoe is unnecessary but has become popular. Two complications of shoe application include creating limb length discrepancy resulting in the contralateral limb overload and possibly contracting the hoof capsule if the shoe remains long. Improvement in lameness is usually seen with application of a soft foot bandage, which may be changed daily or every other day for 2–3 weeks. The prognosis for future soundness is good; rarely are there any long-term ramifications or gait deficits. Differentials include other types of coffin bone fracture, foot abscess or bruising, septic osteitis, and a septic coffin or pastern joint.

Subsolar abscess with or without involvement of the coffin bone is common. It is discussed here because it is generally associated with trauma rather than hematogenous. Lameness may be recognized as early as 3–5 days of age or older. In the young foal, the hindfeet are more commonly involved, and the abscess likely begins with bruising and subsolar hematoma beneath the soft keratinized sole that is present during the first few days of life. It is common for the abscess to involve a large portion of the subsolar tissue or dissect proximally up the dorsal hoof wall or involve the coffin bone (Figure 10.56) with possible sequestration.[2,37] Subsolar abscess generally causes a severe non-weight-bearing or minimally weight-bearing gait. Treatment includes draining purulent material and protecting the foot.

It is common to for abscesses to involve the solar margin of the distal phalanx (type VI fracture), which often requires surgical curettage if sepsis is present.[34–37] Regional perfusion with antimicrobials is frequently performed and clinically appears to be beneficial. Broad-spectrum systemic antimicrobials are routinely administered once drainage is established, or in refractory cases, or if surgical curettage is necessary.

It is not uncommon for the coffin joint to become secondarily involved through extension; arthrocentesis and copious joint lavage are indicated in addition to the above treatments. Generally, with joint involvement subsequent to hoof abscess, there is a guarded to poor prognosis because this is often associated with an aggressive and rapidly spreading infection.

Proximal Sesamoid Bone

Fracture of the proximal sesamoid bones is another common lameness condition on commercial breeding farms. These are most commonly seen in foals younger than 6–8 weeks of age; they are often associated with overexertion from running.[11] Essentially all configurations of proximal sesamoid bone fracture described in the literature occur in foals, but the most common types are apical and basilar fractures with a smaller number having midbody or comminuted fractures (Figure 10.57). It is common for the fractures to be unilateral and involve only one sesamoid, or to be bilateral and uniaxial, although sometimes they are biaxial.

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Figure 10.57. (A) Lateromedial radiographic view of a nondisplaced apical sesamoid fracture. (B) Dorsomedial to palmarolateral radiographic view of a displaced basilar sesamoid fracture. (C) Dorsomedial to palmarolateral radiographic view of a mildly displaced medial basilar sesamoid fracture in a foal at time of injury. (D) Radiograph of the same foal shown in C at 16 months of age after healing.

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The degree of lameness varies from mild and perceptible only on turns to non-weight-bearing. Gross instability of the suspensory apparatus resulting in a dropped fetlock only occurs with biaxial fractures of the body with extreme distraction. Foals are at the highest risk for biaxial fractures when turned out to a paddock after being confined for a period of time.[40] Depending on the fracture configuration, there may be a soft tissue profile over the involved sesamoid. There is often joint distension, especially with the basilar sesamoid fractures. Fetlock flexion and direct manual pressure applied over the involved sesamoid usually elicits pain.[11] The diagnosis is confirmed by radiographs and may be facilitated by ultrasound evaluation.

Treatment for proximal sesamoid bone fractures in foals is conservative and consists of confinement to a stall-sized area for 4–8 weeks, followed by a graduated turnout schedule. The prognosis for future athleticism is good, but the sesamoid commonly heals in an elongated manner, which is often criticized if the horse is sold at public auction.

Only in rare instances is external coaptation with a cast or splint indicated.[40] Most devices are contraindicated for fear of promoting laxity of the flexor tendons. Likewise, surgical repair is usually futile and applies to salvage cases with total compromise of the suspensory apparatus that have marked distraction of the fracture fragments and dropping of the fetlock. The logical surgical technique is circumferential wiring or suturing of the fractured sesamoid bones. External coaptation and intensive rehabilitation to allow gradual loading of the limb are necessary. Irrespective of treatment, after severe separation, the sesamoid bones remodel in a distorted and deformed configuration, and an athletic and sale future is unlikely. Alternatively, surgical arthrodesis of the fetlock may be performed to achieve comfort; however, this is considered only for salvage and not for athletic use.

Carpus and Tarsus

Lameness associated with the carpus is usually from trauma or sepsis. Trauma may be associated with compression injury, and dysmature neonates are at higher risk. Chip fracture also may occur as a result of trauma and is often accompanied by soft tissue trauma. There are almost always outward physical signs of carpal involvement, such as pain on manipulation and palpation, heat, joint effusion, and swelling of periarticular tissue. Most carpal injuries involving cartilage, bone, and intra-articular soft tissue carry a guarded prognosis, and management depends on the type of injury. Displaced fragments that are unstable require removal and have a poor prognosis depending on the degree of articular involvement.

Tarsal lameness may be associated with wedging or crushing of the cuboidal bones as a result of dysmaturity (Figure 10.58).[8,9] Accompanying clinical features include a boxy appearance of the distal tarsus when viewed from the front and a curby or sickle-hocked appearance when viewed from the side. Radiographs should be performed on all dysmature foals to establish a baseline of the degree of mineralization of the cuboidal bones as well as the shape of the bones. Premature and dysmature foals are normally confined to a stall until 6–8 weeks of age or until acceptable mineralization has occurred to minimize wedging or crushing. Tarsal wedging or crushing usually does not cause lameness in the neonate. Gait abnormalities are recognized only after periods of exertion following turnout with other foals or after athletic activity begins. If lameness is present in the neonate with a lack of mineralization of cuboidal bones, septic osteomyelitis must be ruled out.



Figure 10.58. Lateromedial radiographic view of the tarsus of a foal showing collapse of the third tarsal bone.

Common injuries of the tarsocrural joint result in fracture of the distal lateral trochlear ridge of the talus and malleolar trauma with or without fracture. Marked synovial effusion is associated with these injuries, and the diagnosis is confirmed radiographically. If there is displacement of the fragment, surgical removal is the treatment of choice and the prognosis is favorable.

Soft tissue injury is common. A decubital wound of the lateral aspect of the tarsus overlying the lateral digital extensor tendon is the most frequently observed condition. This injury often occurs in young foals less than 2–3 weeks of age that have spent significant time in lateral recumbency as neonates, but it may be recognized in clinically normal foals as well. The condition causes mild lameness unless secondary sepsis extends into deeper bony structures or the tendon sheath, in which case lameness may be severe. Once the tissue opens and begins draining, the lameness resolves. There is a good prognosis with proper conservative wound management and good hygiene. These wounds should be allowed to heal by second intention, which may take weeks to months.

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Elbow and Shoulder

Lameness of the elbow may be associated with trauma or infection. The presenting appearance may range from a subtle shortening of the gait to the classic dropped elbow recognized with olecranon fracture and disruption of the triceps apparatus (Figure 10.59).

Lameness involving the shoulder region of the foal is common and may be of septic or traumatic origin. It is not unusual to have two foals present simultaneously with shoulder injuries from colliding with one another while running head on. They may also undergo collision with a tree or sustain a kick injury. Many collision injuries result in nerve paralysis involving the radial nerve or brachial plexus, whereas kick injuries commonly cause fracture of the supraglenoid tubercle or other portions of the scapula.

Differentiating between nerve injury and fracture can be accomplished with physical exam and radiographs. With nerve injury, the foals are not painful on palpation and manipulation, the shoulder is dramatically dropped, and the limb may be physically placed and maintained in normal position while applying pressure to the dorsal aspect of the carpus. There is usually less swelling over the point of the shoulder with nerve injury than with fracture. Radiographs are normal with nerve injury; however, they may be confusing to interpret because of the physes. Likewise, if a supraglenoid tubercle physeal fracture is initially nondisplaced, it may go undetected. Follow-up radiographs in several days to weeks may be necessary for an accurate diagnosis.

Prognosis for nerve injury is guarded, depending on the extent of injury. Foals with neuropraxia improve in a matter of hours to days, although it may take weeks to return to normal. There is often some degree of muscle atrophy that gradually improves. Ruptured and avulsed nerves do not improve, and these cases may undergo prolonged treatment attempts before the involved limb develops severe contraction or there is breakdown of the contralateral limb. The prognosis for supraglenoid tubercle fracture is good for survival, pasture soundness, and light use, but poor for athletic use. Surgical removal of the fragmented tubercle and open reduction with internal fixation have been performed, but there is little difference between aggressive and conservative management in the long-term outcome regarding athletic potential of these animals.

Stifle

Lameness of the stifle in foals is commonly septic in nature; traumatic injuries and developmental orthopedic disease (DOD) of the stifle in foals occur less frequently. The proximal tibial physis is subject to fracture in a predictable configuration, which produces a Salter-Harris type II fracture.[3] There is frequently accompanying soft tissue trauma that precludes athletic soundness after reduction and surgical repair, but these animals are most often sound for breeding as adults. Other traumatic injuries of the stifle are usually severe and result in intra-articular soft tissue injury involving the cruciate ligaments, collateral ligaments and menisci, or severe bone injury, necessitating destruction.

Pelvis

Injuries of the pelvic region are common and may be difficult to diagnose with great specificity because they



Figure 10.59. Typical dropped elbow appearance seen in fractures of the humerus and olecranon. Notice that the limb is also abducted and held in slight flexion.

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may involve bone, cartilage, muscle, tendon and ligament, nerves, and blood vessels. There is no age limitation on pelvic injuries, which may occur as early as a few days or at any time later; however, most traumatic and septic causes of pelvic lameness occur prior to 4 months of age.[23] Traumatic injuries of the pelvis and coxofemoral joint may result from rearing, twisting, and falling over, with torsional load causing the injury. Tuber coxae injuries are typically associated with external trauma such as a kick from another horse or striking the area during a fall or against a gate post or other fixed object. Injuries of the tuber ischii may occur from falling backward or accelerating backward and directly striking or landing on this bony prominence.

The gait and degree of lameness depends on the structures involved and their location. Fractures of the pelvis almost always involve both sides, with one side more extensively damaged than the other. Displaced pelvic fractures are generally non-weight-bearing lame during ambulation on the side with the most damage. When standing, however, foals generally bear weight on both limbs and may electively stand on the primary fracture side. Generally, the tail is set away from the most severely affected side. If the injury is caudal to the coxofemoral joint, the caudal phase of the stride is prolonged and there is resentment during the cranial phase, whereas the opposite is true for injuries of the tuber coxae. Foals with injuries to the superficial bony prominences such as the tuber coxae, tuber ischii, or greater trochanter of the femur as well as injury to the coxofemoral joint display a pain response on digital palpation. Deeper injuries of the pelvic floor or pubic symphysis may be more difficult to detect on physical palpation.

Radiographs are necessary to confirm the presence of a fracture, and ultrasound may facilitate identification of other injured tissue. Nuclear scintigraphy is occasionally used to evaluate pelvic injuries and can effectively identify regions of injury, but does not allow identification of specific fracture types. Injuries of the physes and accompanying soft tissue are more effectively identified by magnetic resonance imaging.

Treatment for pelvic injury includes stall rest, and the duration depends upon the severity of the injury.[30] Once full weight-bearing returns, controlled exercise in the form of hand walking is beneficial for rehabilitation. The prognosis for pelvic injury depends upon severity. In general, the prognosis is favorable, but severe displacement accompanied by marked soft tissue trauma or significant involvement of the coxofemoral joint carries a poor prognosis. The main differentials for pelvic trauma are septic arthritis, osteomyelitis, septic physitis, and pelvic abscess.

Less severe lameness of the hip region is associated with tearing of the fascia of the lateral aspect of the biceps femoris or the vastus lateralis muscle. These injuries produce a transient lameness that manifests as a shortened gait at a walk. A palpable 3- to 5-cm deficit, oriented obliquely and parallel to the muscle fibers, is present, and an associated hematoma may be observed. Treatment is rest and confinement for 1–2 weeks to allow any hemorrhage to stop. These tears should not be drained or opened for fear of sepsis. Rarely, if ever, do any long-term residual effects of these tears manifest, and future athletic potential should not be affected.

Physeal Fractures

Physeal fractures are common due to the relatively weak physeal bone compared with the diaphyseal bone. Diagnosis of physeal injuries is made on clinical and radiographic findings. Clinical signs vary depending on the degree of instability; they include lameness of varying degrees, pain on palpation, swelling, and occasionally angular deviation of the limb distal to the fracture. The primary differential for a stable physeal fracture is infectious physitis. Fracture of pressure physes (those that contribute to longitudinal bone growth) have been categorized and defined using the Salter-Harris classification scheme, covered elsewhere in the text.[3,6,12] Surgical repair is necessary in foals with displaced, unstable physeal fractures.[29] If adequate reduction and stabilization are achieved, the prognosis for future soundness is good.

Long Bone Fractures

Long bone fractures usually result from external trauma such as a kick or having the limb pinned beneath an object while rolling. Rearing and misloading a limb may result in fracture of the tibia, femur, or pelvis.[21,23] Physical evaluation may be diagnostic in foals with lameness of traumatic origin because unstable fractures or severe soft tissue injuries cause obvious, immediate clinical signs.[47] If a fracture is suspected, radiographs are necessary to confirm the diagnosis and determine the severity of the injury. The prognosis for a foal after fracture depends on the anatomical location of the fracture; the complexity, orientation, and degree of soft tissue injury around the fracture; and whether the fracture is open.[17,21] Secondary problems associated with contralateral limb overload from prolonged lameness or from immobilization often dictate the degree of long-term soundness.

Although mechanical laminitis is not as common as in adult horses, it occurs in foals. More common problems include excessive laxity of the metacarpophalangeal or metatarsophalangeal joint, varus deviation, or deformity of the foot, such as underslung or crushed heels and overgrowth of the toe. Contracture or excessive laxity of the fractured limb associated with disuse may prevent future soundness.

Developmental Orthopedic Disease

Lameness may be associated with DOD in foals.[7,39] In a group of 378 foals evaluated radiographically at 6 months of age, 47% were affected by DOD and 36% by osteochondrosis with some evidence showing Warmbloods may be at higher risk.[26] Although osteochondrosis does not typically manifest until the late weanling and yearling period, it is occasionally recognized clinically as a cause of lameness in foals less than 6 months of age.[40] The development of DOD is multifactorial, but there may be some association with exercise conditions and rapid growth.[27] See earlier sections of this chapter for specific DOD conditions.

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Perinatal and Congenital Conditions

Foals may be born with musculoskeletal conditions that result in lameness or they may develop them shortly after birth. These conditions are relatively infrequent but worthy of mention because historically they have either been misdiagnosed or erroneously given a poor prognosis. As a result, foals that might have matured and had normal athletic potential have been humanely destroyed. Lameness may be directly caused by these conditions or it may be secondary to their occurrence.

Two such conditions of the neonate are flexural limb deformities and excessive laxity of flexor tendons, both of which are relatively common and may result in secondary lameness. Flexural limb deformity may produce mechanical limitations on ambulation, or it may cause pain associated with attempts to correct the deformity. The most common cause of lameness from flexural deformity is excessive trauma to the sole of the foot at the toe region, which causes bruising and abscess formation.

Flexor tendon laxity causes lameness from decubital sores, bruising, and abscess in the heel bulbs of the hindfeet. These may be prevented by lightly bandaging the heel bulbs for protection and preventing luxation of the distal limb by applying extended heels.

Certain structures in the neonate and young foal are prone to rupture, including that of the common and lateral digital extensor tendons of the forelimb, which usually occurs within 72 hours of birth. Foals with mild flexural deformities may be predisposed to develop these problems. In these contracted individuals, the condition may develop at 2–3 weeks of age. The predominant clinical sign is knuckling forward on the ankle and carpus and initially failure to advance the limb. This pathology is frequently mistaken for flexural limb deformity.

The two conditions may be differentiated with physical evaluation. Lateral and common digital extensor tendon rupture may be recognized by the presence of a characteristic swelling over the dorsal lateral surface of the carpus at the musculotendinous junction where the rupture occurs. The limb also may readily be placed in a normal position, whereas this is not possible in foals with flexural deformity.

Treatment is aimed at protecting the limb from excoriation of the dorsum of the fetlock with bandaging until the foal acclimates to walking. Continuous splinting or casting is not usually necessary but may be applied intermittently to facilitate use of the limb. Surgery is not necessary or advisable because the tendons generally heal uneventfully over several weeks.

Ruptured gastrocnemius muscle is recognized in neonates following both normal deliveries and dystocias, but larger foals appear to be predisposed to this condition.[24] Rupture may be partial or total. With partial rupture there is moderate flexion of the tarsus, but the foal can still bear weight on the limb. With a complete tear the foal is unable to extend the limb and will collapse with weight-bearing, and the excessive flexion of the hock with the stifle in a fixed position results in a typical, crouched position on the affected side (Figure 10.60). Prognosis for future soundness is good with conservative treatment, providing the foal is not compromised with other concomitant disease processes.[24,44] Splinting is usually required, and bandages must be changed frequently to prevent focal pressure necrosis, adequately support the limb, and support ambulation while healing occurs.



Figure 10.60. Excessive flexion of the hock in a foal with rupture of the left gastrocnemius muscle.

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INFECTIOUS CAUSES OF LAMENESS

Lameness in foals caused by infectious agents is extremely common, so much so that it is a safe to assume that all lameness in foals is septic in origin until proven otherwise. Foals may be affected shortly after birth or up to 4 months of age, although such lameness may sporadically occur in older foals; approximately half the foals are affected by 60 days of age.[36] It is perplexing that foals appear to be plagued with these conditions more than other species with similar housing and hygiene practices.

The most common infectious conditions causing lameness include the complex of osteomyelitis, septic arthritis, septic physitis, and/or septic tenosynovitis.[15,18,19,32–34,41,45] Although there are numerous pathways by which these conditions may occur, such as external injury, the most common is believed to be hematogenous spread from a primary source.[20] Such sources include the urachus or urachal remnants, gastrointestinal tract, respiratory tract, or a distant site in the musculoskeletal system.[22,36] It is commonly thought that there is a propensity for bacterial colonization in the extensive vascular plexus composed of venous sinusoids, metaphyseal loops, and epiphyseal, metaphyseal, and physeal vessels in growing long bones. The slow and sluggish blood flow and relative vascular stasis in this region allow bacteria to proliferate at or close to the cartilage interface.[6,15] Cartilage matrix degradation and proteoglycan loss may occur within 2 days, and loss of collagen within 9 days after inoculation by bacteria.[4,6,35] The chance of infection is increased in high-risk individuals such as a foal with failure of passive transfer or exposure of a naïve immune system to an aggressive pathogen.

Hematogenous osteomyelitis and septic arthritis have been classified into five types based on the structure involved and location.[15,46] Infectious synovitis, or S-type, most commonly affects younger (under 2 weeks) foals and involves only the synovial structure. Epiphyseal type (E-type) involves the joint and adjacent epiphysis. Involvement of subchondral bone results in eventual radiographic changes, although these may not be evident for a week or more. P-type involves the physis and is seen in foals 1 week to 4 months of age. Lesions are seen radiographically as lytic changes in the physis, metaphysis, or epiphysis.[19] T-type involves the tarsal or carpal cuboidal bones and may result in collapse of these structures. These most commonly occur in neonates that suffer from dysmaturity, although normal foals may be affected. I-type involves periarticular soft tissue abscess infecting a physis or joint and often involves the hip or stifle, or more commonly the distal metacarpus or metatarsus. Foals older than 6–8 weeks are most often involved. The clinical appearance of the involved region is diffuse pitting edema, with eventual abscess development and drainage.

The common presenting complaint of osteomyelitis and/or septic arthritis is acute onset of lameness, and there is often a pyrexic episode at presentation or in the recent past. The degree of lameness varies from mild to non-weight-bearing and largely depends on the severity of the infection and structures involved. The foal may be noted in recumbent positions more often than normal. With joint involvement there is synovial distension as opposed to the diffuse swelling typically seen with osteomyelitis. The region is usually warm to hot and is painful on manipulation. Occasionally, the area may be cold if the swelling is excessive enough to create vascular compromise, or there may be vascular thrombosis associated with the sepsis.

Hemogram is often normal on presentation because of the localized and acute nature of the infection. When hemogram alterations occur, there is an elevation in the white blood cell count, with or without a left shift and an increase in fibrinogen of 500–800 mg/dL or higher. Plasma fibrinogen concentration of more than 900 mg/dL may be useful as an indicator of physeal or epiphyseal osteomyelitis, and values may reach 1500 mg/dL.[38] Serum amyloid A (SAA) has more recently proven beneficial as a marker to identify inflammation associated with sepsis.[31]

If joint involvement is suspected, diagnosis is confirmed through arthrocentesis, joint fluid analysis, and culture and sensitivity. Unfortunately, culture results are often negative because of the bacteriostatic properties of synovial fluid and previously administered antimicrobials.[4,36] Fluid analysis typically reveals more than 20,000 white blood cells and an elevation in total protein. Diagnosis may be more challenging without articular involvement; however, ultrasound evaluation coupled with aspiration and analysis of any fluid pockets may facilitate accurate diagnosis.[22] Radiographs are helpful in establishing a diagnosis, prognosis, and treatment plan. Initial radiographs are often normal but begin to show areas of lysis within a few days of onset. Aspiration of a lytic area may be performed with a needle; however, if this technique is not productive, a sample may be acquired via passage of a drill into the lesion and shavings from the drill may be cultured.[22,32] This portal may also be used for therapy by instillation of antimicrobial agents through a needle or through a cannulated bone screw placed in the hole.

Treatment of septic arthritis includes systemic broad-spectrum antimicrobials while waiting on culture results. Pain control, protection against gastric ulceration, and overall support for the foal regarding hydration and nutritional intake are important, especially in young foals. In cases with joint involvement, local treatment by joint lavage using sterile polyionic fluids is important; alternatively, constant rate infusion therapy with antimicrobials has recently proven beneficial.[1,28] Joint lavage is not necessary if the lesion is isolated to bone. Regional perfusion has proven beneficial, as has intraosseous antimicrobial administration.[22]

Anecdotally, the use of chondroprotective agents parenterally, orally, and intra-articularly is believed to be beneficial in restoring and maintaining joint health, especially in systemically ill foals. Hyperbaric therapy for treatment of osteomyelitis has likewise gained favor for the benefits of revascularization of ischemic tissue and increasing the effectiveness of antimicrobial therapy.[16] In debilitated or cachectic foals, the common clinical impression is that immediately following hyperbaric treatment, the animal is usually brighter and has an improved appetite.

With early diagnosis and appropriate treatment, the prognosis for the survival of foals with septic osteomyelitis is favorable, and a return to function in Thoroughbred racehorses reported between 37% and 48.3%[22,36,42,43];

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however, with a delay in therapy, the prognosis is guarded to grave. Younger foals (under 30 days) that display involvement of bone have a worsened prognosis because of a high incidence of multisystem disease.[18,36,42,43] Multiple sites of involvement have a significant detrimental impact on survival as well as future athletic potential.

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11

CHAPTER

Foot Care and Farriery

BASIC FOOT CARE

STEPHEN E. O'GRADY

INTRODUCTION

The equine foot is unique in that it is a biological entity (hoof structures) that follows the laws of physics (biomechanics). Biomechanics is the study of the mechanics of a biological structure, and it is essential that the veterinarian and the farrier have a working knowledge of the foot's biomechanics to implement appropriate farriery.[3,5,7] Proper farriery promotes a healthy, functional foot, provides biomechanical efficiency, and prevents lameness.[6]

The structures of the hoof complex comprise the hoof capsule, sole, frog, digital cushion, ungual cartilages, and deep digital flexor tendon (DDFT). The equine foot has numerous functions: supporting the weight of the horse, dissipating the energy of impact as the foot strikes the ground, protecting the structures contained within the hoof capsule, and traction.

The veterinary and farrier literature often describes the "normal" foot. However, the concept of normal may be misleading because it does not consider genetics, breed, foot conformation, environmental influences, and the athletic pursuits of the individual horse. Thus, the terms good, ideal, healthy, and functional foot may be more appropriate (Figure 11.1), because they describe a foot that comprises:

1. A thick hoof wall
2. Adequate sole depth (thickness)
3. Solid heel base
4. Growth rings below the coronet that are of equal size at the toe and heel
5. Acceptable foot conformation

Farriery (trimming and shoeing) affects not only the external hoof capsule but also the internal structures of the foot. Often trimming and shoeing methods are based on theoretical assumptions and aesthetic decisions derived from empiric experience rather than consistent guidelines that can be applied to each foot on an individual basis. Farriery assumes the dominant role in maintaining the health of the foot and preserving the integrity of its structures.[6]

The hoof care provided by the farrier depends on that individual's knowledge, training, experience, and skill level. Because the equine veterinarian is responsible for the overall soundness of the horse, a working knowledge of farriery is essential. A thorough knowledge of traditional horseshoeing enables the veterinarian to interact with the farrier at the farrier's level, which ultimately enhances the professional relationship and promotes quality hoof care.[2,4,6] This chapter focuses on basic, fundamental farriery: evaluation of the foot, foot trimming and preparation, shoeing principles, and farriery for common hoof disorders.

For clarity in this discussion, the hoof is defined as encompassing the distal end of the middle phalanx, the hoof capsule, and structures within the hoof capsule. The digit refers to the limb distal to the metacarpophalangeal/metatarsophalangeal (fetlock) joint.

EVALUATION OF THE FOOT

Before any type of foot care or farriery is initiated, the hoof and digit should be visually evaluated, and the observations recorded mentally or as a written record. Many of the smartphones or tablets are ideal for recording this evaluation and can then be stored as part of the horse's permanent record. This evaluation should begin with the horse in the static position on a firm, flat surface and then in motion in a straight line, again on a flat firm surface.

Static Observation

With the horse standing still and viewed from the lateral side, the hoof-pastern axis (HPA) should form a

Adams and Stashak's Lameness in Horses, Seventh Edition. Edited by Gary M. Baxter.
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straight line. The dorsal hoof wall should be parallel to the dorsal surface of the pastern. In the ideal foot, the angle of the hoof wall at the heel should approximate the angle at the toe of the dorsal hoof wall. However, this approximation of the toe and heel angle is generally not an accurate assessment because during movement, the heels of the hoof wall expand or move outward against the shoe—and to a lesser extent against the ground—when the animal is barefoot. This causes the hoof wall at the heels to wear relative to the hoof wall at the toe. Depending on the conformation of the foot and the amount of hoof mass present, this abrasive wear generally causes the hoof angle at the heel to be less than the angle at the toe. The angle of the foot appears to have taken on less importance recently since the hoof angles do not take into consideration the conformation of the horse's individual limbs.

It is generally thought that the foot is trimmed and/or shod appropriately and the hoof angle is correct when the dorsal hoof wall and the dorsal surface of the pastern are parallel.[1] The terms low hoof angle and high hoof angle describe a nonlinear relationship between the dorsal hoof wall and the dorsal pastern region (HPA). An imaginary line that bisects the third metacarpus (MC3) should intersect the ground at or near the most palmar aspect of the weight-bearing surface of the heels of the hoof capsule (Figure 11.2A). Furthermore, an imaginary vertical line extending midway between the dorsal and palmar borders of the middle phalanx (palpated just above the coronet) to the ground should bisect the hoof capsule at approximately the widest part of the foot. The coronet should have an even, gentle slope from the toe to the heel with no focal displacement.



Figure 11.1. A shod foot that demonstrates what is considered good or ideal foot conformation. *Source*: Courtesy of Jeff Ridley.

Viewed from the dorsal aspect, a line bisecting the third metacarpus should bisect the phalanges and hoof so that the hoof is approximately symmetrical, including the mass of the hoof on either side of the line and the heights and angles of the walls. The distal phalanx is often offset to the lateral side within the hoof capsule, which leads to disproportionate weight-bearing. A line drawn between two comparable points on the coronet should be parallel to the ground, and a vertical line bisecting the third metacarpal bone should be perpendicular to a line drawn across the coronet or the ground surface of the foot (Figure 2.37).

Viewing the hoof from the palmar aspect can help to note the height of the heels, whether there is a disparity between the heel height or heel conformation when compared to the contralateral limb, a condition termed mismatched foot; also note whether there is a disparity in the heel bulb height when measured from the ground surface of the hoof to the hairline at the heel bulb on an individual foot. The difference in heel bulb length appears to arise from disproportionate weight-bearing and should be considered when trimming and shoeing the foot.

Viewed from the ground surface of the foot, the width and length of the hoof capsule of the forefoot should approximate each other, although the hoof capsule may be slightly wider than it is long.[6] Stated another way, if a line is drawn across the widest part of the foot, a line from the widest part of the foot to the base of the frog at the heels and a line from the widest part of the foot to the toe should approximate each other (Figure 11.2B). The weight-bearing surface of the heels of the hoof capsule should extend to or coincide with the base of the frog.

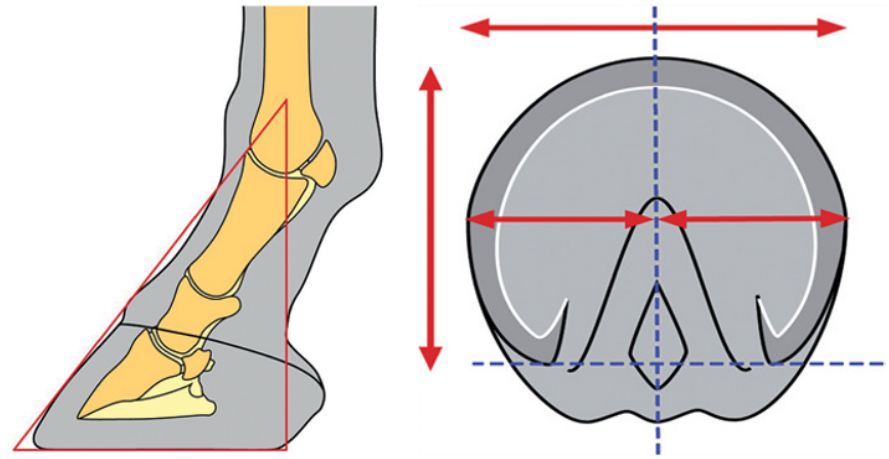


Figure 11.2. Schematic drawings showing the ideal hoof-pastern axis (A), extent of weight-bearing surface, and ideal proportions for the solar surface of the foot (B).

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A line drawn across the widest part of the foot can be used to evaluate both the shod and unshod foot and help determine whether previous farriery was appropriate.

Break-over is the phase of the stride between the heel lifting off the ground and the toe lifting off the ground. The point of break-over is best assessed from the ground surface and should be located in the center of the toe. However, the ideal point of break-over is widely disputed. In a traditionally trimmed and shod horse, break-over is positioned where the margin of the dorsal hoof wall intersects the ground. In a biomechanical sense, there are many situations in which it may be beneficial to position the break-over more palmarly.

The relationship between the longitudinal axis of the frog and the overlying distal phalanx is more constant than the longitudinal axis of the frog to the ground surface of the hoof. The relationship of the untrimmed frog to the sole indicates the position of the distal phalanx within the hoof capsule (i.e. the palmar angle). For example, if the apex of the frog is deeply recessed and the incline of the frog appears to be angling toward the coronary band at the toe, the distal phalanx is generally similarly positioned, creating a negative palmar angle. The central sulcus of the frog should be wide, solid, and shallow so that the index finger or ring finger fits easily into it. Contraction of the central sulcus is so common that it is widely ignored by clinicians. Another important reference point is the dimensions of the frog; the width of the frog should be 67% of the frog length.[8] Ideally, the medial and lateral aspects of the ground surface of the foot should be symmetrical on either side of the central axis of the frog.[6] The sole of the foot should be concave and possess sufficient depth such that it does not deform when pressure is applied.

Dynamic Observation

The key to observing the horse in motion is defining the placement of the foot as it strikes the ground. The strike pattern should be viewed from the side, and ideally the horse should either land slightly heel first or flat, with the toe and heel contacting the ground simultaneously. Marked heel-first landing generally denotes decreased ground surface in the palmar section of the foot or a shoe that is too small. Toe-first landing is considered abnormal, and it may be an attempt by the horse to avoid landing on its heels if they are painful or a club foot conformation where there is a shortening of the musculotendinous unit of the DDFT. Viewing the horse in motion from the front or from the rear allows the examiner to assess the mediolateral orientation of the heels as they strike the ground. Ideally, both heels should contact the ground simultaneously, but often horses with a narrow chest and a rotational limb deformity impact the ground on the lateral hoof wall and then load the medial side. This type of strike pattern results in the medial (or lateral) heel being displaced proximally; this type of hoof capsule distortion is termed a sheared heel.

Hoof Balance

Veterinarians and farriers often use the term hoof balance to describe the theoretically "ideal" shape or conformation of a foot, the position of the hoof relative to the limb above, and how the foot should be trimmed; however, hoof balance lacks an intrinsic definition.[3] The term has been further subdivided to describe various proposed methods for achieving balance, including geometric, dynamic, three-dimensional, and natural balance. In practice, no single method of balance will achieve optimum foot conformation for every horse. For example, a problem may arise with the exclusive use of either geometric or dynamic balance to address both dorsal palmar/plantar and mediolateral trimming. If the landing pattern or limb conformation deviates from normal, one method of achieving hoof balance may produce a foot with a different shape from another, or if the conformation of the limb causes the foot to assume a disproportionate load when landing, trimming the foot to land flat may be detrimental.[3,6]

"Hoof balance" is often used to communicate aspects of hoof care to the farrier. A more effective option is to use a set of biomechanical principles or landmarks that can be used as guidelines that have a universal meaning that can be applied to every horse, allowing the individual foot to be evaluated, trimmed, and/or shod in a consistent, reproducible manner (Table 11.1).

These guidelines standardize the basic approach to farriery by both veterinarians and farriers. Furthermore, they do not interfere with nor contradict the traditional concepts of static, geometric, or natural balance. The guidelines can also be used to modify existing hoof conformation and improve hoof capsule distortions and the landing patterns of the foot.

The guidelines or landmarks described in this chapter are applicable to both the forefeet and hindfeet, but for simplicity, only the forefeet are considered here. In the section on foot preparation (trimming), the connotation of balance is replaced with anatomical landmarks along with dorsopalmar/plantar and lateromedial orientation.

Removing the Shoe

Removing the shoe may appear elementary, but it is extremely important that veterinarians master the technique to avoid damaging the hoof capsule during removal. Shoes are removed during lameness examinations to examine the solar surface of the foot that is covered by the width of the shoe and during pre-purchase examinations to evaluate the entire perimeter of the sole–wall junction (white line) for abnormalities. Shoes should also be removed for radiographs; otherwise, they prevent some of the hoof structures from being imaged.

The first step in removing the shoe is to raise the clinch—the small square section of the nail that exits on the outer surface of the hoof wall and is bent over to secure the shoe—on the outer hoof wall. A clinch that is not straightened prior to pulling the shoe may damage the hoof wall as the shoe is removed. The clinch can be raised using a clinch cutter, a small chisel-type tool

Table 11.1. Hoof parameters to use as guidelines to evaluate and trim the foot.

1. Hoof-pastern axis
2. The center of rotation
3. Heels of the hoof capsule extending to the base of the frog

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Figure 11.3. Clinch cutter. *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.



Figure 11.4. Shoe pullers. Note the knobs on the ends of the handles, which distinguish this tool from hoof nippers (compare with nippers in Figure 12.15). *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.

(Figure 11.3). The clinch cutter is placed under the clinch, and a nylon hammer, which is quiet and prevents shock, is used to tap the clinch cutter upward and straighten the bent clinch. An alternative method is to file the clinches off with a hoof rasp.

Shoe pullers (Figure 11.4) are then placed between the shoe and the hoof, starting at the heel; the pullers are placed between the shoe and the hoof and levered forward, alternating from side to side toward the toe. The shoe is either removed in its entirety, or the shoe can be tapped back in place, exposing the head of the nail above the surface of the shoe just in front of where the



Figure 11.5. Creased nail pullers. Note the short handles, which can be readily used by veterinarians. *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.

shoe was levered. The nails are then removed individually and the levering process repeated behind each nail until all nails are removed. Another method is to use a creased nail puller (Figure 11.5) to grasp the nail head and remove each nail from within the crease or fuller of the horseshoe. The creased nail puller should always be used when removing therapeutic shoes to minimize trauma to an already compromised foot. If a horse loses a shoe or if the shoes must be removed and a farrier is not available, a layer of deformable impression material (Equilox Pink Equilox International, Inc., Pine Island, MN) can be placed on the solar surface of the foot and attached by wrapping the foot with a roll of 2- or 3-inch elastic tape. This provides comfort and prevents damage to the hoof wall until the farrier replaces the shoe.

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PRINCIPLES OF TRIMMING AND SHOEING

Stephen E. O'Grady

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GUIDELINES FOR TRIMMING

The Importance of Hoof Conformation

Foot conformation (shape) is defined by the structures and is important because of its relationship to the foot's biomechanical function. Any changes made to the bottom of the horse's foot through farriery affect the angulation of the hoof, the hoof-pastern axis (HPA), and the alignment of the hoof capsule under the center of rotation (COR).[6] Hoof conformation embraces the shape and function of the foot in relation to the ground, as well as the skeletal structures of the lower limb both at rest and in motion.[2,3,6] Each foot's conformation should be strong and protective and should maximize biomechanical efficiency. When this ideal conformation is theoretically achieved, it has been termed hoof balance. At present, there is no universal method to assess appropriate foot conformation or balance, nor is there a uniform method that implements guidelines when trimming the equine foot.

The Palmar Foot

The soft tissue structures that comprise and form the palmar/plantar section of the foot are often the limiting factor when trying to achieve and maintain good hoof conformation or shape.[11] The primary structures of the palmar foot include the frog, digital cushion, ungual cartilages, and deep digital flexor tendon (DDFT) (Figure 11.6).

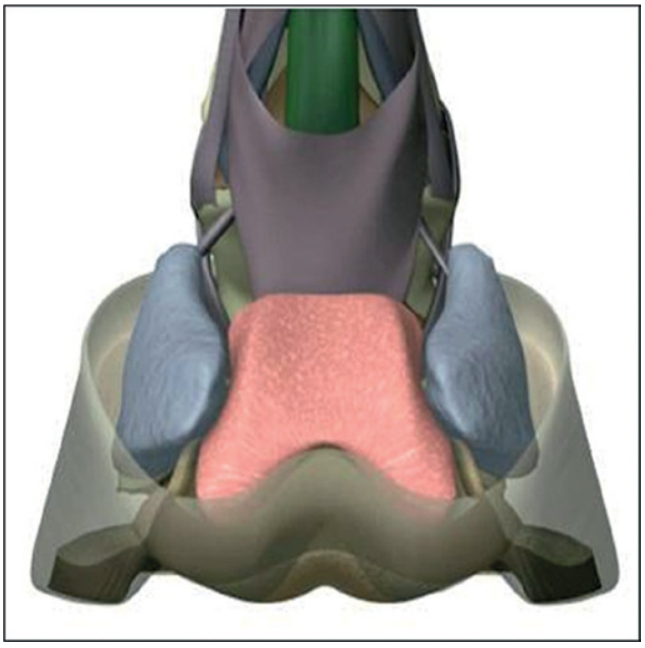


Figure 11.6. Foot model illustrating the structures in the palmar foot responsible for palmar foot conformation. Gray, ungual cartilages; pink, digital cushion; brown, frog. *Source*: Courtesy of Dr. Andrew Parks.

The conformation of the palmar/plantar section of the foot is contingent on the accumulated mass or density provided by these structures. Unfortunately, attaining sufficient mass for good conformation is not always possible because the structures are influenced by genetics (certain breeds such as Thoroughbreds often have decreased structural mass), improper development or maturity, continuous repetitive overload when the structures are immature (especially common in young horses in training), and inappropriate farrier practices. Farriers generally need to address the heel area (trimming, manipulating, or supporting) to achieve the desired HPA, position of the COR, and hoof capsule extending to the base of the frog.

The Hoof-Pastern Axis

The HPA should be the first guideline considered when trimming the foot. With the horse standing on level ground, the metacarpus/metatarsus should be perpendicular to the ground, and when viewed from the lateral side, the HPA should form a straight line along the dorsal surface of the pastern and the dorsal hoof wall from just below the fetlock to the ground. When the HPA is parallel, a linear line passes through the middle of the phalanges (proximal, middle, and distal) from the metacarpophalangeal joint to the ground. The straight alignment of the phalanges places the dorsopalmar orientation of the distal phalanx within the hoof capsule such that the solar surface of the distal phalanx assumes a similar alignment relative to the ground surface of the hoof capsule/ground. Ideally, the palmar angle of the distal phalanx should be 3°–5° greater than the dorsal angle of the distal phalanx; this allows the distal phalanx to descend in a distopalmar direction during weight-bearing and utilize the physiology in the palmar/plantar section of the foot (Figure 11.7).[9]

The angle of the solar border of the distal phalanx generally depends on the conformation of the palmar/plantar section of the foot. The dorsopalmar/plantar orientation of the distal phalanx within the hoof capsule helps prevent disproportionate load concentration on the solar surface of the hoof capsule and changes in the position of the ground reaction force (GRF).[14,16,17] Changes in the GRF in a dorsal direction, such as the broken-forward HPA, or in a palmar direction, such as in the broken-back HPA, suggest disproportionate weight-bearing on the solar surface of the hoof capsule when the foot is loaded. Additional detrimental effects of either a broken-back or broken-forward HPA have been well documented.[1,7,17]

Changes in HPA are generally associated with two common hoof capsule distortions: low or underrun heels or upright or clubfeet. A broken-back HPA is often caused by a low heel or excessive toe length combined with a low heel or underrun heel. The low heel is generally correlated with a lack of soft tissue mass in the

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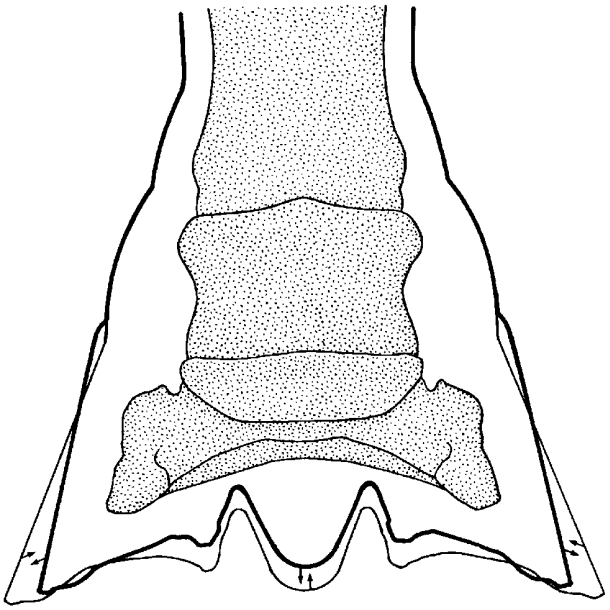


Figure 11.7. During weight-bearing, the frog and sole are forced downward, causing expansion of the hoof wall. The reverse occurs when the foot is unweighted.

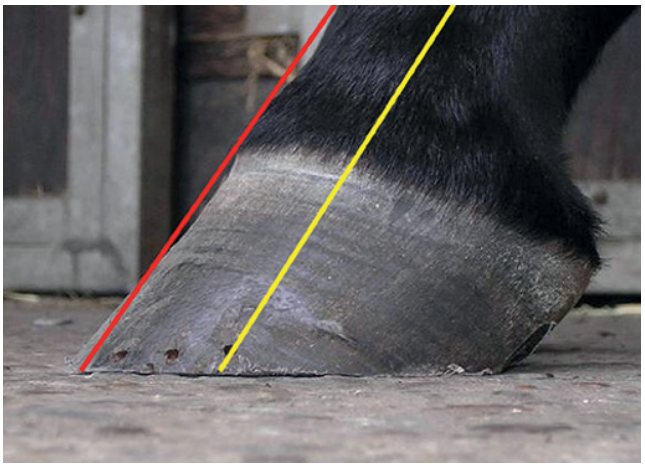


Figure 11.8. Hoof-pastern axis. Note that the dorsal surface of the pastern and the dorsal surface of the hoof wall form a straight line (red line). The yellow line depicts the corresponding digital alignment.

palmar/plantar section of the foot. This lack of soft tissue mass decreases the ability of the foot to dissipate the energy of impact during landing and the forces acquired during the stance phase of the stride, thus shifting some of these forces to the laminar interface and the bone. The broken-back HPA also places the distal interphalangeal (DIP) and to a lesser degree the proximal interphalangeal (PIP) joints in dorsiflexion, promotes load bearing in the heel area of the foot, and increases the stresses in the DDFT.

In a broken-forward HPA, the heels grow long while the frog generally recedes below the hoof wall, causing the energy of impact generated during weight-bearing to be transferred directly through the laminar interface to the bone, bypassing the soft tissue structures in the palmar/plantar section of the foot. The DIP and PIP joints are placed in flexion, which promotes load bearing on the dorsal margin of the DIP joint. The flexed DIP joint results in excessive weight-bearing to be placed on the dorsal section of the foot.

Until recently, the veterinary and farrier literature recommended that the normal hoof angle be 48°–55° for the forefeet and 52°–60° for the hindfeet. These recommendations have been shown to be erroneous, because they do not take into consideration the conformation of the horse's individual limb. Therefore, the foot is trimmed appropriately, and the hoof angle is correct for the individual horse when the dorsal hoof wall and the dorsal surface of the pastern region are aligned in parallel planes (Figure 11.8).[2,3,14] A parallel HPA is easy to access visually and can be confirmed radiographically when necessary.

The Center of Rotation

The second guideline or landmark used for trimming the foot is the COR. A vertical line drawn on a lateral radiograph from the center of the lateral condyle of the distal middle phalanx to the ground should bisect the bearing surface just palmar/plantar to the middle of the foot (Figure 11.9A).[4,6,7,10,11,14] This line demarcates the biomechanical COR of the DIP joint and should be in close proximity with a line drawn across the solar surface of the foot through the middle one-third of the frog or the widest part of the foot. The widest part of the foot is located 5–10 mm dorsal to the COR and forms a reliable landmark on the solar surface of the foot that is repeatable, can be used as a reference point when trimming, and can also be used in the evaluation of foot conformation and the current farriery that has been performed on the horse (Figure 11.9B). The widest part of the foot is also what farriers refer to as "Ducketts" bridge (Duckett D, Ambler PA. Personal Communication, 2008).

The widest part of the foot is the one point on the solar surface of the foot that remains relatively constant regardless of the shape or length of the ground surface that is dorsal or palmar to this point. In a biomechanical sense, because the widest part of the foot is just dorsal to the COR or the biomechanical pivot point, there are moments created on either side of the COR (Figure 11.10). Therefore, when considering biomechanical efficiency, the distance and force (moment) on either side of the line drawn through the widest part of the foot should approximate each other (equilibrium) when the horse is standing at rest. Following the trim, the ground surface of the ideal foot should be as wide as it is long, and the ground surface of the hoof capsule at the heels should not project dorsal to the base of the frog.[8,14,16]

Heels of the Hoof Capsule Extending to the Base of the Frog

The third guideline is the heels of the hoof capsule extending to the base of the frog. Located within the hoof capsule dorsally are osseous structures that accept load through the lamellae interface and soft tissue structures palmarly/plantarly that are thought to absorb concussion during load bearing and dissipate the energy of impact.

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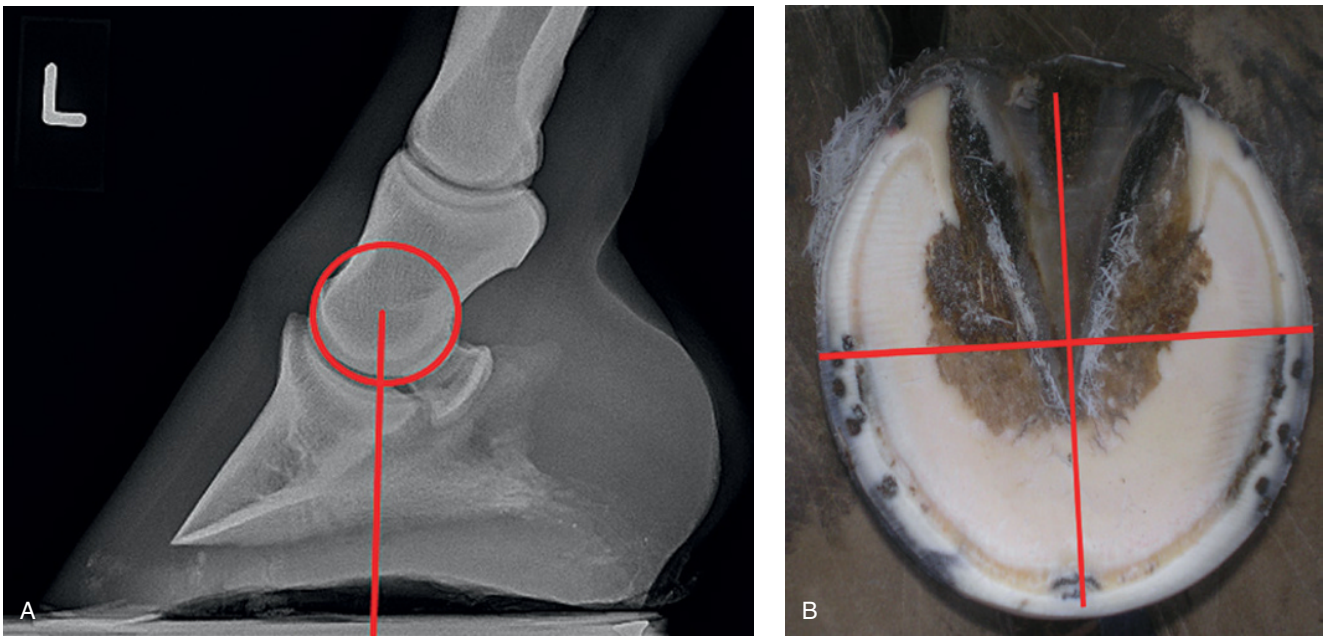


Figure 11.9. (A) Lateral radiograph demonstrating the center of rotation that should approximately bisect the weight-bearing surface of the foot (red line). (B) Solar surface of the foot showing the middle or widest part of the foot, which should be just dorsal to the center of rotation. Note that the foot is basically as wide as it is long.

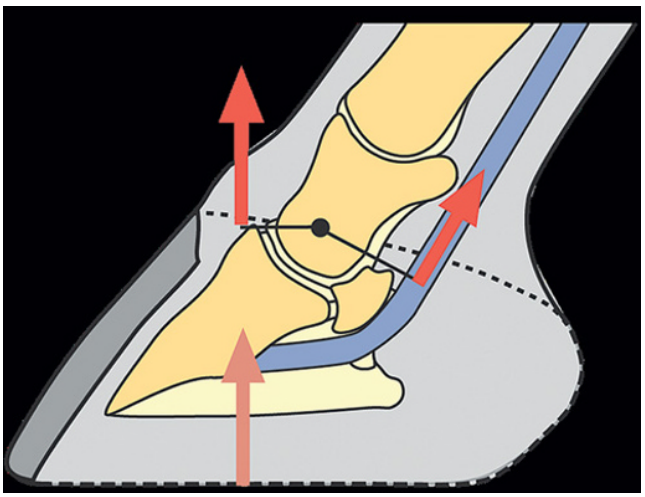


Figure 11.10. Schematic drawing illustrating the moments about the DIPJ. Note the extending force (dorsal red arrows) and a flexing force (palmar red arrow) on either side of the center of rotation.

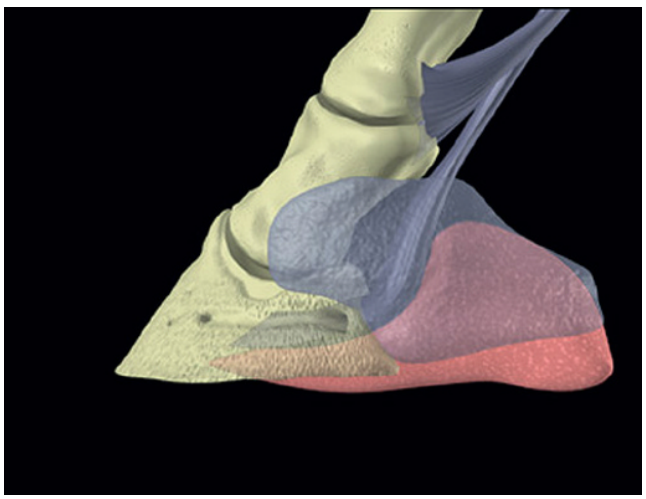


Figure 11.11. Model illustrating the necessity for the hoof capsule to approximate the frog to enclose the structures in the palmar foot. *Source*: Courtesy of Dr. Andrew Parks.

Within the hoof capsule, the distal phalanx occupies approximately two-thirds of the space, and the soft tissue structures occupy one-third. For effective physiologic functioning of the foot, the osseous and soft tissue structures must be located on the same plane within the hoof capsule and complement each other (Figure 11.11). The hoof wall at the heels of the hoof capsule should extend to or approximate the base of the frog.

Appropriate or consistent trimming of the palmar/plantar section of the foot has always presented problems for the clinician, for a variety of reasons. Variations in hoof conformation, farrier training, empiric considerations, and owner pressure not to trim the heels often dictate how the heels are trimmed. Due to the normal angulation of the horn tubules, the heels of the hoof capsule grow forward rather than tall. If the heels are not trimmed appropriately and are allowed to migrate dorsally toward the center of the foot, the laminar interface and the bones of the digit assume the function of the soft tissue structures. Often, the hoof conformation does not allow the heels of the hoof capsule to be trimmed to the base of the frog for anatomical reasons but an attempt should always be made to trim the heels of the hoof capsule to the same horizontal plane as the frog. If the hoof wall at the heels cannot be trimmed to the base of the frog, the branch of the shoe or some other form of farriery can be used to extend the ground surface of the hoof capsule to the base of the frog. Farrier methods for the palmar section of the foot are discussed later in the chapter.

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TRIMMING THE FOOT

The hoof care performed by the farrier is perhaps the most important routine procedure for maintaining and promoting the health of the equine foot. Farriery is a difficult occupation, and in order to provide the best service, the farrier should be provided with the best possible working conditions. The owner, client, or trainer should respect the farrier by providing a clean environment in which to shoe the horse. If an acceptable place is not provided, then the farrier should request one. The farrier should carry a good-quality fly spray and spray the horse as needed to control flies with the owner's permission. It is important to keep the area clean while working. Sweeping the floor after each foot is trimmed can be effective. The foot trimmings and manure should always be swept at least 6 feet behind the horse. Sweeping to the front or the side of the horse should be avoided as the debris will still attract flies to the horse's face or body. The broom is an important tool that every farrier should carry in his/her vehicle. Another helpful tip is to place an ordinary wood clamp in the tail hairs at the base of the tail to decrease tail swishing as the tip of the tail may injure your eyes when an odd fly lands on the horse. The farrier should have shade in warm weather and protection from cold weather in the winter. The area must be well lit and have an adequately sized space with a firm flat surface. Often the horse's owner or trainer must hold the horse to keep it quiet while being shod. If physical restraint is necessary, the client rather than the farrier should always perform it. Sedation or tranquilization should be discouraged when shoeing the horse, but if required, it is always best to have a veterinarian administer the medication.

The basic trim is the most significant aspect of good basic farriery. The farrier can use landmarks—the HPA, COR, and heels of the hoof capsule extending to the base of the frog—to carefully evaluate each foot and apply the appropriate trim. Using these three guidelines allows the farrier to be consistent with each foot trimmed. Sometimes good-quality radiographs are useful to locate or confirm the guidelines and provide additional parameters for applying the appropriate farriery.

The first observation should be the HPA in the standing horse, as described previously. To confirm that the HPA is straight or parallel, the horse should stand squarely on all 4 feet with the third metacarpal bone positioned vertically to the ground, on a hard, level surface, and be viewed from the side. If the HPA is either broken forward or backward, the initial trimming can be used to address this condition. The COR also can be located and marked on the outer surface of the hoof capsule if desired. A very practical technique is to palpate the dorsal and palmar border of the middle phalanx just above the coronet. Half the distance between the borders of the middle phalanx is determined, and from this point, a vertical line is dropped from the coronet to the ground (Figure 11.12). This line appears to have a good correlation with the widest part of the foot on the solar surface.

A leather apron should always be worn when trimming or shoeing the horse to protect the operator's legs. Trimming begins with picking up the foot and cleaning the solar surface with a hoof pick followed by using a wire brush, which allows the farrier to evaluate the structures on the solar surface. Before placing the foot between the farrier's legs, the ground surface of the foot is sighted by placing a hand on the medial side of the dorsal distal surface of MC3 just above the fetlock and allowing the distal limb (digit) to hang freely. The farrier's line of vision should be over the hoof to assess the lateral to medial orientation of the solar surface relative to the ground (Figure 11.13). If either the lateral or medial side of the hoof is excessively high or low relative to the opposing side, this can be addressed during the



Figure 11.12. The vertical line, as shown, usually approximates the center of rotation and the middle of the foot.



Figure 11.13. The examiner's line of vision should be placed over the hoof, which is permitted to hang in a relaxed natural position rather than being pulled outward. This permits accurate assessment of mediolateral orientation and inspection of the bottom of the foot.

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trim. It is equally important to evaluate the height of the heel bulbs (measured from the hoof wall at the heel to the hairline) and note any marked disparity between them. A notable difference in height (greater than 5 mm) denotes a sheared heel hoof conformation and should be addressed during the trim.[18]

The size and position of the frog should be noted on the ground surface of the foot. The width of the frog should be 67% of its length, and the surface of the frog should be on the same plane as the lateral and medial hoof wall at the heels of the hoof capsule.[14] The author believes that if the surface of the frog protrudes below the level of the hoof capsule at the heels or is recessed below the heels of the hoof capsule, this should be noted, addressed, and corrected, if possible, during the trim. If the frog protrudes below the hoof capsule, as is often seen in horses with low or underrun heels, the frog can be trimmed lightly to remove any excess horn and then the horse can be placed on a solid surface for 12–24 hours without shoes. This allows some compression of the frog and allows the heels of the hoof capsule to migrate distally, placing all of the structures on the same horizontal plane. If the frog is recessed below the level of the heels, an attempt should be made to trim the heels of the hoof capsule to the point where all structures located on the ground surface of the palmar foot are on the same horizontal plane. The length of the frog far exceeding the width is a clear indication of a chronic foot problem in which the proper physiology in the palmar foot has not been utilized. In this case the frog is usually accompanied by narrow heels and bars that show curvature instead of being straight; this condition is referred to as contracted heels.

Next, a line is imagined or drawn (with a magic marker or felt tipped pen) across what is judged or measured to be the widest part of the foot. This point can be measured with a ruler if desired. As described above, this line approximates the COR (Figure 11.9) and can be used to determine the dorsal and palmar proportions of the foot.

Before beginning the trim, the ground surface of the foot is aggressively cleaned again with a wire brush. The author removes no horny material from bulk of the solar surface of the foot other than any loose or exfoliating horn that is present over the frog or sole. In most cases, all horn material that remains after the foot is cleaned with a wire brush is left intact.

Excess length of the hoof wall at the toe and quarters of the hoof is determined by paring the sole–wall junction (white line) to live solid sole with an appropriate hoof knife (Figure 11.14), and then the excess horn relative to the trimmed sole is removed using hoof nippers (Figure 11.15), being careful to leave the adjacent sole for protection. The author removes the excess hoof wall length beginning just dorsal to the heel quarters with the nippers placed in a tapered position to start the cut and then moving in sequential increments to the opposite heel quarter. Using a hoof rasp (Figure 11.16), the heels of the hoof capsule are trimmed to the same horizontal plane as the frog and then blended into the toe and quarters, creating a flat level surface.

The author prefers to use the rasp in a side-to-side motion rather than in a forward motion except at the toe. This allows better control and prevents uneven



Figure 11.14. Offset hoof knife (A) and a loop hoof knife (B). *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.



Figure 11.15. Hoof nippers. Note the flared ends to the handles that help distinguish these from shoe pullers (compare with Figure 11.4). *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.

areas of the foot from being created. Attention is paid to extending the heels of the hoof capsule to the base of the frog when possible, with the intention of creating a solid heel base. If there is insufficient hoof wall for the end of the heel to reach the base of the frog, or if the heels are damaged, this distance can be lengthened by extending the branches of the shoe or by some other farriery method. The key to trimming the heels is to complete the trim at the heels with the hoof wall and the frog on the same horizontal plane if possible (Figure 11.17). The medial or lateral hoof wall can be trimmed cautiously relative to the other when changes to the lateral medial

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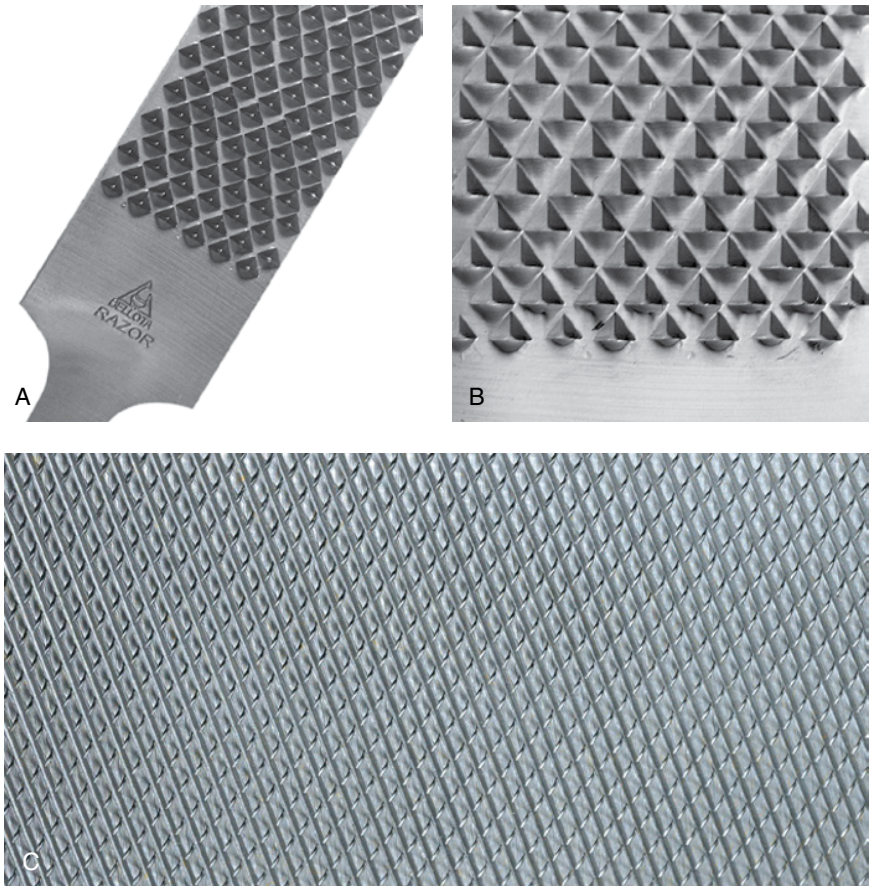


Figure 11.16. Hoof rasp (A). Note the coarse side of the rasp (B) that is used to remove hoof wall and the smooth side (C) that is used to level the foot. *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.

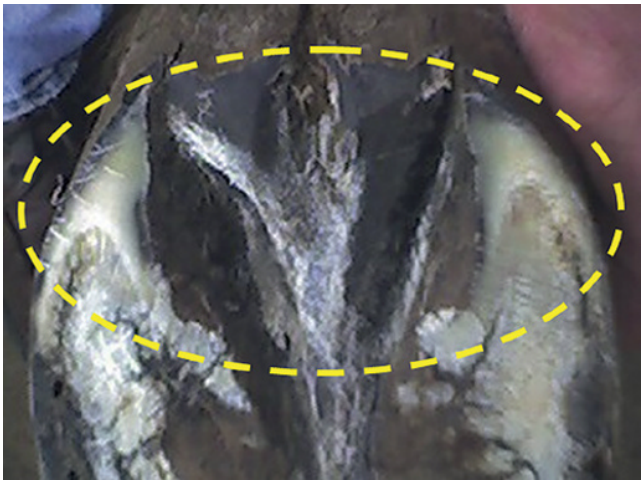


Figure 11.17. Heels of the hoof capsule trimmed to the base of the frog. Note that the hoof wall at the heels and the frog are on the same horizontal plane (yellow circle).

orientation of the foot are necessary, such as in the case of sheared heel conformation.

An imaginary line drawn from the initial line across the widest part of the foot to the base of the frog should approximate another line drawn from the widest part of the foot to perimeter of the hoof wall at the toe. When making this final assessment, the distance from the middle of the foot to the toe is generally greater than the distance to the base of the frog. When this occurs, the foot can be placed on a foot stand, and the outer perimeter of the distal dorsal hoof wall at the toe can be reduced with a rasp. This places the COR in the middle of the foot or in the middle of the shoe when shod. The foot is further shaped by removing flares from the outer surface of the hoof capsule to concentrate weight-bearing on the hoof wall under the limb. Following the trim, a shoe can be applied to complement the trim, protect the trim, and change the biomechanics of the foot when necessary.

TRIMMING THE BAREFOOT HORSE

Recently, it has become popular to maintain horses in the barefoot or natural state. The author favors horses being maintained without shoes when possible. On the other hand, the author believes that horses can be shod in a physiologic manner such that minimal damage to the hoof capsule will occur. Factors to consider when making this decision to leave the horse barefoot include:

- Protection for the before ground surface of hoof capsule when hoof wear exceeds growth
- Traction that is necessary for athletic activities in performance horses and safety for horse and rider
- Therapeutic shoes or devices to treat lameness or diseases of the hoof or to address limb conformation

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Any one or a combination of the above factors may dictate the necessity for shoes. The decision also depends on the intended use of the horse, the owners' expectations, and the type of hoof care the horse receives, especially during the transition period from shod to barefoot. Much of the horse industry is involved in competitive athletic disciplines, and the question arises, "Can this horse compete and perform at a given standard without shoes?"

Wear vs. hoof wall growth takes into consideration the genetics and breed of the horse, structure and conformation of the hoof, working surface, farriery practices, and, most importantly, the horse's intended use, all of which influence wear of the feet. The quality and integrity of the structures of the foot is usually the determining factor.

Maintaining the horse barefoot is best accomplished when—or potentially when—the hoof wall is thick and solid, there is good sole depth, and there are soft tissue structures in the palmar/plantar section of the foot that are of sufficient mass. Breeding practices have influenced the structure of the feet, and unfortunately, this has not always been for the better. Quarter horses have been bred for fashion, whereas Thoroughbreds have been bred for speed, and the result is often poor-quality feet. Usually, and especially in Thoroughbreds, the foot is often prevented from growing and maturing into a so-called good or functional foot due to husbandry or training practices.[14]

Hoof development, particularly during the first 3 years, depends on stimulation from regular exercise and turnout. Yearlings are often shod for sales for purely cosmetic reasons or in an attempt to alter faulty foot conformation. The majority of racehorse's feet remain healthy until the time they are broken and enter training, usually as 2-year-olds. As training begins, the hoof capsule and its related structures are still immature, the animal is confined to a stall or small paddock with exercise severely limited, a rider is placed on its back that leads to additional weight-bearing on the feet, and the horse begins to work. Training leads to abnormal stresses being placed on an underdeveloped foot along with excessive wear. Next, the horse shows discomfort, and shoes are then placed on the feet for protection. The first thing that happens when shoes are applied is that the solar surface of the foot loses contact with the ground and its ability to adapt. Furthermore, the method of foot trimming and the size and placement of shoes by the farrier must be considered.

The combination of the above factors can and often does change the structures of the foot forever, or the factors can lead to a weak foot that is hard to maintain without shoes. Taking a digital photo of a horse's foot at the start of training and then taking another photo 6 months to a year later and comparing the difference can confirm this observation. The feet are often overtrimmed and shoes are placed on young horses too early and often unnecessarily if just a few modifications were made to the farriery and the training program to allow the feet to continue to develop.

The anticipated exercise program is also a key factor. Many horses fare well without shoes, if they are not asked to perform. Whereas light riding of barefoot horses may be feasible, competition may not be possible. Finally, the surface upon which the horse is kept and exercised influences the wear on the feet. A hard or abrasive surface, such as sand, is not as forgiving as a soft, deformable footing.

The need for traction on certain ground conditions such as wet grass can also dictate the choice of barefoot vs. shod. Shoes themselves act as a traction device by providing additional cup to the horse's foot. Shoes, and various traction devices that are added to shoes, allow horses to hold their footing, prevent slippage, enhance propulsion with the hindfeet, and improve overall performance in competitions such as eventing, jumping, steeplechase racing, and polo. Winter equestrian sports such as fox hunting are aided by traction devices because of the diverse weather and footing conditions. Traction devices protect the horse and give the horse and rider confidence while performing. Often overlooked is the fact that they also protect the rider, whether trail riding or competing. Borium or studs provide additional safety from slippage and allow a horse to be ridden, to pull a carriage up and down hills, or to pull a sleigh on snow and ice.

Sliding plates used in reining horses may be considered anti-traction devices because they decrease the friction between the ground and the hoof.

Finally, therapeutic shoeing generally is a component of or sometimes even the entire treatment for lameness localized to the foot. Lameness results from repetitive stresses or overload placed on a given structure(s) of the hoof capsule or within the hoof, leading to damage. Shoes can be used to change the forces/stresses on a given structure and unload damaged areas of the foot. They are used for realignment of the distal phalanx in laminitis, provide continuity of the hoof capsule after resection in white line disease (WLD), stabilize hoof cracks and distal phalanx fractures, and provide protection following a puncture wound or foot surgery. Angular or flexural deformities in young horses may be treated or aided by several types of shoes.

A transition period to allow the foot to adapt is generally needed when changing from shod to barefoot.[12] The hoof wall must toughen and the sole must become thicker and stronger to compensate for not wearing shoes. Horses are much easier to maintain in a barefoot manner if they have never had shoes. Another factor in the transition period is the length of time that the horse has worn shoes; this affects the length of time needed for the horse to develop the necessary sole protection once the shoes are removed. Often, the foot structures have inadequate mass or are irreversibly damaged and thus incapable of adaptation.

The horse should be taken out of work if a decision is made to remove the shoes. The author recommends a 30- to 90-day transition period during which time the structures of the horse's feet can toughen and adapt to going without shoes. At this point hoof care changes from trimming the foot to shaping the foot.[13] The only tools necessary are a wire brush and a rasp. Nothing is removed from the bottom of the foot. Using a rasp, the heels are moved back to the base of the frog (when possible) such that the heels of the hoof wall and the frog are on the same plane. Unlike when applying shoes, the hoof wall is rasped on an angle (instead of a flat plane) to create a rounded edge. The angle of the hoof wall is

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created with the rasp placed at a 90° angle, starting on the outer side of the sole–wall junction (white line). This manner of rasping reduces the hoof wall length and creates a sharp edge that is then smoothed with the rasp used in a horizontal direction, resulting in the rounded perimeter. Flares, or excess toe, are removed from the outer hoof wall (shaping). The shaping is finished by slightly beveling the toe on the ground surface of the foot from the toe quarters forward to decrease leverage, which promotes sole growth and to toughen the sole–wall junction. This bevel should not be created if firm pressure (using thumb pressure or hoof testers) on the sole causes the sole to deform.[13]

The length of the adaptation phase often can be predicted according to the initial structure of the horse's foot and should be modified as necessary. When there is minimal sole depth (as evidenced by hoof testers applied to the sole), the horse should be confined or placed in a small area of soft footing and walked daily on a firm surface until the structures of the foot begin to change and adapt. Placing the horse in a protective boot will protect the foot but may not provide the necessary stimulation for adaptation. At no time should the horse show marked discomfort, because this defeats the purpose. If the horse's sole has not become firmer and noticeable growth of sole does not appear on the inner border of the sole–wall junction after 30 days, then it may be worthwhile to take lateral radiographs to assess sole growth or reconsider whether this method of hoof care is in the best interest of the horse.

THE HORSESHOE

The basic configuration of a horseshoe is a curved bar made from a variety of materials that is rectangular in cross section and shaped to conform to the contour of the ground surface of the hoof wall. The shoe should be wide enough to cover the ground surface of the hoof wall, the sole–wall junction (white line), and the sole adjacent to the sole–wall junction. The shoe has four surfaces: the foot and ground surfaces and the inner and outer perimeter or margins. The parts of the shoe are named after the corresponding section of the hoof: toe, quarter, and heel. Each shoe has two branches, medial and lateral, that extend from the center of the toe to the medial and lateral heels, respectively. The width of the shoe is often referred to as the web. The shoe is punched or machine stamped with nail holes, three or four in each branch, 2–3 of which are generally used in each branch.

Materials and Size

Horseshoes may be made from steel, aluminum, titanium, synthetic polymers, or various composites; the construction material dictates the shoes' weight and durability. Most horseshoes are made of steel due to their durability, cost, and workability. However, aluminum is used frequently because of its lighter weight and ease of shaping for many farriers. Recently, shoes made from various synthetic polymers and composites have been recommended for specialty uses, but they have not replaced the traditional materials.

Shoes may be hand forged from steel or aluminum bar stock or manufactured. Handmade shoes offer few advantages over the manufactured shoes that are available on the market today. It is more important that the farrier be skilled at forging, so he/she can modify manufactured shoes to fit the foot and hand forge a shoe from bar stock when necessary. Heating the shoe makes altering the shape much easier, and it has advantages when fitting the shoe, such as seating clips (when used) and detecting uneven sections on the ground surface of the foot. Excessive heat generated by repeatedly placing a red-hot shoe on the horse's foot should be avoided, as the heat appears to damage the inner structures of the sole–wall junction.

Bar stock is usually rectangular in cross section and available in sizes from 0.6 to 1.2 cm thick and 1.2–3.1 cm wide. The most common size is 0.8–1.9 cm. Concave bar stock has a crease stamped in the bar stock and is tapered from the crease toward the inner margin on the ground surface of the shoe. A shoe made from this type of bar stock is thought to increase traction on all terrains and possess a self-cleaning action. It is frequently used in Europe but has also become popular in the United States. Pre-made concave shoes can also be purchased.

Manufactured shoes, also referred to as keg shoes, are catalogued using numerical sizes. Although the sizes are usually consistent within and between a given manufacturer's product lines, there is no universal standard for sizing keg horseshoes. Manufactured shoes may be generically shaped to the general shape of a horse's foot or specifically designed for a forefoot or hindfoot. The dimensions of the stock of both manufactured and hand-forged shoes affect the weight and stiffness of the shoe, coverage of the ground surface of the foot, height to which the shoe elevates the foot off the ground, and rate at which the shoe wears.

Shoe weight influences the biomechanics of movement. The heavier the shoe, the more energy is expended accelerating and decelerating the limb at the beginning and end of each stride. Therefore, the lightest possible shoe should be used that is compatible with protecting the wall and adjacent sole and providing the stiffness and wear required. Shoes made from concave stock are lighter than those made from regular bar stock. The width and thickness of the shoe usually is uniform around the circumference of the shoe so that the biomechanical influences of shoe weight and the stresses imposed are generally balanced about the axis of the limb. Although the width or web of a shoe is related to the thickness of the hoof wall, and at least in part the size of the foot, it is common to increase the width of the web of the shoe to provide increased protection to the margins of the sole. The thickness of the shoe is related to the rate at which it is expected to wear and to a lesser extent the rigidity needed to prevent the shoe from losing shape. The height that the shoe raises the foot off the ground also is influenced by the thickness of the shoe, but this is usually a secondary consideration.

Several common modifications are made to the ground surface of the shoe by forging, and some modifications can be made with an electrical grinder or sander. Softening the 90° angles at the junction of the shoe's

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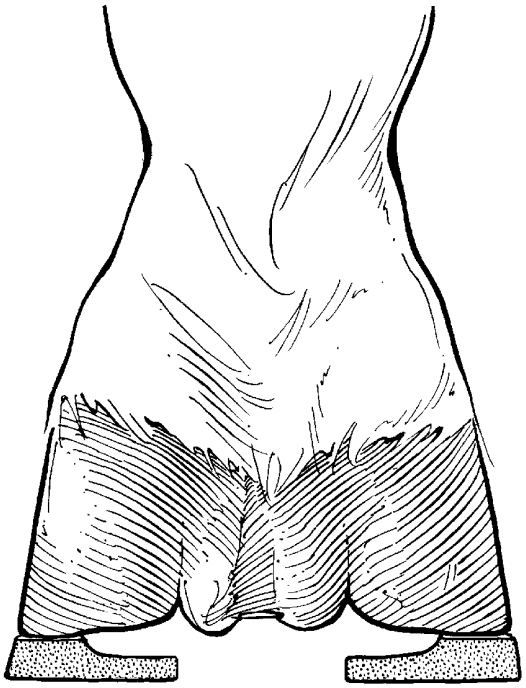


Figure 11.18. A wide-web shoe with a concave solar surface covers and protects the sole without applying pressure to it.



Figure 11.19. Half-round shoe.

ground surface and outer margin by beveling or rounding, called safing, increases the ease of break-over. A shoe with a rounded outside perimeter is thought to improve the ease of break-over in any direction. A similar modification used by the author is achieved by using a half-round shoe, which is made from half-round bar stock that resembles a semicircle in cross section (Figure 11.19). Half-round shoes can be purchased, but the semicircle does not have the same curvature as a shoe made from bar stock.

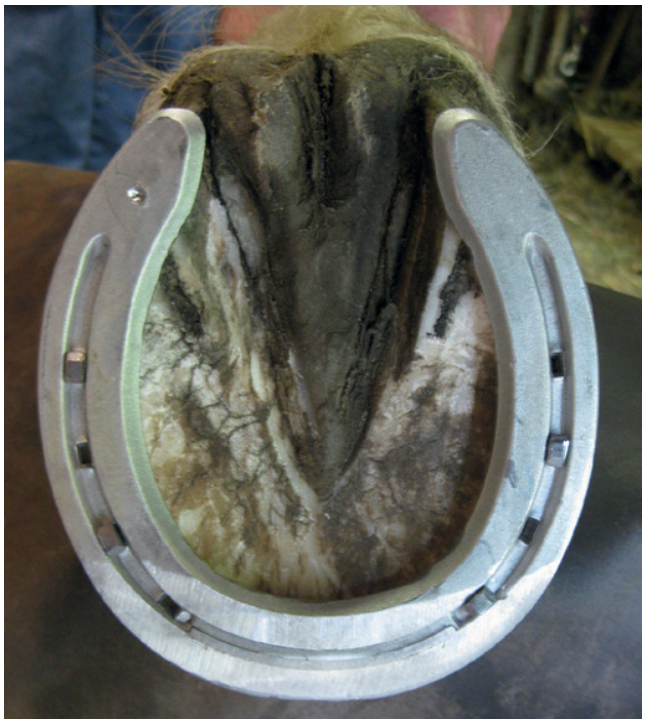


Figure 11.20. Using an electric grinder, break-over can be created in any shoe beginning at the inner margin of the shoe creating a tapered effect toward the outer margin.

The toe section of flat shoes frequently is rolled or beveled only at the toe to improve the ease of break-over. This modification can readily be accomplished by forging. For enhancement of break-over, the author uses a grinder and begins the roll at the inner margin of the ground surface of the horseshoe and creates a tapered roll from this point to the outer margin. This decreases the thickness of the outer margin of the shoe by half the thickness of the material (Figure 11.20).

A similar effect can be achieved by rockering the toe, which is accomplished by bending the full thickness of the toe area of the shoe proximally from toe quarter to toe quarter. When rockering the toe of a horseshoe, it is important to leave sufficient hoof wall mass at the toe to accommodate the rocker created in the horseshoe with the result being an intimate fit between the shoe and the toe area of the foot. This intimate relation is created by burning the rocker in to the toe or by using a hoof rasp to create the bevel in the toe of the hoof to accommodate the rocker in the shoe. Less commonly, a flat shoe may be asymmetrically beveled or rounded to improve the ease of break-over in a desired direction or toward a given side of the shoe to encourage a horse to break-over toward that side and direct or accommodate the flight of the foot. Rounding the outer margin of the medial branch of a shoe on the ground surface of the shoe with a hammer or grinder is called boxing. See Table 11.2 for a list of commonly used farrier terms.

There is little traction between the flat surfaces of steel or aluminum shoes and the ground. Grooves are created in the ground surface of the shoe, called fullering or creasing, to increase traction. A similar modification

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Table 11.2. Basic farriery terms.

Term	Definition/explanation
Bar shoe	To increase the ground surface area of the shod foot. It allows weight-bearing to be redistributed from one area of the foot to another
Patton bar shoe	A Patton bar shoe is designed to add significant length and varying degrees of elevation to the palmar/plantar section of a hoof capsule. The initial shoe may be designed as an egg-bar, a straight-bar, or an open-heel shoe
Square toe	A square toe is a shoe modification made to a hind shoe. The material in the toe of the shoe is forged to be square across the dorsal border allowing the shoe to be fit underneath the dorsal toe of the hoof capsule. The square toe shoe gives a foot direction as it breaks over, looking for the path of least resistance. By setting the shoe underneath the toe, the point of break-over is changed, and the flight of the foot is altered to a more upright animated stride
Rolled toe	A rolled toe is a shoe modification that may be applied to the ground surface of both front and hind shoes. It may be forged into a shoe or crated with a grinder from the dorsal border of the shoe back to the inside of the web of the shoe at the toe in a graduated or tapered manner. This modification allows one to change the point of break-over in a shoe
Rocker toe	A rocker toe is a shoe modification where the outer 2/3 dimension of the branch of the shoe at the toe is forged in a manner that is on a different plane than the rest of the shoe. The toe plane would be forged upward, emulating the base of a rocking chair. This modification is applied to both front and hind shoes and may be considered a more aggressive manner of changing the paint of break-over and the flight of the foot via break-over
Forging	Forging is a process where a piece of metal is heated in a forge to increase its malleability. The metal is then forged between an hammer and an anvil, manipulating the material in a manner that it may be drawn, stretched, or creased and nails hole punched into it, as it is shaped into a specific horseshoe. Forging would also encompass the development of numerous shoe modifications such as bar shoes, trailers, clips, etc.
Fullering/ creasing	The shoe modification is used regularly to create a groove in the approximate middle of the branch on each side of a horseshoe where nail holes will be placed in the shoe. The crease is forged into a shoe with a hammer and creaser that is driven into heated steel displacing the steel, crating the groove. This crease creates an opportunity to remove a shoe utilizing a tool called a creased nail puller. Removing one nail at a time eliminates pressure applied to the sole during the removal process. Additional creases may be applied to a shoe to increase traction. In addition, as the crease fills with dirt, it will increase traction and widen an area of the shoe, offering increased ground surface
Swedging	Swedging is a forging exercise where you are changing the shape and dimensions of a piece of bar stock by placing heated steel into a swedge block, striking it with a sledge/power hammer to change the shape and dimensions of the original metal used. A swedge block is generally designed with a 3″ × 3″ × 3″ block of mild steel with a post welded in place to be fit into an anvil's hardy hole. The block may be designed into numerous variations or configurations depending upon the section of the steel desired
Boxing	Boxing is a modification where material is removed from the outer perimeter of the hoof surface of a horseshoe, creating a beveled or slanted edge rather than a sharp flat edge. This process allows the shoe to be fit wider than the margins of the hoof capsule itself. Boxing reduces the likelihood that the horse will step on its own shoe and pull it off
Safing	Safing is a shoe modification where material is removed from the ground surface of the outer perimeter of the medial branch of a shoe from the widest part of the foot back to the heel. This modification creates a beveled or slanted edge on the ground surface of the horse shoe on the inside branch where a horse is likely to step on himself. The beveled edge makes the shoe less likely to injure the horse, if the horse steps on the opposite foot or interferes during limb flight
Clip	A clip is a thin portion of material of a horseshoe that is forged toward the hoof surface of a shoe in a vertical manner from the outer edge of the shoe. The clip should be approximately the same height and width as the dimension of the branch of the horseshoe. Clips are descriptive in nature such as toe clips (would be drawn in the toe of the shoe), quarter clips, and side clips drawn relative to where they are located on the side of the shoe. A clip's sole purpose is to be fit against the hoof wall and minimize the shearing action encountered on horseshoe nails as the horse applies torque when turning or stopping in an abrupt manner
Trailer	A trailer is a shoe modification that is applied to the lateral branch of a hind shoe. The length of a trailer is approximately the same length and width as the dimension of the branch of the shoe that it is being applied to, i.e. a 3/4-inch trailer is appropriate on a shoe that is made from 3/4-inch bar stock. The trailer is forged so it deviates laterally at the heel of the lateral branch of the shoe, thus increasing lateral support to the foot during the weight-bearing phase of the stride. The added length is designed to give the foot direction as it lands

in racing or training plates is termed swedging. The fullering in the horseshoe becomes compacted with dirt; the friction between the dirt in the fullering and the dirt on the ground is greater than steel on dirt, thus aiding in traction. In addition, the nail heads are embedded within the crease with 1–2 mm of the nail head protruding above the crease, which also aids traction and helps to keep the nails tight.

The full circumference of the shoe may be fullered as in the concave shoe, or it may be limited to the branches of the shoe and less frequently just the toe. Fullering a single branch of the shoe enhances traction on one side

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and delays break-over on that side of the foot. When the branches of the shoe are fullered, the nail holes are centered in the groove, which conforms with the inside and outside of the nail heads. A classic example of a shoe that uses two of these modifications is a half-round, half-swedge shoe worn on the hindfeet by harness horses. The inner branch is half round in cross-sectional profile and the lateral branch of the shoe is swedged. Thus, the medial branch, enhanced by the half round, breaks over rapidly, whereas the break-over of the lateral branch is delayed by the swedge.

The only common modification to the solar surface of the shoe is beveling of the inner half of the web toward the inside with a hammer or a grinder, called seating out or concaving. Horses with flat, thin soles benefit from a seated-out or concave shoe to decrease pressure on the sole adjacent to the wall (Figure 11.18). Safing decreases the likelihood of the shoe being stepped on by another foot and pulled off. Boxing the heels allows the farrier to fit the shoes with more expansion (width) at the heels without a ledge being present, which can easily be stepped on by another foot.

Clips are triangular-shaped projections that extend proximally from the periphery of the shoe (Figure 11.21). Clips are preformed on manufactured shoes or they may be forged in the shoe at the time of fitting. When a shoe is fitted, the outer surface of the clip is congruent with the surface of the hoof wall. Movement between the shoe and the hoof capsule is reduced, which decreases the shear stress on the hoof nails. A single clip placed in the center of the toe or side clips placed at the toe quarters can be used on front shoes, while side clips placed at the toe quarters or quarters are generally used on the hind shoes. Clips may be positioned elsewhere around the periphery of the shoe when additional stability of the hoof capsule is necessary.

Extensions

An extension on a horseshoe is any projection of the shoe that extends outward beyond the perimeter of the foot in the horizontal plane. Extensions may be placed anywhere around the circumference of the foot. They may be forged into a shoe or welded onto the outside margin. An extension such as a wide lateral heel branch in a hind shoe is available as a manufactured shoe. A similar effect is obtained by using an oversized shoe fitted either forward or backward or by setting one branch of the shoe wide.

Every extension has the potential to cause the shoe to act as a lever. This lever action may be static, when the animal is not moving and the foot is bearing weight, or dynamic, particularly during the landing and break-over phases of the stride. When force is exerted on the extension by the ground, the forces are increased in the adjacent wall and decreased in the opposite wall as the center of pressure of the GRF shifts. In addition to acting as levers, extensions increase the surface area available for ground contact, which decreases the amount of shoe that descends into soft footing at that point in the circumference of the foot. The term floating or flotation effect refers to the web width of a section of the shoe that limits the amount of this part of the shoe that descends into soft footing (Figure 11.22).

Lateral or medial extensions may be used to force the opposite wall of the hoof capsule to the ground or support the wall adjacent to the extension. In doing so, the lever decreases the compression in the opposite wall and increases the compression in the side of the hoof on which the extension is being used. Heel extensions frequently are used on one side of the foot or biaxially (Figure 11.23). When used in performance horses, extensions are continuations of the heel of the shoe and are called trailers.

Trailers are used almost exclusively on the lateral branch of hind shoes. Egg-bar shoes that extend palmar/plantar to the heels of the hoof capsule act as heel extensions. They are most commonly used on the front feet but can also be used on the hindfeet when necessary. Force applied on the butt of an egg-bar shoe decreases the moment about the DIP joint and the tension in the DDFT. Therefore, horses convalescing from a DDFT injury may benefit from heel extensions used in conjunction with some type of heel elevation. The egg-bar shoe acts as a palmar extension, and when the horse is on a soft surface, it reduces the sinking of the heels into the surface at the beginning of the stride and acts as a heel wedge during the support phase of the stride.[5] On the



Figure 11.21. A steel hind shoe with quarter clips (A) and a clipped steel hind shoe fitted to the foot (B).

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other hand, egg-bar shoes can be fitted too long, and the extension beyond the heels acts as a lever, causing excess pressure on the heels of the hoof capsule. Heel wedges are known to decrease the force on the navicular bone.[5] However, the consequence of this benefit is that it is often used on horses with damaged heels and the heels are then placed under greater compressive stress. Heel extensions alter the way the foot strikes the ground. If, as happens frequently, the heels are closer to the ground at impact, the extension or butt of the egg bar contacts the ground first. A lateral trailer, either in line with or diverging up to 45° from the midsagittal plane of the foot, forces the foot to pivot toward the side of the trailer as the foot lands. The toe of the foot is directed laterally after impact and break-over is redirected.



Figure 11.22. Shoe with one branch wider than the other, showing the flotation effect on one side of the foot.

Bar Shoes

A bar is the part of a shoe that extends from one branch of a shoe toward the other, forming a closed unit. In a complete bar shoe, one branch extends to the other. In a partial bar shoe, the bar extends part of the way across the shoe. Most bars extend from one heel to the other to form a closed shoe. A bar may extend from one quarter to the other, or even diagonally across the shoe. Several patterns of complete bar shoes are commonly used, including the straight-bar, egg-bar, heart-bar, heart-bar–egg-bar (or full-support shoe), and Z-bar shoe (Figure 11.24).

Indications for a bar shoe include increased stability to the hoof capsule, increased ground surface area, local protection, recruitment of additional weight-bearing area of the foot, and ability to unload a section of the foot. Some type of bar shoe is frequently used, often in conjunction with other shoeing methods, to increase stability within the hoof capsule or distal phalanx. Bars that extend palmar/plantar to the normal position of the heel of the shoe act as a palmar/plantar extension. Bars that are set under the ground surface of the foot can be adjusted to protect that part of the foot from ground contact, apply pressure to that part of the foot, unload that part of the foot, or recruit that part of the foot for weight-bearing. A straight-bar shoe may decrease pressure on the palmar/plantar third of the frog and protect the underlying navicular bone. The heart-bar shoe is used to recruit or increase the role of the frog in weight-bearing, thus using the frog to create a weight-sharing relationship with the hoof wall and



Figure 11.23. A shoe with a trailer (A) and its application on the left hind foot of a horse (B). The trailer should be as long as the width of the horse shoe. *Source*: Courtesy of Jeff Ridley.

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Figure 11.24. (A) Egg-bar shoe. (B) Straight-bar shoe. (C) Heart-bar shoe. (D) Z-bar shoe. *Source*: Courtesy of Jeff Ridley, CJF, TE.



Figure 11.25. A roadster shoe with a lateral heel calk and a medial wedge to improve traction. *Source*: Courtesy of Jeff Ridley, CJF, TE.

laminar interface. Heart-bar shoes also may be used to support the palmar/plantar aspect of the foot to reduce the stress in the adjacent wall and permit unloading of one or both heels but only if the integrity of the soft tissue structures of the frog and digital cushion have sufficient mass and are intact.

A partial bar shoe extends part of the way across the ground surface of the foot, most commonly from one heel onto the frog, which increases or reduces weight-bearing on a single heel. Farriers refer to this type of shoe as a half-bar or G-bar shoe. The bar of a Z-bar shoe is shaped with two 90° bends that are incorporated into a three-quarter shoe so that one leg of the Z is attached to the heel of one branch and the other is attached to the quarter of the opposite branch. It is very important that when the Z-bar shoe is fabricated, a frog plate is used to recruit the frog to share the load. The author is reluctant to use a Z-bar shoe as this type of shoe does not unload or protect the section of the foot that is not covered by the shoe.

Traction Devices

Various devices can be added to the ground surface of a shoe to increase traction. Traction devices also can influence the speed and direction of landing and break-over. Calks are projections of almost any size and shape—although most are round, square, or rectangular—on the ground surface of a shoe (Figure 11.25). Blocks, stickers, and studs, made of steel or steel with a tungsten carbide core, are all different types of traction devices or calks. Toe grabs on racing plates are essentially curved, elongated calks. Borium, tungsten carbide particles in a brass matrix, is brazed onto the surface of a shoe with an acetylene torch to create a roughened surface to prevent slippage as well as wear of the shoe. Horseshoe nails with tungsten on the heads of the nail can be used to attach the shoe and simultaneously apply traction. These nails are helpful in cold areas with ice and snow.

Some calks are permanent, whereas others are temporary and can be removed. Permanent calks are forged into the heel of the shoe, molded into the shoe at the time of manufacture, or welded or brazed onto the shoe at the time of fitting. Drive-in calks are semipermanent (can be changed if necessary) and are driven into a pre-drilled hole in the shoe. Temporary calks, called screw-in calks or studs, are screwed into drilled holes that have been tapped (create threads) so they can be inserted and removed as needed. Plastic inserts or cotton can be used to plug the hole when not in use. The size and shape of the studs may be changed with the various athletic pursuits or ground conditions. Calks may be positioned at any point around the circumference of the shoe. The choice of whether to use calks, which type to use, and where to position them is usually based on trainer and farrier preferences and the horse's performance discipline. Bilateral heel calks typically are used on jumpers and event horses. Racehorse plates may be equipped with toe grabs, with or without one or two heel calks (blocks or stickers). Draft animals usually have shoes with biaxial heel calks and less frequently a large toe calk.

Any traction device placed on the surface of the shoe inevitably alters the mechanics of the foot by altering the way it contacts the ground. The harder the ground surface or the taller the calk, the greater the effect. Calks concentrate stress in the wall immediately proximal to the calk. Therefore, the lowest, broadest calk compatible with adequate traction is recommended. A single calk at

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either the heel or toe alters mediolateral and dorsopalmar orientation. A single heel calk acts in much the same way as an extension or trailer, causing the foot to turn toward the side with the calk as the foot lands. Symmetrical placement of two pairs of calks, one pair on either side of the toe and one pair at the heels, encourages the foot to break-over in the center of the toe.

PLACEMENT AND APPLICATION OF THE SHOE

When a shoe is placed on the solar surface of a horse's foot, the horse is no longer standing on its foot; rather, it is standing on a shoe. Therefore, it is logical to place a shoe with biological congruency such that it conforms to the trimmed foot. For example, most horses have a natural wear pattern on the toe of the front shoe; therefore, the shoe should incorporate some form of break-over enhancement to simulate the animal's natural wear pattern. The shoe should be accurately fitted to the outline of the properly prepared hoof wall with the shoe placed central to the widest part of the foot (Figure 11.26). The shoe should be constructed from the lightest and simplest material that provides traction, protection, and adequate support to the foot for the work being performed.

The ground surface of the foot and the solar surface of the shoe must be flat and level to provide a tight fit between the surfaces. The shoe is fitted so that its outer margin closely follows the perimeter of the hoof wall from the toe to the last nail hole in the shoe. It is acceptable to fit the shoe marginally palmar or planter to the perimeter of the hoof wall at the toe. From the last nail hole to the end of the branch, the shoe should extend 0.0625–0.125 cm beyond the wall; or this distance can be varied according to the conformation of the foot. This protrusion of the shoe beyond the hoof wall is termed expansion. The author prefers the branches of the shoe to extend 1–2 cm beyond the end of the trimmed heel.

The shoe should be placed (or the nail holes punched such that they allow the nails to be placed) in the white line. Nail hole location is important to prevent interference with the movement of the palmar/plantar foot—the last nail hole in the branch of the shoe should be located at or forward of the widest part of the foot. The abaxial end of the branch at the heel should be rounded to provide sufficient space between the frog and branches of the shoe to pass a hoof pick to clean the sulci. The solar surface of the shoe can be concaved toward the inner margin to prevent sole pressure. The fitted shoe should lie central to the widest part of the foot with similar approximate distances from this point to break-over and from this point to the base of the frog. If the heels of the hoof capsule are damaged or do not correlate with the base of the frog, the branch of the shoe can be used to increase the ground surface of the foot by extending the branch to the base of the frog. If the shape of the solar surface of the foot is not ideal, the shoe should be fitted to provide even distribution of weight and, when possible, the shoe should be placed to accentuate the ideal shape.

Preparing the nail holes and driving the nails is often an overlooked part of farriery. If the hoof capsule is thought of as a cone, the necessity of driving the nails at the same angle becomes apparent. In this regard, hand-forged shoes provide an advantage of being able to angle the nail holes when the shoe is punched; this is called nail pitch (Figure 11.27). The nail holes in manufactured or machine shoes are stamped in bulk, so the opening on the solar surface of the shoe is closer to the outer margin of the shoe and the nail hole is not angled. When a nail

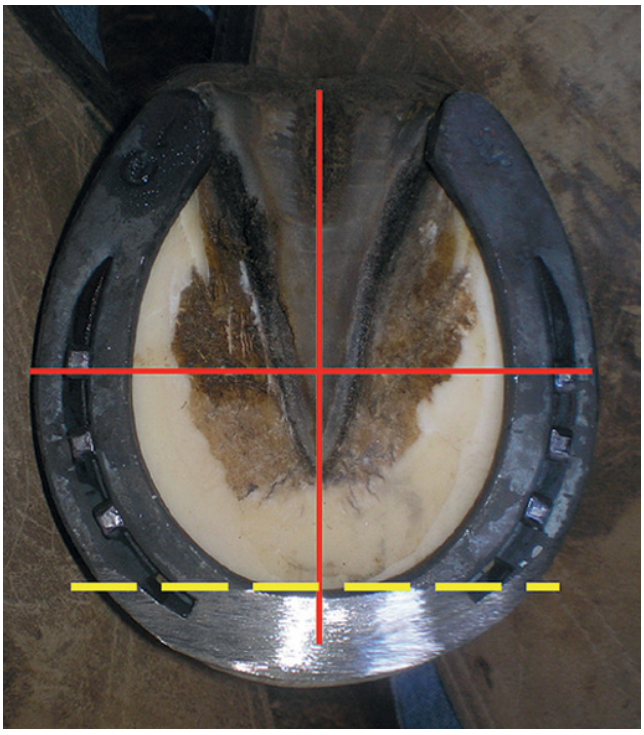


Figure 11.26. The shoe is fitted such that the widest part of the foot is located in the middle of the shoe (red lines). Note the break-over created in the shoe (dotted yellow line).



Figure 11.27. Nail pitch. Note the slope of the first two nails and the decreased pitch of the heel nail.

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is placed through a nail hole in a manufactured shoe, the shank of the nail exits straight rather than being angled to the slope of the hoof capsule. This prevents the nail from gaining solid purchase and height within the hoof wall. Low nailing is usually a precursor to cracks and fissures in the hoof wall. This should and can be easily corrected by re-punching the nail hole at an angle and by back punching the nail hole on the solar surface of the shoe.

Back punching is accomplished by using a pritchel placed on an angle adjacent to the inner side of the nail hole and directed toward the outer side of the shoe. The pritchel is used to create a small notch by removing a tiny piece of steel or aluminum material from the opening. This allows a nail inserted in the re-punched hole to immediately assume an appropriate angle.

The nail head must also have a tight fit within the nail hole. There should be no movement of the nail shank when the nail head is tapped down in the nail hole because movement will cause the shoe to loosen shortly after it is applied. If movement occurs, either a different nail with a different size or style of nail head should be used or the nail hole should be altered to accommodate the desired style of nail. The fewest number of nails and the smallest size of nail that will hold the shoe securely in place during the shoeing interval should be used to attach the horseshoe. Clips on the shoes will prevent shear on the nails and thus allow fewer nails to be used.

The horseshoe nail is unique in that there is a bevel next to the point that forms a short, single-sided wedge with the slanting side directed from within to without. On the same side of the bevel, there is a pattern or trademark located on the head of the nail that can be seen and sometimes felt. To be driven into the foot properly, the bevel and roughened side of the nail head must face toward the inside or midsagittal section of the foot. Another way to remember this is that the frog must be able to see the pattern or trademark on the head of the nail. If the nail is placed with the bevel to the outside, the nail will not exit the hoof wall and may enter the sensitive structures of the foot.

Before nailing the shoe, the author removes a thin slice of sole adjacent to the white line from quarter to quarter to discourage sole pressure. Two or three nails are driven into each branch of the shoe without excessive pounding, paying strict attention to the sinking and sound of the nail as it is driven through the hoof wall. Nails should be driven in the direction in which it is desired that they pass through the hoof wall and exit approximately 2–3 cm above the junction of the shoe and the hoof or approximately one-third of the distance from the ground surface of the hoof to the coronary band. The ends of the nails that exit the hoof wall should be immediately wrung off or bent over against the hoof wall to prevent injury to the horse or farrier if the horse pulls its foot away. After the desired numbers of nails are placed in the shoe, the nails can be tightened or drawn up by placing a clinching block under the bent edge of the nail and striking the head of the nail with a hammer. The author does not perform this step because it often draws the shoe too tight against the sole–wall junction, which can cause discomfort in a horse with a thin sole or hoof wall.

Finishing the foot is generally accomplished with the horse's foot placed on a foot stand. On the outer surface of the hoof wall, the bent ends or points of the nails should be cut with nail cutters so that approximately 3–4 mm of the nail protrudes, which will form the clinch. A rasp or a hoof gouge is used to clean or remove the disrupted horn from under the nail where it emerges from the hoof (Figure 11.28). Using clinching tongs, the end of the nail or clinch is pulled down firmly against the hoof wall or into the small channel created by the gouge (Figure 11.29). A clinch is sufficiently long when it equals the width of the nail at the bend. The holding power of the clinch is in the bend rather than the length of the clinch itself. Finally, the hoof wall and clinches are smoothed with a rasp, and the entire area is sanded with a commercial sanding block to remove any rasp or file marks. This gives the finished hoof a natural appearance without rasp marks, especially if hoof dressings are applied.



Figure 11.28. Hoof gouge. *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.



Figure 11.29. Clinching tongs. *Source*: Courtesy of FPD Farrier Products Distribution, Shelbyville, KY 40066.

NON-NAIL ALTERNATIVES IN FARRIERY

The use of various adhesives to attach shoes has become popular. The glue-on shoe can be attached to the hoof with an adhesive either applied directly to the hoof wall or by tabs or a cuff that are attached to the circumference of the shoe and extend proximally. The cuffs are made of semirigid plastic or synthetic cloth. Alternatively, the adhesive may be directly interposed between an aluminum shoe and the ground surface of the wall and immediately adjacent to the sole.[15]

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Attaching a shoe with adhesives may offer several advantages over nailing, such as when nailing is painful, when there is insufficient wall for nailing the shoe, and as a therapeutic option. However, there are disadvantages as well. Recently, it has been shown that adhesive attachment of a shoe significantly limits the expansion of the palmar section of the foot.[19]

Glue-on shoes are more expensive and more time consuming to apply, and the application of adhesives to the side of the wall decreases the quality of the underlying horn over time. There is also a perception that glued-on shoes do not stay on as well as nailed-on shoes, but this impression is in part the result of poor case selection or poor application. Glue-on shoes should be considered a transient device to produce hoof mass or to treat a hoof disease or defect before reverting back to applying traditional shoes or leaving the horse barefoot.

Glue-on shoeing has been used extensively since the introduction of the acrylics (polymethylmethacrylate) (Equilox International, Inc., Pine Island, MN) and urethanes (Vettec Hoof Care Products, Oxnard, CA). These composites consist of a resin and a catalyst that polymerize in a matter of minutes when mixed together, forming a bond between the hoof wall and the shoe. Heat is generated in this process because it is an exothermic reaction. Once the composite has cured, it has a flexible consistency very similar to the outer hoof wall of the foot.

Indications for Glue-On Shoes

Glue-on shoes can be used for damaged hoof walls, hoof walls with poor horn consistency (especially during the summer months), or hoof walls that are thin and benefit from not using nails to attach the shoe. Occasionally, a horse will have its feet trimmed too short while being shod and become sore. In this case glue-on shoes lift the sole off the ground without placing nails in a short foot with minimal hoof wall thickness. Soundness can be restored almost immediately. However, it appears that the use of glue-on shoes has increased due to lack of farriery skills to apply appropriate traditional farriery.

In horses with chronic laminitis, realignment of the distal phalanx within the hoof capsule can be readily achieved with glue-on technology. Combined with the appropriate trim, glue-on shoes permit realignment of the solar surface of the distal phalanx with the ground. In WLD, the accepted treatment is resection of the affected hoof wall along with thorough debridement. Often the farrier is left with inadequate hoof wall to attach a shoe with nails, in which case glue-on shoes provide a means to attach the shoe while leaving the resected area open for observation and daily treatment. Long-toe, underrun heel foot conformation often leads to a persistent low-grade lameness and has always presented a challenge for veterinarians and farriers to manage. In this case, a composite can be used to extend the heel further palmarly to increase ground surface, and when combined with the appropriate shoe, the extended or created heel is placed in a better functional position under the limb. Furthermore, the composite that is placed between the shoe and the foot prevents the abrasive movement of the heel of the hoof capsule against the shoe. This lack of movement prevents further wear, allowing regrowth of the hoof wall and bars at the heel. However, the composite between the foot and the shoe appears to inhibit normal heel expansion and may lead to contracted heels if this practice is continued for any length of time.

Methods of Glue-On Farriery

There are two accepted methods to glue on a shoe. In the direct method, an aluminum shoe is attached with an adhesive (Equilox International, Inc., Pine Island, MN) to the ground surface of the foot (Figure 11.30). An aluminum horseshoe is pre-shaped to the hoof, and the hoof-bearing surface of the shoe is roughened with a grinding disc to increase cohesion. A two-part acrylic combined with fiberglass strands is used to bond the prepared shoe to the hoof wall. Meticulous attention to preparation of the hoof is essential because the heat from the exothermic reaction may lead to an abscess and the outer hoof wall may degenerate with continued use.

The second method is the indirect method. Here, a patented aluminum, rubber-soled horseshoe attached with a fabric cuff (Sound Horse Technologies, Unionville, PA) or an aluminum shoe, which is enveloped in rubber, forming a one piece unit with a cuff (GluShu, Gateshead, United Kingdom). The appropriate size shoe is selected, the cuff is trimmed as necessary, the outer hoof wall is prepared, and a composite is applied to the outer wall to bond to the cuff. Sole depth may increase with these shoes as the fabric or rubber cuff becomes the hoof wall and a urethane rim pad inserted between the shoe and the solar surface of the foot provides an interface to decrease concussion and eliminate sole pressure. However, the constrictive nature of the cuff encompassing the hoof wall must be questioned. Another indirectly applied shoe is an aluminum shoe encased in plastic with attached tabs around the circumference of the horseshoe; the shoe is glued to the hoof wall with cyanoacrylate adhesive (Equilox International, Inc., Pine Island, MN).

Glue-on shoes can be expensive and time consuming to apply. Success depends on proper hoof wall preparation, shoe fit, and composite application. Glue-on shoes



Figure 11.30. Direct method to apply a glue-on shoe in which the shoe is attached with a composite to the ground surface of the foot.

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should not be used to replace the necessary farrier skills that are used to resolve routine and difficult foot issues, nor should glue-on technology be used to replace the basic fundamentals of good basic horseshoeing.

All composites cure with an exothermic reaction; therefore, caution should be exercised when repeatedly applying these products to a thin or compromised hoof wall because the long-term effects on the hoof wall have not been determined nor has marked improvement been reported. Nevertheless, glue-on shoe technology offers another option to attach horseshoes to the ground surface of the equine hoof when a non-nailing, non-traumatic alternative is indicated.

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FARRIERY FOR COMMON HOOF PROBLEMS

STEPHEN E. O'GRADY

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CONDITIONS OF THE FOOT THAT RESPOND TO FARRIERY

The biomechanics of therapeutic shoeing and its scientific application have been described in Chapter 8. In this section the basic principles of farriery will be applied to help resolve several types of abnormal hoof conformation or hoof capsule distortions that can occur in both lame and non-lame horses. Farriery approaches to some of the common clinical hoof problems encountered in equine practice will also be discussed. Hoof capsule distortion and the four types of abnormal hoof conformation that will be addressed include the long toe and/or low heel, the club foot, sheared heels and mismatched feet.

Radiography and Farriery

One of the main diagnostic tools besides physical examination that can help assess hoof wall distortions and abnormal conformation is radiography. Radiography can be used to assess both the external hoof conformation and many of the internal structures of the foot.[4,5,22,31,39] Considerable information can be obtained from the image regarding the overall shape and mass of the hoof capsule, the soft tissue structures, and the position of the distal phalanx within the hoof capsule. Lack of performance or many subtle lameness conditions that are localized to the foot are caused by hoof capsule distortions, poor foot conformation, improper landing patterns, and soft tissue damage that result from excessive biomechanical stresses being placed on the hoof capsule, the podotrochlear apparatus, and the DIP joint.

Lateral to medial and dorsopalmar 0° horizontal (DP) views of the foot can be used as a precise guide to implement basic or therapeutic trimming and shoeing (Figure 11.31). From the lateral medial view, the hoof-pastern axis (HPA), center of rotation (COR), sole depth, the angle of the solar border of the distal phalanx, break-over, and the proportions of the ground surface on either side of the COR can be assessed. When the solar plane of the distal phalanx shows a negative palmar angle, the need for heel elevation and the amount to apply, as well as whether the integrity of the structures of the heel will accept the pressure, can be determined. The dorsopalmar 0° horizontal view allows the clinician to evaluate the lateral medial orientation of the distal phalanx within the hoof capsule, the position of the distal phalanx relative to the ground, and the effect of the position of the distal phalanx on the joint spaces of the digit. The need to assess the position of the hoof capsule relative to the long axis of the digit is often overlooked. The DP radiographic view allows the position of the coronary bands to be assessed in the case of sheared heels and the position of the distal phalanx relative to the disparity in heel heights.

Hoof Capsule Distortion

The hoof capsule is composed of the hoof wall, sole, frog, and bulbs of the heels. Its unique continuous bond between its components form a casing on the ground surface of the limb that affords protection to the soft tissue and osseous structures enclosed within the capsule.[13,33,36] The hoof wall is a viscoelastic structure that has the ability to temporarily deform under load and then return to its original shape when the load or weight

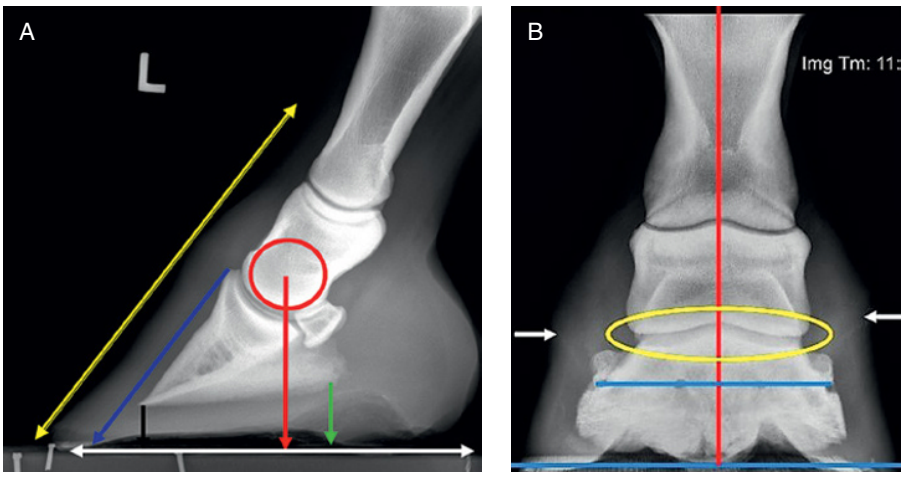


Figure 11.31. (A) Lateral radiograph that illustrates hoof-pastern axis (yellow), center of rotation (red), sole depth (black) and palmar angle of distal phalanx (green), break-over (blue), and the proportions of the ground surface (white). (B) DP radiograph that illustrates the axial alignment of the digit (red), position of the foramina in the distal phalanx relative to the ground (blue), alignment of the joint space (yellow), and position of the coronary band at the heel bulbs (white).

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is removed due to its elastic nature. It is well accepted that abnormal weight distribution on the foot or disproportionate forces placed on a section of the hoof wall, over time, cause it to assume an abnormal shape.[6,7,13,33–36] These abnormal stresses within the foot will also predispose the foot to injury or disease. Increased stress or weight-bearing placed on a section of the hoof capsule may originate from a single source or from multiple contributing factors such as weight of the horse, abnormal limb conformation, strike pattern of the foot, amount of work, type of footing, and inappropriate farrier practices. The responses of the foot to stress and differing patterns of weight-bearing are threefold: change in position of the coronary band, change in hoof growth rate, and deviation(s) of the hoof wall.

Excess stress placed on a section of the hoof capsule can manifest itself in a variety of ways including compressed growth rings, flares or underrunning of the hoof wall, dorsal migration of the heels, and either focal or diffuse displacement of the coronary band.[34,36] As hoof capsule distortion of the forelimbs is commonly associated with lameness and various disease processes, only the forelimbs will be considered in this chapter. As the "normal" foot has not been defined, each view of the foot will begin with what is perceived to be an ideal, good, or healthy foot.[2,31,33–36,39] Palpation of the hoof capsule often complements the visual examination, and the areas where palpation is relevant will be included. The goal of evaluating the hoof capsule is to identify deformation and changes in the growth pattern that may indicate abnormal distribution of forces (stresses) on the foot. As hoof capsule distortion and abnormal loading usually accompany lameness, farriery will form part of or sometimes the entire treatment to reverse the process. Farriery is used to help redistribute the load and help improve or resolve the hoof capsule deformation.

The Mechanism of Distortion

Evaluation of the hoof capsule morphology will indicate where the hoof wall is unduly stressed; however, the evaluation has to be coupled with an understanding of the abnormal distribution of forces that lead to hoof capsule deformation. Understanding the biomechanical forces leading to hoof capsule distortions is also helpful for the clinician when applying the appropriate farriery to modify these stresses. There are many excellent reviews of basic biomechanics of the hoof in the veterinary literature.[6,7,13,33,35] Increased load or weight-bearing by a portion of the wall has three potential consequences: (1) deviation of the wall outward (flares) or inward (underrunning) from its normal position, (2) movement of the wall proximally, or (3) decreased hoof wall growth.[33–36] A reduction in load or weight-bearing on the foot generally has the opposite effect. Briefly, in the standing horse, the weight of the horse borne by the limb is supported by the ground, which opposes the weight with an equal and opposite force. The force exerted on the foot by the ground is termed the ground reaction force (GRF). The term center of pressure (CoP) is the point on the ground surface of the foot through which the GRF acts on the foot. The CoP varies among horses but is approximately located in the center of the solar surface of the foot in the standing horse.[6,7,13,33,35] However, when the horse is moving, the location of the CoP changes dynamically. The position of the CoP at any point in the stride determines the distribution of forces between the medial to lateral and the dorsal to palmar aspects of the foot. When the CoP is moved to one side of the foot, that side of the foot will be subject to increased forces. If the CoP is moved in a palmar direction, the weight-bearing or load on the palmar hoof wall will be increased.

Relating the CoP to hoof capsule distortions, if the CoP is located more medially, over time, a medial hoof wall flare (bending) and a lateral wall underrunning will develop. If the CoP is moved dorsally due to increased tension in the DDF tendon (DDFT), the hoof capsule will likely develop a higher heel with a flare in the dorsal hoof wall. Farriery is used to change the location of the CoP (to some extent) and change the distribution of forces on the ground surface of the foot. When evaluating hoof capsule deformation, limb conformation should be considered as abnormal limb conformation affects the landing pattern and stance phase of the stride. Few horses have ideal limb conformation, and any change in conformation is likely to change the distribution of forces within the hoof capsule leading to deformation.

Low-Heel Foot Conformation

The palmar section or heel area of the foot plays a critical role not only in soundness but also in the health and maintenance of the horse as a whole. It is subject to almost incomprehensible external and internal biomechanical forces, which inherently predispose the palmar foot to injury or disease.[2,19,20,22,31] However, the application of farriery for this section of the foot, whether routine, or for addressing abnormal conformation or prevention remains controversial. Long toe, low heel is the generality used to describe this foot conformation, but low heel accompanied by a long toe may be the preferred term. A low or underrun heel conformation is defined as the angle of the heels being considerably less than the angle of the dorsal hoof wall. When this difference is considerable, it is characterized by a broken-back HPA—the angle of the dorsal hoof wall being lower than the angle of the dorsal pastern. A common cause is not trimming the heels appropriately, which causes them to grow forward and lose their angle. The low-heel foot conformation is so common in equine practice, especially in Thoroughbred horses, that it is thought to be normal. In one study of foot-related lameness, low-heel conformation was found in 77% of the horses,[47,48] whereas in a different group of horses that where lame without evidence of navicular disease, 73% had underrun heels.[52] In another study of Thoroughbred racehorses, underrun heels were found in 52% of the horses.[14]

A low hoof angle results in dorsiflexion of the DIP joint, which concentrates weight-bearing on the palmar section of the foot and increases strain on the DDFT. The low-heel conformation changes the angle of insertion of the DDFT on the distal phalanx, which places increased stresses on the navicular apparatus.[8] When excessive toe length is present, the increased stresses in the palmar foot are potentiated at break-over. This abnormal hoof conformation also removes the foot's ability to absorb concussion and dissipate the energy of impact, thus

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contributing to all forms of palmar foot pain, chronic heel bruising, and DIP joint synovitis. There is also experimental evidence that a low hoof angle compromises circulation in the palmar section of the foot.[38,50]

The terms low heel and underrun heels or even collapsed heels are often use interchangeably; however, it may be helpful to distinguish between the two. In the case of a low heel, the angle of the heel is markedly lower than the angle of the dorsal hoof wall; however, the structure of the heel is relatively good in that the buttress, angle of the sole, tubular horn, and bars (termed the heel base) that comprise the heel structure are intact. With the underrun heel, the structure of the heel is compromised such that the hoof wall at the heel is thin, separated from the adjacent sole and rolls under the hoof in an axial direction, the angle of the sole is missing, the bars are compromised, and the heel–ground surface of the hoof wall does not reach the base of the frog.

As low or underrun heels progress, this condition can be readily observed both visually and radiographically, the point at which the angle of the solar border of the distal phalanx is lower palmarly than it is dorsally. A common error in routine farriery is not continually trimming the ground surface of the hoof wall at the heels to the base of the frog or not trimming the hoof wall at the heels to the same plane as the frog. This type of trimming is potentiated by applying a shoe that is too small. A negative angle of the solar border of the distal phalanx, as noted radiographically, indicates that the soft tissue structures (frog, digital cushion) in the palmar foot are underdeveloped or have decreased in mass, usually due to damage from overloading (Figure 11.32).[2,8,20,22,31] The compromised structures of the palmar section of the foot allow the palmar margins of the distal phalanx to descend distally. This type of hoof conformation alters the structural mass of the foot as the compromised heels lose the ability to accept weight and dissipate the energy of impact. Furthermore, it appears that sufficient soft tissue structures in the palmar section of the foot are necessary to act as a framework for the regrowth of any significant hoof wall in this area. Biomechanically, it changes the angle of insertion of the DDFT on the distal phalanx and increases the peak force on the navicular bone and navicular bursa.[8]

The farriery for low or underrun heels is difficult, and often the conformation of the heels can only be maintained rather than improved.[2,22,31] The traditional treatment for underrun heels is to use an egg-bar shoe, often accompanied by some form of heel elevation to raise the angle of the heels and correct the broken-back HPA that is associated with this condition. Traditionally, these shoes have been fitted long to help extend the heels with the idea that the increased ground surface of the shoe supports the palmar section of the foot and the egg bar supports the heels. However, it is questionable whether support can be applied to compromised structures (heels) that no longer have the structural mass or integrity to accept weight. The egg-bar shoe moves the GRF palmarly, but the length of the bar acts as a moment arm or lever on the heels, creating excessive pressure. The egg-bar shoe places the bulk of the weight-bearing on the soft tissue structures of the already compromised palmar foot, thus preventing further hoof wall growth at the coronet. Furthermore, the length of the bar creates a heel-first landing that when coupled with the leverage puts excessive pressure on the compromised heels.

Farriery for this condition should be directed at improving the heel structures when possible, utilizing all of the structures in the palmar foot to share the load and heel elevation when possible. Allowing the horse to remain barefoot for a period of time with controlled exercise can markedly improve the heel structures. This will eliminate the movement of the hoof wall against the heels of the shoe to friction against the ground and will



Figure 11.32. Long-toe, low-heel foot conformation. Note the broken-back hoof-pastern axis and the acute angle of the coronet (A). Lateral radiograph revealing the angle of the solar border of the distal phalanx is negative (B). Note the lack of structural mass under the palmar process of the distal phalanx and the "knob" shape of the heel blub.

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load all of the structures in the palmar foot, both of which will tend to increase the ground surface of the hoof wall at the heels.[24]

Foot preparation for low heels begins by visualizing the two basic landmarks on the ground surface of the foot: the widest part of the foot and the base of the frog. The palmar section of the foot is trimmed using the widest part of the foot as a starting point; using a rasp the heels are then trimmed to healthy horn, making sure that all of the structures of the heel and the frog are on the same horizontal plane. The toe is shortened accordingly, again using the widest part of the foot as a guideline. With low-heel conformation, the frog is often situated distally below the level of the hoof wall at the heels. When a prolapsed frog is present, the shoe should be removed, any loose or exfoliating horn is trimmed from the frog, and the horse stood on a hard surface such as a rubber stall mat for 24–48 hours. An alternative to this is to fabricate a frog pad from a full wedge pad to place focal pressure on the frog, tape it on the foot, and stand the horse on a hard surface. This technique creates a level, flat surface in the palmar foot with all structures on the same plane. Having the structures, regardless of their integrity, on the same horizontal plane redistributes the weight creating a "load-sharing" effect.

The trim is complemented by a shoe that attempts to place the load over the entire palmar foot, not just on the hoof capsule at the heels (load sharing concept), and provides heel elevation to correct the HPA when possible. If the structures of the heel are intact and the hoof wall angle simply needs to be raised, an open aluminum wedge shoe or an open steel or aluminum flat shoe with a wedge pad can be used to achieve the desired heel angle. If the heels are damaged or compromised, a load-sharing effect can be accomplished by using a straight-bar shoe with a pad or degree pad placed between the shoe and the foot. A properly fitted heart-bar shoe is useful if the frog and digital cushion have sufficient structural mass to withstand the pressure from the frog plate. An open steel or aluminum shoe with a plastic or aluminum stability or "spider" plate riveted to the shoe, a thin heel plate welded or riveted between the branches of the shoe, or a plastic bar wedge attached to the shoe can be used to distribute the load. Holes are drilled in the heel plate or bar wedge, and some form of silastic material is placed underneath the plate or wedge to create a deformable interface to spread the weight-bearing function over all the structures in the palmar foot. The holes drilled in the plate or pad are necessary to keep the impression material in place. Impression material is placed on the ground surface of the entire foot, in the case of the spider plate or starting in the middle of the frog and extending palmarly as far as desired with the others. The shoe and plate or pad are placed on the foot, and the heel of the shoe is pressed gently into the impression material, forming a slight wedge. Two nails are placed in the toe of the shoe, and the foot is held off the ground until the impression material cures in the desired position (Figure 11.33). Glue-on technology may be helpful to improve the structures in the heel but should not be applied long term because damage to the hoof wall is thought to occur from the heat generated by the composite.[53]

Farriery for low-heel conformation is often based on trial and error and combinations of various methods but depends on assessing the integrity of the structures present, the footing, and the athletic pursuit of the horse. Any form of heel elevation should be accompanied by

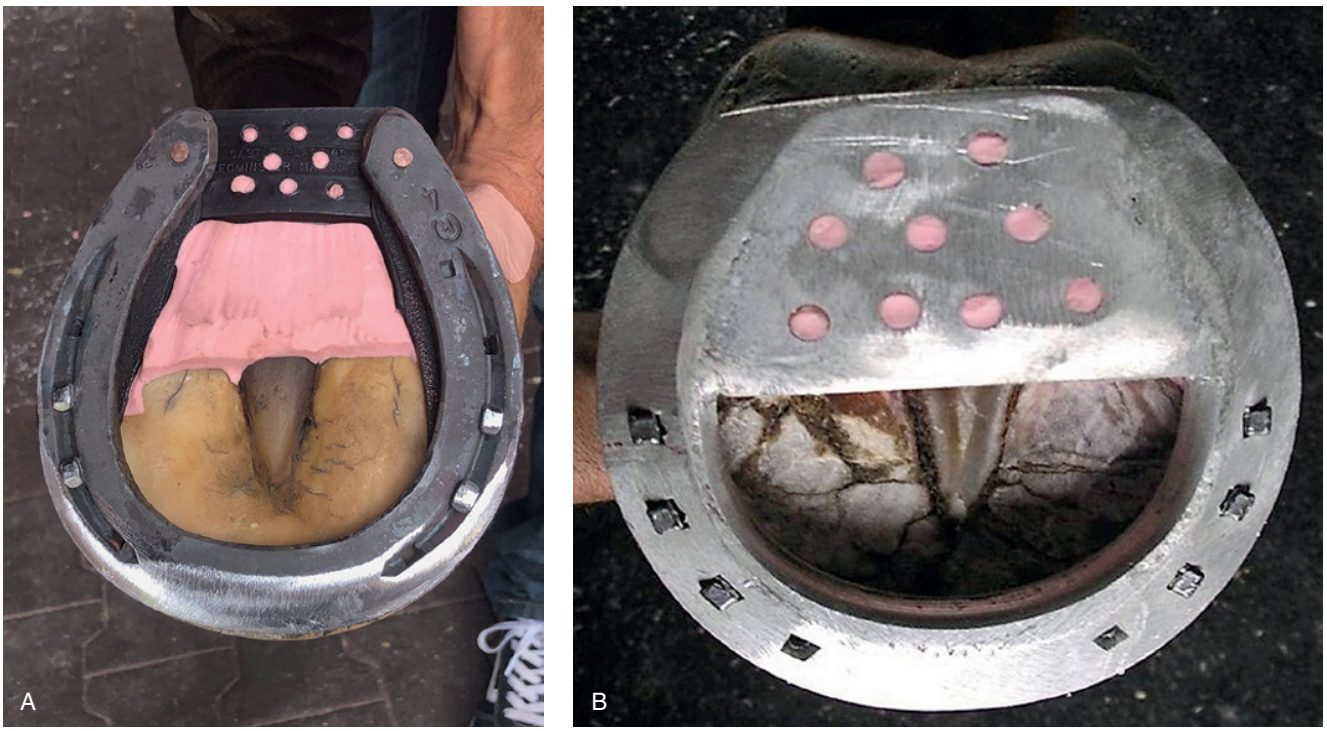


Figure 11.33. Impression material is placed in the palmar section of the foot, and a pad is used to distribute the load across the heels (A). Heel plate shoe with impression material is placed under the palmar section of the foot (B).

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enhancing break-over to remove any leverage at the toe. Moving break-over palmarly can be accomplished in a variety of ways such as rolling or rockering the toe of the shoe or creating a rolled toe in the shoe using a hand grinder where the break-over begins at the inner branch of the shoe and extends dorsally in a tapered fashion. Moving break-over palmarly/plantarly decreases the moment applied to the DIP joint and appears to decrease the maximum tension in the DDFT, which occurs toward the end of the stance phase at the beginning of break-over.

Club Foot

A club foot has a high hoof angle associated with a flexural deformity of the DIP joint, while an upright foot will have a high hoof angle but has a straight HPA. A club foot is characterized by a broken-forward HPA, which occurs when the angle of the dorsal hoof wall is greater than the angle of the dorsal pastern (Figure 11.34).[12,26,29] This broken-forward HPA or flexural deformity is created by some degree of shortening of the musculotendinous unit (DDFT and associated muscle bellies), causing the DIP joint to be drawn into a flexed position.

Flexural deformities have been reported as a cause of decreased athletic performance, a shortened stride length, and chronic low-grade lameness in the mature horse.[29,48,53] A shortening of the musculotendinous unit causes more hoof wall growth at the heel (compensating for the shortened muscle tendon unit) than at the toe, and the frog generally recedes below the ground surface of the hoof wall due to the excessive heel height. The energy of impact is now assumed by the hoof wall, bypassing the soft tissue structures in the heel area and transferring the load directly onto the bones of the digit through the laminar interface. Hoof abnormalities associated with a club foot conformation are thin flat soles, poor dorsal hoof wall consistency, toe cracks, hoof wall separations, and white line disease (WLD). A club foot promotes toe-first landing, a decreased cranial phase of the stride, and increases weight-bearing in the dorsal aspect of the foot. Poor performance and injuries associated with a high hoof angle are thought to include DIP joint inflammation due to abnormal position/loading of the joint, sole bruising, and increased strain on the suspensory ligaments of the navicular bone.

High hoof angles with no or mild phalangeal misalignment can generally be improved by gradually lowering the heels in a tapered fashion from the apex of the frog to the heels. Shortening the intervals between trimmings is helpful as it will minimize excessive heel growth. This increases the ground surface of the foot and attempts to reestablish weight-bearing on the entire solar surface of the foot. Break-over is moved palmarly at the same time to compensate for any increased tension in the DDFT created by lowering the heels.

Farriery for a club foot or high hoof angle with concurrent phalangeal misalignment is a greater challenge. Flexural deformities are usually diagnosed and treated while the horse is immature, although mild flexural deformities are often ignored or treated improperly in foals. When these animals enter training, mild flexural deformities can be exacerbated by chronic foot bruising, the type and the amount of exercise, ground surface, inappropriate farrier care such as improper or infrequent shoeing, or some type of underlying pathology.[29] The objective of farriery is to load the palmar section of the foot, compensate for the shortening of the DDFT, and improve the HPA when possible. Therefore, farriery is directed at trimming the heels, but the amount to remove can be difficult to determine. In mild to moderate club feet, the amount of heel to be removed can be estimated by placing the thick end of a 2° or 3° wedge pad under the toe of the foot and allowing the horse to stand on it. If the horse does not resent the tension placed on the DDFT, the heels can be trimmed the thickness of the wedge pad in a tapered fashion starting at the widest part of the foot. The toe is shortened by decreasing the length and removing the concavity from the dorsal hoof wall with a rasp. The

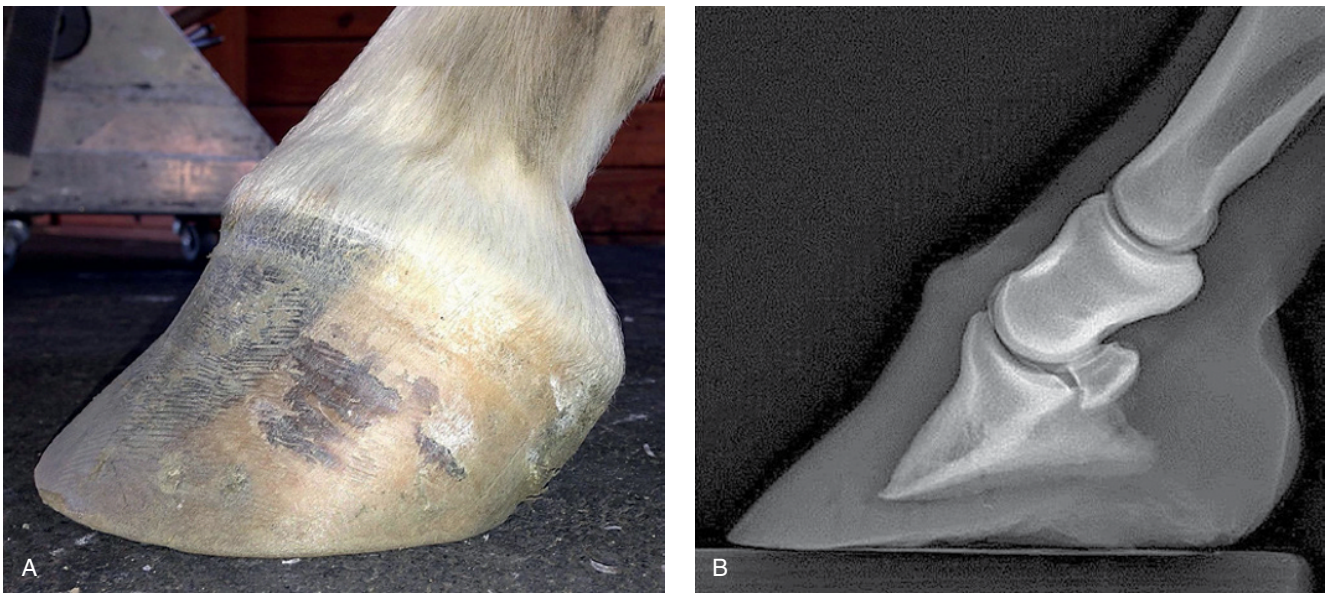


Figure 11.34. Example of a moderate club foot. Note the disparity of the growth rings and the broken-forward hoof-pastern axis (A). Lateral radiograph of this same foot (B). Note the flexural deformity of the DIP joint and the concavity in the dorsal hoof wall.

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trimmed foot is fitted with a shoe that extends 1–2 cm beyond the end of the heel and has the break-over forged or ground into the shoe starting just dorsal to the apex of the frog and tapering toward the toe to further decrease the stresses on the DDFT.

With a more advanced club foot, the heels should still be trimmed to load the heels and unload the toe, but heel elevation must be added to compensate for the shortening of the musculotendinous unit of the DDFT. This can be determined after the trim by placing the trimmed foot palmar to the contralateral limb to observe whether there is a space between the heels of the foot and the ground. If a space is noted, the author uses a wedge shoe or attaches a wedge pad or a bar wedge to the shoe in order to load the palmar section of the foot and to compensate for the shortening of the musculotendinous unit (Figure 11.35). This method allows the heel area to be weight-bearing but at the same time decreases the stresses in the DDF muscle tendon unit. Break-over is applied as described above.

Severe flexural deformities that result in chronic lameness can be treated by performing a desmotomy of the accessory ligament of the DDFT combined with the appropriate farriery.[29,46] See "flexural deformities" in Chapter 10 for further information regarding this procedure.

Sheared Heels

Sheared heels are a hoof capsule distortion resulting from displacement of one heel bulb proximally relative to the adjacent heel bulb (Figure 11.36).[18,19,22,30] This disparity between the lateral and medial heel bulb is generally 0.5 cm or greater. Sheared heels appear to develop as an adaption/distortion of the hoof capsule as a consequence



Figure 11.35. The foot can be placed palmar to the contralateral foot to observe whether there is a space between the heels and the ground, which corresponds to shortening of the DDFT muscle tendon unit (A). Heel elevation applied to a club foot after trimming (B).

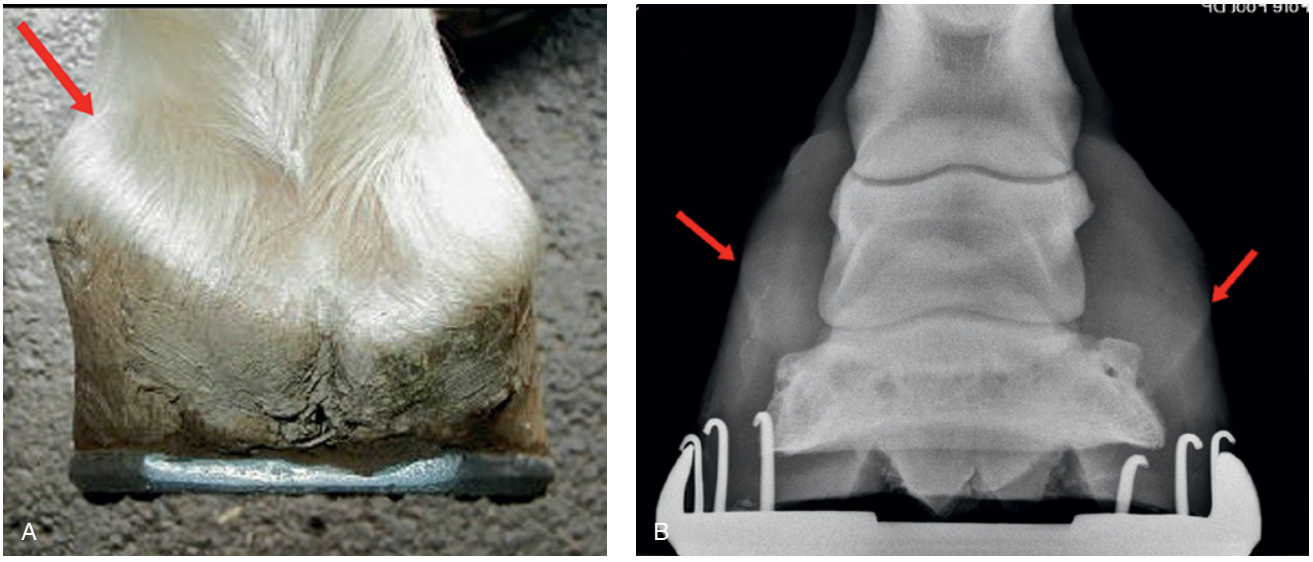


Figure 11.36. (A) Dorsopalmar view of a foot with sheared heels. Note the disparity between the length of the two heels and the deformation of the structures on the medial side of the foot (red arrow). (B) DP radiograph of a horse with sheared heels. Note the disparity in the height of the coronary bands (red arrows), yet the plane of the distal phalanx remains parallel with the ground.

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of limb conformation that results in an abnormal strike and loading pattern of the foot on the ground.[30] When the weight of the horse is not distributed uniformly over the entire hoof during the landing and stance phase of the stride, one focal area of the foot, usually a heel or heel and accompanying quarter, receives a disproportionate amount of the total load during impact and displaces proximally. Although a number of sound horses have this type of hoof capsule distortion, the author believes that when the adaptive ability of the hoof capsule is surpassed by the excessive load on one section of the foot, this type of hoof conformation can be the sole source of unilateral palmar foot pain. This pain could originate from the compression of the soft tissue located between the middle phalanx and the hoof wall when the heel is displaced proximally. The continual disproportionate loading and increased compressive stresses on one quarter/heel predispose the foot to subsolar bruising, corns, quarter/heel cracks, fracture of the bar, and a deep fissure within the base of the frog that is susceptible to thrush (Figure 11.36). This type of foot conformation is readily observed by picking up the foot and noting the relative disparity in distances from the heel of the hoof capsule to the hairline at the bulbs of the heels between the lateral and medial heel (Figures 11.36 and 11.37).

The etiology of this condition is not completely understood. Inappropriate lateral to medial orientation (balance) of the foot where the foot does not land flat has always been implicated, but this has not been substantiated. A correlation with rotational limb deformity (lateral) that changes the flight pattern of the limb and ultimately the manner in which the foot lands has also been suggested.[2,29] Furthermore, there appears to be a correlation between an offset distal phalanx within the hoof capsule and sheared heels. Commonly, the distal phalanx is offset laterally within the hoof capsule, which places additional load on the medial side of the foot, causing the medial heel to displace. The unequal distribution of vertical forces on a given side of the foot over time appears to result in a biological remodeling rather than the heel being pushed proximally or "jammed" to use the common farrier term (i.e. the heel is "growing out of shape rather than being pushed out of shape").[4,5,22,23,30].



Figure 11.37. Sheared heel with a fissure through the base of the frog (yellow arrow). Note the opposite direction of forces applied to the walls during landing (red arrows).

Biomechanically, the position of the coronary band is related to a balance between hoof wall growth at the coronary band and the rate of migration of the hoof wall distally. Furthermore, the rate of migration of the hoof wall is also a balance between an active process occurring in the lamellae to cause them to move distally and the force on the wall from the GRF. This appears to be the mechanism of displacement in horses with sheared heels/quarters (Denoix and Parks. Personal Communication, 2012). Proximal displacement of the soft tissue structures occurs palmar to the distal phalanx and appears to result from mechanical stress. To substantiate this statement, the author reviewed 50 DP radiographs on horses' feet with at least one heel bulb that was displaced proximally 0.5 cm or greater. In each case, the solar surface of the distal phalanx was basically horizontal (parallel) with the ground, indicating the disparity in heel height did not originate from the lamellae within the hoof wall located adjacent to the distal phalanx (Figure 11.36).[4,5]

Farriery is directed toward unloading the hoof wall and decreasing the forces on the displaced side of the foot. This is accomplished by improving the shape of the hoof capsule and landing pattern by applying the appropriate shoe. Intuitively, if the heel is longer on the displaced side (measured ground surface to hairline), it is reasonable to trim the displaced side and create additional ground surface under the affected side.

Again, when possible, the author will remove the shoes and trim the heels of the palmar foot such that the hoof wall and frog are on the same plane. The horse can be left without shoes for 24–48 hours or longer if necessary. Leaving the horse barefoot for a brief period allows the displaced heel to descend, the compressed area between the coronet and middle phalanx will widen, and the palmar section of the foot will assume a more relaxed position.

With the heels in a more acceptable position, the author uses a double trim method to address sheared heels in an attempt to improve and unload the displaced quarter/heel. The foot is trimmed appropriately using the guidelines of a parallel HPA, the COR, and the heels trimmed to the base of the frog.[22,23] If the displacement of the heel is significant, a wide-web steel or aluminum straight-bar shoe is fitted as symmetrically as possible underneath the long axis of the limb using the apex of the frog as a central marker.[4,5] The bar shoe effectively increases the surface area of the foot, provides more expansion (ground surface) on the displaced side that has a straighter wall, and decreases the vertical movement of the heel bulbs. Before applying the shoe, a second trim is performed under the proximally displaced quarter heel, which goes from 0 mm at the ipsilateral toe quarter (e.g. inside toe quarter for medial sheared heel) to an average of 7 mm at the affected heel. The amount of heel that can be removed with the second trim depends on the sole depth at the seat of corn and on the severity of the proximal displacement of the coronary band at the sheared heel.

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This amount of heel ideally corresponds to the difference in length/height between the two heels. Lowering the hoof wall at the quarter/heel will create a wedge-shaped space between the shoe and the hoof wall on the displaced side of the hoof, permitting the displaced heel to descend distally and settle into a more acceptable configuration. No nails should be placed in the shoe palmar to the origin of the second trim as this would inhibit the displaced section of wall from descending distally. This type of selective trimming combined with a straight-bar shoe effectively unloads the section of the heel/quarter that is being displaced and decreases the disproportionate forces on the foot (Figure 11.38). In less severe cases, an open steel or aluminum shoe can be used instead of a bar shoe adhering to the principles of trimming described above.

Mismatched Feet

The management of mismatched hoof angles remains a controversial subject for both the farrier and veterinarian. Mismatched feet are usually defined as forefeet conformation that have a high or upright hoof angle on one foot and a low hoof capsule angle on the contralateral foot (Figure 11.39).[13,35,36] The difference between the forefeet conformation of mismatched feet is variable in severity and can range from a high hoof angle with a straight HPA or a club foot with a flexural deformity in one foot to an overloaded low heel on the contralateral limb. The mass, integrity, and difference in heel height is usually the main contributing factor in creating the mismatched dorsal hoof angles (Figure 11.40). Limb length disparity has been suggested as a cause for mismatched feet although it has not been substantiated. Mismatched feet may contribute to poor performance, subtle lameness, and a shortened cranial phase of the stride. Traditional farriery seeks to elevate the heel on the low foot and therefore match the hoof angles of the forefeet. However, this practice should be discouraged, and treatment should be based on farriery principles to improve the structures and function of the individual foot.

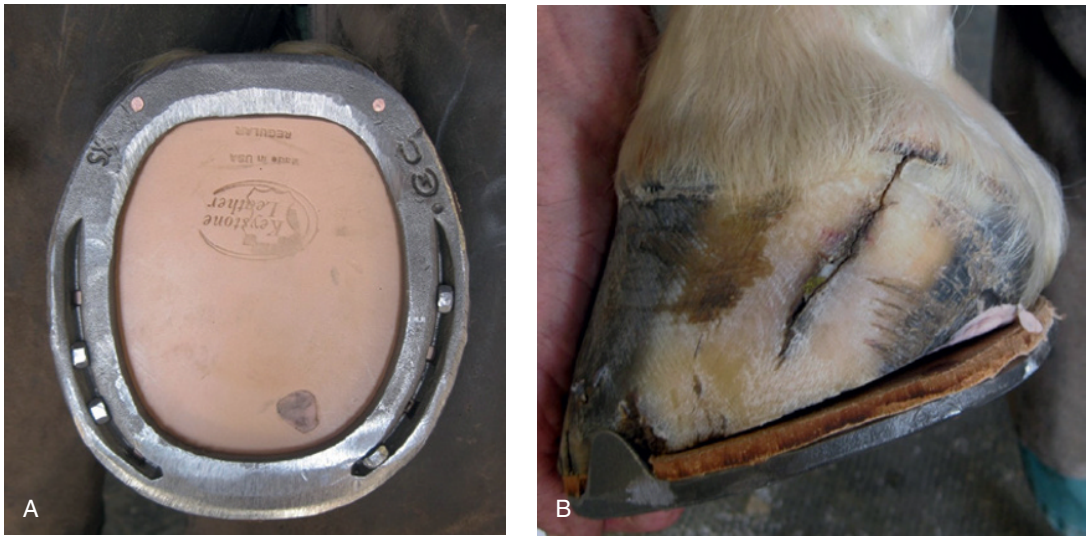


Figure 11.38. Straight-bar shoe used to stabilize the hoof capsule and decrease the vertical movement of the heels (A) shoe with the medial heel unloaded (B). Note the quarter crack, which is often present with a sheared heel.



Figure 11.39. A pair of mismatched forefeet. Note the different hoof angles (A, B) and the mild broken-forward hoof-pastern axis on the upright foot on the left (A).

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Horses with a disparity between dorsal hoof wall angles will generally have a straight HPA, and the hoof wall growth below the coronet from the toe to the heel will be uniform. In this case, the author suggests using good farriery principles to apply the appropriate trim and shoe each foot on an individual basis. Managing horses with mismatched feet where one foot has a markedly high hoof angle or a club foot becomes more complex, and the reader should refer to the previous section on the club foot. Horses with mismatched feet will present with a shortened cranial phase of the stride on the limb with the upright foot often coupled with a discernible lameness. Low or compromised heel structures may be noted on the opposite foot from overloading the heel secondary to excess weight on the contralateral foot over time. Managing these horses can be difficult, and the proper shoeing protocol may not be inherently obvious. It is common to see horses with mismatched feet shod with two different size shoes; often a smaller shoe is used on the upright foot with the misguided thought that the shoe will not be pulled. This practice should be discouraged as the ground surface on both forefeet should be the same.



Figure 11.40. A pair of mismatched feet shows the difference in the structural mass in the palmar section of the foot, which is primarily responsible for the difference in toe angle.

One of the objectives of trimming the upright or club foot is to redistribute the load to the palmar aspect of the foot to restore functionality and increase stride length. When approaching the foot with the low angle, the clinician is often inclined to wedge up the heels to improve the HPA and match the other foot. However, this will place additional stress on the already compromised heel structures. Although the HPA will appear improved immediately following the shoeing, the long-term effect is exacerbation of the low angle, further crushing of the heels, and prolapse of the frog below the ground surface of the foot. The heels should be trimmed back to the widest point of the frog if possible, and the toe length should be reduced as described in the section on long-toe, low-heel conformation. It is extremely important to obtain good-quality radiographs of the foot prior to trimming to determine the amount of heel and especially sole that can be removed. The CoP on a low angle foot is further palmar than that of the upright hoof, and therefore the shoeing protocol is directed at moving the CoP away from the overloaded heels. Additionally, redistributing the load or load sharing with the weight-bearing structures of the low angle foot may help decrease the forces directed to the heels. This can be accomplished with impression material, a pour-in pad, and a spider plate or a heel plate added to the palmar aspect of the shoe (Figure 11.41).



Figure 11.41. Mismatched feet showing a spider plate and impression material on the foot with the low heel (A) and a 2° wedge insert on the other foot with the high heel (B).

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CLINICAL CONDITIONS AFFECTING THE HOOF

Hoof Wall Defects

Hoof wall defects constitute structural damage to the hoof capsule and can range from superficial to full thickness. The defect can be vertical or parallel to the horn tubules or horizontal, which is perpendicular to the horn tubules of the hoof wall. Defects or cracks can be classified according to their location in the hoof capsule, such as toe cracks, quarter cracks, and heel cracks, and also according to their depth within the hoof capsule (i.e. full vs. partial thickness).

A horizontal hoof wall crack originates at the coronet as a disruption of the coronary corium and moves distally as the hoof wall grows. The disruption of the coronary corium is caused by a rupture at this site usually from an abscess or infection traveling proximally from the solar surface of the foot to the coronary band. Direct trauma to a section of the coronet can also occur contributing to a horizontal crack. Once the coronet is healed, horizontal cracks do not generally enlarge, and they grow out uneventfully. Vertical hoof wall cracks can either originate at the ground surface and extend proximally or begin at the coronet and extend distally. Contributing factors include direct hoof wall trauma or overloading and hoof capsule distortions.

Quarter Cracks

Quarter cracks are the most common hoof wall defect encountered and often cause foot lameness and/or decreased athletic performance in racehorses and sport horses. A true quarter crack is a full-thickness defect (extends into the dermis) that originates at the coronary band and travels distally, causing hemorrhage, instability, inflammation, and/or infection. Quarter cracks often bleed as a result of the invasion and movement within the dermis (Figure 11.42). Causes of quarter cracks may include trauma to the coronary band, preexisting damage to the corium from previous cracks or infection, abnormal hoof conformation, short shoes, or an abnormal landing pattern when the foot strikes the ground asymmetrically. Any one of these causes may place continuous excessive stress or pressure on a focal area of the proximal hoof wall. Quarter cracks result when excessive forces placed on a section of the hoof wall exceed the hoof capsule's ability to deform and accept additional load (Figure 11.38). Various techniques for stabilizing and repairing quarter cracks have been described; however, in the author's opinion, the most important aspect is to determine and correct the cause of the hoof wall defect.

Farriery

The author prefers, when possible, to remove the animal from work and apply the necessary farriery to address the hoof capsule distortion, rather than to repair the crack initially. If the appropriate farriery is applied, there can be substantial solid hoof wall growth proximal to the defect at the next reset in 4–5 weeks, indicating the abnormal forces or stresses in the wall with the defect have been reduced. Obviously, this approach is not an option in performance horses that must continue to compete.

Abnormal hoof conformation and any inappropriate farriery should be evaluated and corrected. Short shoes leave insufficient ground surface under the heels and put the weight-bearing surface in front of the vertical axis of the limb. In such instances, a vertical line drawn from the origin of the quarter crack invariably coincides with the end of the shoe (Figure 11.42B). Abnormal foot landing patterns are often contributory as many horses contact the ground asymmetrically, landing first on one side of the hoof and then impacting on the opposite side. This strike pattern is generally related to a rotated limb conformation and can result in the sheared heel foot conformation. In fact, it is extremely rare to see a horse with a full-thickness quarter crack that is not accompanied by a sheared heel. The foot should be trimmed and shod with the affected quarter below the crack unloaded in the same manner as described previously for a sheared heel. Any horse with a full-thickness quarter crack should be placed in a bar shoe until the crack has grown at least halfway down the hoof wall. Various configurations of bar shoes such as a straight bar, egg bar, or heart bar can be used. All of these shoes effectively increase



Figure 11.42. (A) Acute quarter crack with hemorrhage. Note the tightly packed growth rings distal to the coronet as a result of chronic overload. (B) Chronic quarter crack. The origin of crack coincides with the end of shoe that is too small (arrow), and the coronet is displaced proximally.

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the ground surface of the foot on the affected side, provide palmar support, and decrease the independent vertical movement at the bulbs of the heels.

The Repair

If the horse must continue in work, a repair should be performed along with the appropriate farriery. The objective of the repair is to stabilize the crack distal to the coronet and promote healing. If a full-thickness crack is present, just placing a composite over the defect generally does not provide stability; an implant should be used and then covered with a composite patch. Infection at the origin of the quarter crack is characterized by marked lameness, pain when pressure is applied to the defect, and a swollen, discolored coronary band above the defect. Occasionally, exudate can be expressed when digital pressure is applied to the coronet. If infection is present, the crack should be opened and bandaged with a suitable disinfectant agent for at least 48 hours before the repair. If the crack needs to be stabilized at the same time the farriery is performed, the clinician should wait at least 24 hours before repairing the defect to permit the coronet to descend distally to a more suitable position. Repairing the defect with the coronet displaced proximally will compress the horn tubules necessary to produce solid horn above the defect.

A very easy and consistent repair technique will be described.[23,30] Before starting the repair, the hoof wall surrounding the defect should be thoroughly cleaned and dried. The entire length of the defect is explored using a Dremel® tool with a tungsten carbide bit, being careful not to create any unnecessary hemorrhage. All loose undermined horn should be removed, the defect is widened, and the burr is used to create a solid ledge on either side of the trough. The hoof surface on either side of the defect should be sanded with a coarse sanding block.

Two sets of paired 3/64th-inch holes, one-quarter inch apart, are then drilled across from either side of the crack beginning at least 3/8 to 1/2 inch from the margin of the crack and ending within the ledge of the trough. Stainless steel 21-gauge wire is formed into a "hairpin" shape 2.5 inches long with a small steel tab on the closed end of each wire unit. One wire unit is passed through the holes from a palmar to dorsal direction, and another wire unit is passed through the opposing holes in a dorsal to palmar direction into the depth of the crack. The ends of the wires are pulled tight and bent outward. The steel tab placed on the wire unit will now lie against the outer hoof wall. Additional sets of wire units can be used depending on the length of the defect or until the desired stabilization is achieved. Medicated hoof putty or some type of drain is placed in the trough, and the ends of the opposing wires are joined together and twisted until resistance is felt (Figure 11.43). The excess wire in front of the twist is cut off within the defect. The implant is then covered with an acrylic composite combined with fiberglass strands to form a patch.

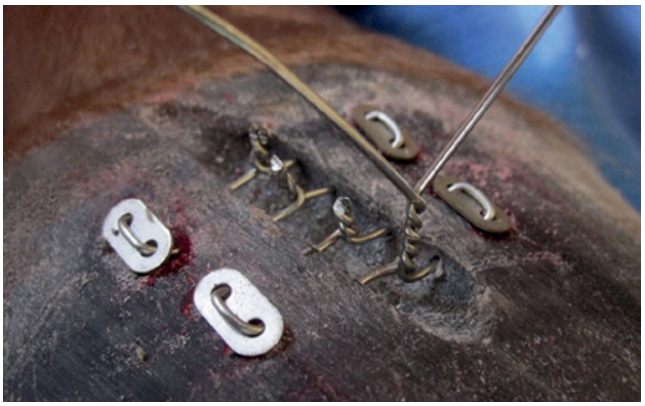


Figure 11.43. Placement of wires for a quarter crack repair.

Toe Cracks

Full-thickness toe cracks occur in the dorsal hoof wall extending from the coronet distally but rarely reach the ground surface. The defect is usually movable, opening when load is removed from the foot and closing when load is applied. The defect is usually associated with excessive length of the dorsal hoof wall with marked concavity, which can be seen with either a long-toe or club foot conformation. Farriery is directed toward correcting or improving hoof conformation if possible, decreasing the length of the toe, and shifting the weight-bearing function palmarly in an attempt to unload the toe.

Unloading the toe can be accomplished by beginning the trim at the apex of the frog and trimming palmarly in a tapered manner until the hoof wall at the heels and the frog are on the same plane. This creates two planes on the ground surface of the foot, so when a shoe is placed on the foot, there will be a space created between the hoof and the shoe at the toe. Farriers refer to this as "floating" the toe. Excess toe length with the concavity is removed from the dorsal surface of the hoof wall. The appropriate size shoe, fitted palmar to the perimeter of the dorsal hoof wall, is then applied. As this condition is often seen with a club foot conformation, if the heels are trimmed to improve the foot conformation, heel elevation may need to be included to compensate for the shortened muscle tendon unit of the DDFT. Moving the break-over palmarly in the toe of the shoe will further decrease stresses in the dorsal hoof wall. The trim and application of a shoe in a manner that unloads the toe are often sufficient to stop the movement of the defect and promote healing. If further stabilization is needed, a metal or brass band can be fabricated and attached to the dorsal hoof wall with screws (Figure 11.44). The band should match the contour of the coronet and be placed at equal distances on either side of the defect. The band should always be attached to the dorsal hoof wall with the foot in the unloaded position with the crack open so the horn papillae at the origin of the defect are not compressed, which allows them to produce solid horn.

Heel Cracks

Heel cracks are generally associated with poor palmar foot conformation secondary to lack of foot mass such as low or underrun heels.[15,31] This type of foot does not have sufficient ground surface under the heels to accept weight. This type of crack is not as common as a toe or quarter crack, but can be difficult to treat. Some type of bar shoe with or without a "spider" plate

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Figure 11.44. Brass band attached to dorsal hoof wall to stabilize a toe crack. Note that the contour of the band matches the coronet. Also note that the defect does not extend distally to the ground surface of the foot.

is recommended to increase the ground surface in the palmar foot, redistribute the weight-bearing on the solar surface, and unload the affected heel. The horn in this section of the foot is generally too thin to accept an implant, and therefore the hoof wall palmar to the defect is either removed or the heel can be rebuilt using an appropriate composite material.

White Line Disease

WLD is a keratolytic process that originates on the solar surface of the hoof and is characterized by a progressive separation of the inner zone of the hoof wall.[15,17,21] The separation occurs in the nonpigmented horn at the junction between the stratum medium and stratum internum. A separation in the hoof wall is considered to be a delaminating process potentially brought on from mechanical stress, inappropriate farriery, environmental conditions affecting the inner hoof wall attachment, and possibly some toxicity such as selenium.[15] The separation, which can originate at the toe, the quarter, and/or the heel, appears to be invaded by opportunistic bacteria/fungi leading to infection that can progress to varying heights and configurations proximally toward the coronet. Interestingly, WLD does not invade the coronet, and the disease process nearly always occurs secondary to hoof wall separation.

Etiology

WLD can affect a horse of any age, sex, or breed. One or multiple hoofs may be involved and the affected hooves can be barefoot or shod. One or multiple horses on the same farm may be affected and the problem occurs worldwide. Multiple causes of WLD have been proposed, but none have been proven. Causes include excessive moisture or dryness that initiate and perpetuate separations or allow pathogens to invade the white line area, poor hygiene, and infectious organisms (bacteria, fungi, or a combination of these organisms). The fact that WLD can be resolved with debridement alone further detracts from infection as a primary cause. Mechanical stresses placed on the inner hoof wall that lead to a separation appear to be the most logical cause, although it does not explain why only certain horses are affected. These mechanical stresses may include excessive toe length and various hoof capsule distortions such as long-toe/underrun heel, club foot, or sheared heels.

Clinical Signs and Diagnosis

WLD presents no threat to the soundness of a horse until damage is sufficient to allow mechanical loss of the attachment between the external lamellae and the inner hoof wall (stratum medium), resulting in displacement of the distal phalanx in a distal direction (rotation and/or sinking). In the early stages of WLD, the only noticeable change on the solar surface of the foot is a small separation containing powdery material located at the inner part of the hoof wall adjacent to the sole–wall junction (white line). This area may remain localized or it may progress proximally to form a larger separation in the hoof wall. As the separation becomes more extensive and extends into a quarter, a concavity (dish) can be seen forming along one side of the hoof, and a bulge is present on the opposite side directly above the affected area at the coronary band (Figure 11.45). The distal phalanx is stabilized or held in place within the hoof capsule by the



Figure 11.45. Extensive separation under lateral hoof wall causing a shift of the distal phalanx in the hoof capsule. Note the concavity in the medial wall, bulging of the lateral wall, and the soft tissue enlargement above the coronet on the lateral side.

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lamellae; therefore, any significant loss of epidermal lamellae on the side with the separation allows the distal phalanx to shift toward the side with the separation. Other clinical signs may be evident including slow hoof wall growth dorsal to the separation, poor consistency of hoof wall, and a hollow sound noted when the outer hoof wall over the separation is tapped with a hammer.[17,21] Unfortunately, the disease often goes undetected until the horse begins to show discomfort.

Radiography can be very informative and should be considered necessary if WLD is advanced and causing lameness. Good-quality radiographs can show the extent of the hoof wall separation and whether rotation of the distal phalanx within the hoof capsule has occurred. Radiography is helpful to differentiate between WLD and laminitis and can also be used as a guide when trimming and shoeing the horse.

Farriery

Correction of any hoof capsule distortion that may have contributed to the hoof wall separation is essential in the management of WLD. Treatment for WLD is directed toward protecting and unloading the damaged section of the foot with therapeutic farriery combined with resection of the hoof capsule overlying the affected area(s). Resection disrupts the continuity and weight-bearing strength of the hoof wall; therefore, some type of shoe or device should be applied to stabilize the hoof wall and transfer the weight-bearing to a non-affected section of the foot. If the separated area of the foot is determined to be extensive, it is important to plan the method of support, the design of the shoe, and method of attachment prior to the outer hoof wall being resected.

The type of shoe used and the method of support depend on the extent of the damaged hoof wall to be removed. As the toe is involved in most cases of WLD, it is helpful to move the break-over in a palmar/plantar direction. The ground surface of the foot is trimmed from the apex of the frog palmarly, thus creating two planes on the bottom of the foot, which effectively unloads the toe. The shoe is fitted, so break-over is just dorsal to the distal phalanx in an attempt to unload the dorsal hoof wall and remove any leverage at the toe. Unloading the hoof wall distal to the resection is essential to remove the stresses from the inner hoof wall and promote good growth. This also minimizes pain by preventing the "pinching" effect at break-over that often occurs at the junction of normal hoof wall and the proximal aspect of the resection.

If the resection is extensive and/or if rotation of the distal phalanx is present, some type of support shoe or a wooden shoe should be used to stabilize the foot.[25] An alternative method is to use a bar shoe or open shoe and pad combined with some type of silastic material to redistribute the weight on the foot (Equilox International, Inc., Pine Island, MN). If necessary, attaching glue-on shoes to the ground surface of the foot is another useful option for shoeing the horse with WLD.[28] However, acrylic repair of the defect following the resection is not recommended and should only be considered after all tracts are resolved.[15,17,21,37] The composite hides and/or fosters the organisms under the repair, and the composite may weaken the surrounding normal hoof wall. Commitment from the owner to institute environmental changes and continue the treatment schedule for WLD as the hoof wall grows down is essential for success.



Figure 11.46. Dorsal hoof wall resection. Note the solid margins around the periphery of the resection.

Complete hoof wall resection (removal of outer hoof wall to expose diseased area) and debridement of all tracts and fissures in the affected area are often necessary. The debridement should be continued proximally and marginally until there is a solid attachment between the hoof wall and external lamellae (Figure 11.46). The veterinarian or farrier should not reach blood during debridement.

Treatment with topical disinfectants and medications following hoof wall resection has been described and remains controversial.[17,21,37] None of the topical preparations being used to treat WLD have any proven efficacy. Various antiseptics and astringents such as merthiolate or 2% iodine may be helpful not only as a drying agent but as a dye marker to outline the remaining tracts that should be removed in subsequent debridement. After thorough hoof wall resection, the affected area is left to grow out with debridement performed at frequent intervals. A wire brush can be an effective method for owners to keep the resected area clean daily. The clinician should explore and debride any remaining tracts at 2-week intervals. When all tracts are resolved and grown out, a continued examination is indicated at routine re-shoeing intervals every 4–5 weeks to prevent recurrence.

Equine Canker

Equine canker is described as an infectious process that results in the development of a chronic hypertrophy of the horn-producing tissues.[16,27,41] It generally originates in the frog and may remain focal but has the capacity to become diffuse and invade the adjacent sole, bars, and hoof wall. Canker can occur in one or multiple feet. The disease was initially described in draft breeds but can commonly affect any breed or sex. The etiology remains elusive, but wet environmental or moist, unhygienic conditions have traditionally been thought to act as a stimulus. However, canker is commonly seen in horses that are well cared for and in horses that receive regular hoof care.

Clinical Signs and Diagnosis

Canker generally originates in the frog and can be mistaken for thrush in the early stages. Thrush is limited

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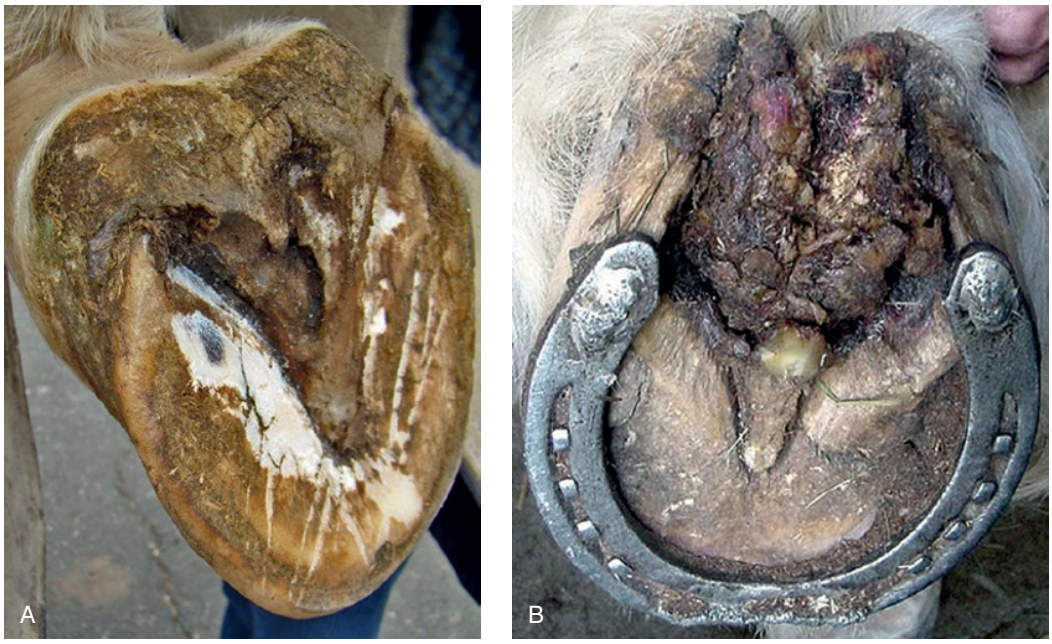


Figure 11.47. (A) Thrush. Note the deterioration of the frog that is recessed below ground surface of foot. (B) Severe canker. Note the proliferation of the diseased horn.

to the body or the base of the frog if a fissure is present, whereas canker invades the horn of the frog anywhere throughout its structure. There is often a proliferation of tissue with canker vs. a loss of tissue with thrush (Figure 11.47). In the early stages, canker may present as a focal area of granulation tissue in the frog that bleeds easily when abraded. Canker is characterized by numerous small fingerlike papillae of soft off-white material that resembles a cauliflower-like appearance.[27,41] The condition is frequently but not always accompanied by a foul odor and is usually covered with a caseous white exudate that resembles cottage cheese.

A presumptive diagnosis of canker is based on the gross appearance of the affected horny tissue along with a fetid odor; however, a definitive diagnosis may be confirmed with a biopsy. Histologically, the lesion is usually interpreted as a chronic hypertrophic moist pododermatitis of the frog.[51] Cultures per se are unrewarding as they typically produce an assortment of environmental organisms such as *Bacteroides* sp. and *Fusobacterium necrophorum*, the usual opportunistic bacteria found in the solar surface of the horse's foot.

Treatment and Prognosis

Historically, horses with canker have had a guarded prognosis. More recently, consistently successful treatments have been described.[27,32,41] Treatment consists of thorough, careful debridement of the affected tissue followed by a regimen of topical therapy applied daily that is continued until the disease has resolved. Recently, the use of corticosteroids during the treatment phase has improved the results. An important aspect that is often overlooked at the onset of treatment is to trim the foot appropriately prior to surgery. All loose exfoliating sole should be removed from the solar surface of the foot. The bars and sole of the hoof capsule adjacent to the frog are explored to be sure the horn in this area is not involved; all underrun or loose horn is removed down to solid structures. The heels are trimmed such that the hoof wall at the heels and the frog are on the same plane or approach the same plane. The frog should not be recessed between the heels of the hoof wall at the beginning of treatment or the affected tissue will not heal.

The debridement of the cankerous tissue can be performed standing under local anesthesia or under general anesthesia if considered necessary. The use of a tourniquet is important to create a bloodless field to better delineate the demarcation between normal and diseased tissue. All abnormal tissue should be removed until the tissue resembles a pink velvet color with numerous pinpoint hemorrhages that corresponds to the dermal papilla. Debridement should be careful, gentle, thorough, and wide rather than aggressive, deep, and radical. Unnecessary removal of the dermal tissue under the lesion should be avoided as this tissue is necessary for regrowth of healthy horn and cornification. Cryotherapy of the debrided area is then recommended to remove residual bacteria from the surface in a double freeze–thaw pattern.

In large defects where the debrided tissue is recessed below the ground surface of the hoof wall, impression material should be used to form an insert over the surgical site in the bottom of the foot. It should extend to the plane of the ground surface of the foot but not above the surface to avoid excessive pressure and discomfort. The impression material ensures contact of the medication against the tissue in the depths of the wound, exerts mild pressure to minimize the production of exuberant granulation tissue, and helps promote physiologic function of the frog.

There are a variety of topical preparations that can be used following debridement, but the astringents (drying agents) appear to play the most important role. Gauze

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sponges moistened in a solution of 10% benzoyl peroxide in acetone and sprinkled with metronidazole powder are used by the author. Other topical products that may be equally effective include a solution of tricide/gentocin/lincomycin or an oxytetracycline/metronidazole paste. No caustic preparations should be used on the debrided site as they will burn or damage the dermal tissue that is necessary to restore healthy horn and cornify.

The use of systemic antibiotics such as chloramphenicol or oxytetracycline has been advocated, but the author questions this practice as the majority of cases resolve with local treatment only. All horses should be treated orally with systemic prednisolone at a dose of 1 mg/kg q24h for 7 days, 0.5 mg/kg q24h for 7 days, and then 0.25 mg/kg q24h for 7 days.[32] The author uses this approach as an adjunct to the local treatment discussed above and has found it to be beneficial.

Aftercare performed by the owner daily or at least every second day is extremely important for success. The foot should be cleaned vigorously using a gauze pad soaked with a suitable antiseptic solution, any suspected area of disease is debrided with a dry gauze pad, the foot is rinsed with saline, topical treatment is reapplied, and the foot is rebandaged. The foot should remain bandaged until the affected area has cornified. A shoe with a treatment plate has become popular with canker cases, but the author feels it is contraindicated as it neither keeps the foot as clean or dry as necessary post-debridement. Once the surface has a complete layer of thin cornification, a treatment plate can be used if desired. A commitment from the owners is necessary for success, because aftercare takes several weeks, depending on the stage of the disease, until the affected tissue becomes cornified.

Thrush

Thrush is a degenerative condition that affects the body and base of the frog generally caused by a bacterial infection. The disease begins when bacteria penetrate the outer horn or epidermis of the frog and is characterized by deterioration of the frog and the presence of black necrotic exudate with a foul odor.[1,3,41] In severe cases, the bacteria can reach the frog dermis, causing pain and lameness. Blood may be encountered when the frog is cleaned with a brush or on the end of hoof picking during routine grooming. Severe cases of thrush must be differentiated from canker, which is a proliferative disease where abnormal frog tissue increases in comparison with thrush, which is a degenerative disease where the horn of the frog deteriorates (Figure 11.47A and B).

Etiology

The overall health of the frog is an important component of the etiology of thrush as it does not occur in a healthy frog. A healthy, well-formed frog is broad and firm and deforms with thumb pressure. The frog should fill the space between the heels of the hoof capsule, and, when measured at its base (the widest part), the frog width should be at least 70% of its length.[49] A large healthy frog acts as an expansion joint that holds the heels apart, shares the load-bearing function with the other structures of the palmar foot, and helps to absorb concussion. Under normal conditions, weight-bearing stimulates the normal physiology of the frog, ensuring its continued health. A well-shaped frog that fills the space between the heels also promotes a natural self-cleaning mechanism such that when the foot is loaded, the frog expands, expelling any accumulated dirt or debris from the frog sulci.

An unhealthy frog is markedly smaller in size and recessed below the level of the ground surface of the hoof, thus creating a void in which debris can accumulate. This can occur for a variety of reasons including many of the recognized hoof capsule distortions such as the low heel, club foot, or sheared heels along with inappropriate farriery practices. Other contributing factors for thrush include wet, unhygienic stable conditions, especially when horses continually stand in urine and manure-soiled bedding, neglecting daily routine foot care and lack of exercise.[1,3,34,41]

A fundamental problem with palmar foot conformation involves the frog being below the level of the heels, which reduces the stimulation from the ground, causing the frog to atrophy further. As the frog recedes within the hoof capsule, the frog losses its "self-cleaning mechanism," which allows material (dirt/manure, etc.) to accumulate over the frog, creating excessive pressure. Over time, this constant pressure on a compromised frog leads to increased deterioration and atrophy. Weakened by the reduced protective horn of the epidermis, the frog tissue becomes susceptible to penetration by bacteria and, consequently, development of the disease. Furthermore, the accumulation of debris over the frog creates an environment conducive to bacterial growth, especially for anaerobic bacteria. The recessed frog often loses its ability to act as an expansion joint and the heels begin to contract. The diseased frog can no longer share in supporting the horse's weight, which shifts most of the load-bearing function solely onto the heels of the hoof capsule.

No specific organism has been identified as the sole cause of thrush. Two anaerobic bacteria species, *Bacteroides* sp. and *F. necrophorum*, are opportunistic organisms found in the soil and are commonly isolated from the bottom of the horse's foot. On the positive side, when the necrotic tissue of the frog is debrided and the conformation of the structures in the palmar foot is improved, the anaerobic conditions will be improved, and bacterial growth will be decreased. Furthermore, if antimicrobials are required, these organisms are quite susceptible to most topical preparations.

Clinical Signs and Diagnosis

The diagnosis of thrush is usually straight forward and based on examination of the frog and surrounding structures. There is usually an increased amount of moisture on the bottom of the foot and a black exudate in the sulci of the frog. This exudate, which varies in quantity, usually has an offensive odor.[3,34] The affected sulci of the frog are often deeper than usual and may extend into the sensitive tissues of the foot causing them to be painful. The frog will generally be small, recessed within the hoof capsule, lack a solid firm consistency, and have a necrotic appearance. There may be a fissure present in the central sulci extending from the base of the frog to the hairline at the bulbs of the heels (Figure 11.48). The frog may also be undermined, and areas of necrotic horn can often

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be detached from the underlying tissue.[49] Lameness is present in severe cases that involve the dermis, and swelling of the distal limb may be seen.



Figure 11.48. An untrimmed foot with thrush, the frog recessed below the ground surface of the foot, sheared heel conformation, and a fissure present in the central sulci of the frog extending to the hairline.

Fissure in Central Sulci of the Frog

There will often be a fissure located in the central sulcus of the frog in horses that have thrush. The author feels the fissure is a primary structural defect in the soft tissue structures in the palmar/plantar section of the foot that secondarily becomes infected leading to thrush. The fissure is often seen in horses with a hoof capsule distortion (such as low heels with instability and sheared heels), a recessed frog, compromised soft tissue structures, and an abnormal strike pattern. The asymmetrical landing pattern results in an unequal force placed on each heel, causing the heels to move in opposite directions. This movement in opposite directions causes a fissure to form in the weakest part of the soft tissue, which is the central sulcus of the frog (Figure 11.49A and B). Significant movement in the fissure can often be demonstrated by moving the heel bulb in opposite directions.

Treatment

Farriery

Treatment for thrush begins with improving the hoof capsule, the conformation of the palmar/foot, and the health of the frog. Thrush will not be resolved as long as the frog remains recessed below the ground surface of the foot. The amount of improvement in the frog is correlated with the amount of chronic damage especially involving the frog dermis. If the horse can be given time off work, the shoes should be removed, the heels trimmed to the same plane as the frog, the perimeter of the hoof wall rounded, and the horse left barefoot. When barefoot, load is shared by all the structures of the palmar foot, and the integrity of structures improves rapidly and it appears to promote regrowth.[24] Treatment of the thrush as described below is performed daily.

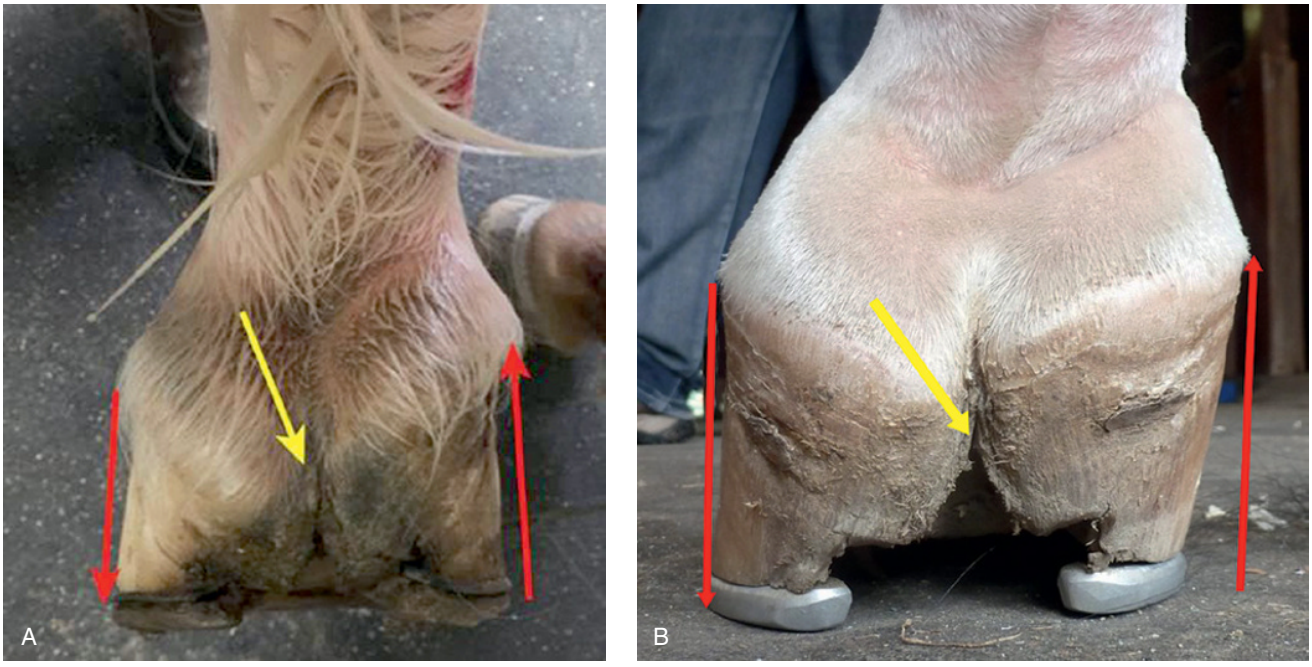


Figure 11.49. (A) Foot conformation showing the frog receded and asymmetric foot landing leading to sheared heels. The difference in force on each heel exerted in the opposite directions (red arrows; A and B) results in the formation of a fissure in the base of the frog (yellow arrows; A and B) (B) A horse with severe club foot combined with a sheared heel. Note the loss of frog mass between the heels.

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If shod, the basic trim begins by drawing a line across the widest part of the foot and trimming the heels of the hoof capsule such that they are on the same plane as the frog. This can generally be accomplished in most cases. The toe length is reduced dorsally to the line drawn across the foot, resulting in approximate proportions on either side of the line.[22,24] If significant heel is removed from an upright or club foot, heel elevation may be necessary to accommodate the resultant stresses placed on the DDFT. When a fissure is present in the central frog sulcus, it should be stabilized to decrease the vertical movement of the heels that will inhibit healing. This can readily be accomplished by applying a straight-bar shoe.

Medical

Any detached areas of necrotic horn on the surface of the frog should be debrided with a sharp hoof knife. The frog is cleaned initially by soaking the foot in a saturated (add salt until it no longer dissolves) solution of Epsom salts ($MgSO_4$). Topical antiseptics/astringents including 2% iodine, merthiolate, chlorohexidine, and various copper sulfate solutions can be applied on a daily basis. Topical antibiotic ointments/solutions appear to be unnecessary, and caustic preparations that contain phenol, formalin, or formaldehyde should be discouraged.

Daily Aftercare

Horses should have their feet cleaned and the frogs brushed vigorously with a stiff brush after which an astringent is applied on a daily basis.[1,3,41] If there is a fissure present in the central sulcus, the area above the fissure should be flooded with the antiseptic/astringent solution to allow penetration into the fissure and then packed with a folded gauze pad. The horse should be kept in a dry, clean stall, bedded on wood shavings or sawdust, and turnout should be in a dry paddock. Exercise should be encouraged to maintain/improve the normal physiology of the foot. Repeated trimming of any detached horn on the frog may be required until the infection is controlled. Routine farriery should be scheduled every 4–5 weeks paying close attention to the trim, the foot conformation, the frog, and the integrity of the soft tissue structures of the foot. Prevention is always superior to treatment; therefore, emphasis should be placed on good farriery as the primary means for preventing this disease.

Fractures of the Distal Phalanx

Fractures of the distal phalanx are not an uncommon injury in equine practice. Although this fracture is quite common in racehorses, it can occur in any breed. Causes include racing on a hard track, kicking an unyielding object, landing on blunt objects while exercising, and penetration of the hoof with a foreign body. The involvement of the DIP joint influences the outcome of the case and future soundness. Fractures that involve the articular surface of the DIP joint usually account for increased lameness, a longer convalescence, and a poorer prognosis for a return to full athletic soundness. On the other hand, nonarticular wing fractures carry a much better prognosis. Distal phalanx fractures are classified into 7 distinct categories based on their configuration and location. More detailed information regarding distal phalanx fractures can be found in Table 4.1.

Affected horses show a variation in lameness from a toe touching lameness to a moderate to severe supporting limb lameness that is acute in onset. Trotting on a hard surface generally accentuates the lameness. Increased digital pulse, sensitivity to hoof testers over the sole, and swelling around the coronary band can be noted. With time, the sensitivity of the hoof testers generally becomes localized to the area distal to the fracture. Clinical signs are similar to those displayed with a subsolar abscess or a severe subsolar bruise. Once the clinical examination has isolated a foot-related problem and no abscess is isolated, high-quality radiographs will often confirm a fracture. Nuclear scintigraphy often reveals the fracture in the acute stage before it becomes apparent on radiography. It is the author's opinion that digital nerve anesthesia should not be used prior to radiographs in acute lameness situations, even if clinical signs are minimal, because full weight-bearing on the foot may cause the fracture line to increase in length, become comminuted, or cause nonarticular fractures to become articular. See Chapter 4 for further information on distal phalanx fractures.

Farriery

External stabilization of the hoof can be used to treat some horses with distal phalanx fractures. The distal phalanx can be stabilized within the hoof capsule using one or two methods: a bar shoe with a continuous rim or contiguous clips or a foot cast. A straight-bar shoe with a continuous rim attached to the perimeter of the shoe, encasing the basal border of the hoof wall, prevents or limits expansion of the hoof wall, decreases the independent movement of the heels, and stabilizes the distal phalanx. The solar surface of the foot is packed with impression material before applying the continuous rim shoe for a number of reasons. First, when any form of restrictive shoe is applied to the outer hoof wall, the foot tends to contract, especially at the heels, over a short period of time. Solar/frog support in any form appears to lessen this process quite markedly. Second, the distal phalanx descends in a distal palmar/plantar direction when weight is placed on the foot with no form of counterpressure below. Also, if the fracture is articular with the larger fragment being displaced ventrally, this added support may increase the stabilization in this direction. The full rim shoe is relatively easy to construct and has advantages over the bar shoe with side clips, which are placed randomly and apply limited focal pressure to the outer hoof wall.

The second method is to apply a foot cast. Foot casts are relatively simple to apply and effectively stabilize the distal border of the hoof capsule. In preparation for casting, the hoof capsule should be clean and trimmed if needed. A piece of casting felt is cut to size to cover the heel bulbs both medially and dorsally, and second piece of felt is applied to the proximal pastern just distal to the fetlock. A roll of 2- or 3-inch fiberglass casting tape is then wound around the perimeter of the ground surface of the foot. The concavity of the sole and the frog sulci should be filled with impression material to distribute the load of weight-bearing across the solar surface of

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Figure 11.50. Fiberglass foot cast extending to the level of the proximal pastern.



Figure 11.51. Lateral radiograph of a horse with thin soles that would be prone to sole bruising.

the foot. One to two additional rolls of 3-inch fiberglass casting tape are used to envelop the foot and extending to the proximal pastern (Figure 11.50). An acrylic composite may be applied to the toe on the ground surface of the cast to prevent the cast from wearing through the ground surface.

Foot casts should be changed approximately every 3 weeks or as needed. The prolonged application of foot casts will cause some contraction of the hoof capsule and may cause pressure sores on the skin immediately proximal to the heel bulbs.

Marginal fractures (type VI) occur at any site along the solar margin of the distal phalanx and are commonly secondary to another condition such as chronic sole bruising, chronic laminitis, or osteomyelitis. In general, these solar margin fractures are not of primary concern themselves, but rather an indication of another underlying condition such as foot with limited protection due to very thin sole thickness. These horses respond to shoeing with a wide-web straight-bar shoe that is well concaved (using a grinder or skilled forging) on the inner solar surface of the shoe.

MISCELLANEOUS CONDITIONS OF THE FOOT

Sole Bruising

Foot bruising and foot infections are two common causes of acute onset of severe foot lameness. Feet that lack sufficient sole depth to afford adequate protection are susceptible to bruising (Figure 11.51).[44] Sole depth for a good foot should be greater than 15 mm, and the sole should not deform when digital pressure or hoof testers are applied.[39] Bruising can occur when the ground surface of the foot strikes a hard object or excessive trauma from working on a hard surface. It appears to cause diffuse hemorrhage or edema formation in the dermal tissue above the horny sole, the increased pressure being responsible for the discomfort. Clinical signs can range from mild to severe lameness, increased digital pulse, and marked response to hoof testers. Hoof tester response generally is diffuse rather than isolated to a small focal area. The diagnosis is made by ruling out the other common differentials, including laminitis, hoof abscesses, puncture wounds, and distal phalanx fractures.[10,11] The old adage of finding signs of bruising such as red discoloration in the sole of a lame horse at the time of examination or during farriery is not accurate, because this discoloration is generally seen in a white foot and may result from previous hemorrhage in the solar corium that has permeated into the horny sole over time.

The bruising resolves with stall confinement on deep sawdust bedding and a 3- to 5-day course of NSAIDs followed by a period of controlled exercise. The most important part of treatment is a farriery plan to improve sole depth. Farriery options include leaving the sole intact during trimming, evaluating the length of the toe when placing the shoe, modifying break-over, and adding some mild heel elevation. Placing a leather or plastic pad between the ground surface of the foot and the shoe protects the thin sole and provides comfort, but generally does not improve the structures of the foot. The use of a rim pad between the foot surface and the shoe is a common farrier practice to raise the foot off the ground. This practice may provide transient improvement; however, it concentrates the weight-bearing on the wall. When the horse's foot loads, the weight of the horse pushes the sole toward the ground, thus inhibiting any further sole growth (Figure 11.52).

Sole pressure often leads to sole bruising, and this type of bruising is usually noted shortly after the horse is trimmed or shod. In this case the hoof wall is trimmed shorter than the adjacent sole so that when a shoe is applied to the foot and clinched or when the horse stands on a hard surface, excess pressure is placed on the sole.

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Treatment for sole pressure is to concave the inner foot surface of the shoe to relieve the pressure on the sole or, in the case of the horse without shoes, apply a shoe with the inner foot surface of the shoe concaved or deep seated to raise the solar surface of the foot off the ground.

The last form of bruising is the so-called corn, which is focal hemorrhage that occurs in the angle of the sole at the heels between the hoof wall and the bar (seat of the corn). The hemorrhage results from trauma, originates from the dermal lamellae, and appears as a small, focal, red discoloration in the seat of the corn. It may be present in one or both heels. There may be a marked hoof tester response, and the source of pain can be confirmed using unilateral local anesthesia. Confusing the diagnosis are abscess, hoof wall separation, and fracture of the bars. Treatment of corns involves removing the pressures on the affected heel by unloading the heel with some form of bar shoe.



Figure 11.52. Foot with a rim pad placed between the solar surface of the foot and the shoe, which raises the sole higher off the ground. The weight of the horse forces the sole through the branches of the shoe toward the ground.

Hoof Abscesses

Hoof abscess, a localized accumulation of purulent exudate located between the germinal and keratinized layers of the epithelium, most commonly subsolar or submural, is probably the most common cause of acute lameness.[42] Entry is either through a break or fissure in the sole–wall junction (white line), a misplaced nail, or a puncture wound somewhere in the solar surface of the foot. Organisms also may enter the foot by way of a full-thickness hoof wall defect or crack.

Most horses with a foot abscess show an acute onset of lameness. The degree of lameness varies from being subtle in the early stages to non-weight-bearing. The digital pulse is usually bounding, and with careful observation, unless the abscess is in the middle of the toe, the intensity of the digital pulse is much stronger on the side of the foot where the abscess is located. If the abscess is long standing, there may be soft tissue swelling in the pastern or above the fetlock on the side of the limb corresponding to the side of the foot where the abscess is located. The site of pain can be localized to a small focal area through the careful use of hoof testers.

The most important aspect of treating a subsolar/submural abscess is establishing drainage. The opening should be sufficient to allow drainage but not so extensive as to create further damage to the hoof capsule. When pain is localized with hoof testers, a small tract or fissure will commonly be found in the sole–wall junction (white line) (Figure 11.53A and B). The wound or point of entry may not always be visible, because some areas of the foot such as the white line are somewhat elastic and wounds in this area tend to close. In this case, a suitable poultice should be applied to the foot daily in an attempt to soften the affected area, and eventually a tract will become obvious.



Figure 11.53. Foreign debris will gain entry and accumulate in a small separation (red arrows) or fissure located in the sole–wall junction (white line) anywhere around the perimeter of the foot (A) including a fissure (circle) on the abaxial surface of the bars adjacent to the sole (B).

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Figure 11.54. Small loop knives, 2 mm bone curettes and other suitable probes used to explore a tract in horses with suspected abscesses (A). A horseshoe nail also makes an excellent tool to establish and promote drainage (B).

The offending tract or fissure located within the sole–wall junction is followed using a thin, small loop knife, hoof or bone curette, or another suitable probe (Figure 11.54). Just a small opening is necessary to obtain proper drainage. Under no circumstances should a subsolar or submural abscess be approached through the sole. The application of a medicated poultice (3M Animal Care Products, St. Paul, MN) that has been soaked in hot water is applied to the foot for the first 24–48 hours. The author prefers the sheet version of this poultice rather than the poultice pad because the whole foot, including the coronet, should be enveloped in the poultice. Another method to encourage drainage is to apply a soak bandage. In this method, layers of practical cotton are crisscrossed to form a heavy bandage to envelope the foot. Magnesium sulfate (Epsom salts) is placed on the inner foot surface of the bandage, and the bandage is attached to the foot. The bandage is saturated with hot water and saturated periodically over the next 24–48 hours. Using either of these methods eliminates the need for continued foot soaking.

The horse should show marked improvement within 12–24 hours. Following the poultice or foot soak bandage, the hoof is kept bandaged with an appropriate antiseptic until all drainage has ceased and the wound is dry. The opening is filled with medicated hoof putty (Keratex Medicated Hoof Putty, Brookeville, MD), and the shoe is replaced when the horse is sound. Often an abscess tract is identified, but drainage cannot be established at the sole–wall junction. A small vertical channel can be created adjacent to the abscess tract in the outer hoof wall using a small pair of half-round nippers. Drainage can usually be established using a small probe in a horizontal plane (Figure 11.55).



Figure 11.55. Small channel created in the hoof wall with small half-round nippers or rongeurs, so infection can be approached in a horizontal direction.

Puncture Wound in the Sole

Another common cause of a perceived subsolar abscess is penetration of any region of the horny sole by a sharp object. This is not actually an abscess but rather a diffuse infection caused by the solar corium being inoculated with organisms from the penetrating object. Pain is immediate and usually followed by infection within 3 days. Using the hoof testers for guidance, the site of the puncture wound is easily identified. Radiographs can be helpful to rule out a fracture of the distal phalanx. Drainage is the treatment of choice, and the wound should be opened carefully in a conical configuration using a small, thin loop knife.[9,40,42,43,45] The conical shape of the wound prevents the wound from closing prematurely and prevents dermal tissue from prolapsing and becoming trapped in the wound. The foot is bandaged with an appropriate antiseptic, and the use of systemic antibiotics is at the discretion of the clinician. When the wound has cornified, a shoe with a pad between the foot and the shoe is applied. See Chapter 4 for further details on puncture wounds of the foot.

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Nail Abscess

Another form of abscess results from a horseshoe nail driven deep to the stratum corneum into dermal tissue. Dermal tissue can be inoculated by bacteria from a misplaced nail in two ways. First, the nail can be driven directly into the laminar corium. When the nail enters dermal tissue, the horse shows discomfort as the nail is driven into the foot and there is hemorrhage present where the nail exits the outer hoof wall. Blood observed at the exit of the offending nail alerts the farrier of the misplaced nail. The blood also acts as a physiologic rinse to dilute or eliminate bacterial contamination. Removal of the nail and application of an appropriate antiseptic usually prevent infection.

The second scenario that occurs frequently is when the farrier is driving a nail; the horse shows pain, indicating the nail is invading sensitive tissue. The farrier then generally removes the nail, places it in another spot or direction, and again drives it into the foot. The original nail (even if removed) may inoculate the dermis with organisms and contribute to an abscess. If the nail has entered the foot inside the sole–wall junction (white line), the owner should be alerted by the farrier to potential problems. To avert an abscess, the horse may be placed on an oral broad-spectrum antibiotic for 3–5 days as a prophylactic measure.

Finally, there is a "close nail," in which the nail is placed such that it lies against the border of the dermal corium just inside the hoof wall. Pressure against the corium and the movement of the hoof wall structures combined with the organisms introduced with the nail often lead to an abscess as described above. There is usually a lag period of 7–14 days or even longer before clinical symptoms or lameness is observed. Treatment again revolves around removing the nail and establishing drainage.

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NATURAL BALANCE TRIMMING AND SHOEING

GENE OVNICEK

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INTRODUCTION

The term "natural balance" has been used for both labeling commercial products and describing an established protocol for trimming and shoeing horses. Natural Balance® shoes have been developed and marketed by Equine Digit Support System, Inc. (EDSS), since the mid-1990s as a treatment and performance product. Additionally, EDSS has developed other complementary treatment and performance products with the Natural Balance name. The Natural Balance products are simply tools for farriers that are consistent and manufactured with specific design features to produce a given result.

Natural balance hoof care protocols, on the other hand, are hoof evaluation and preparation guidelines that farriers can use to treat hoof distortions that can lead to lameness or performance issues. The natural balance guidelines complement most good, conventional hoof care practices. The basic goal of the natural balance trimming and shoeing protocols is to establish equilibrium in and around the distal interphalangeal (DIP) joint by recognizing and eliminating hoof distortions that cause undue leverage and strain on the DIP joint and the hoof itself.

The structures that are used to evaluate distortions, balance the foot, and determine the quality of foot function include the frog, bars, exfoliated sole, and hoof wall. Other considerations that are employed when evaluating distortions and foot function are how the foot engages the ground (e.g. toe first, flat, or heel first), effort or leverage incurred at the time of break-over, and the use or discipline of the horse. For example, horses that are asked to turn sharp corners or circles at high speed may require a different amount or type of leverage reduction built into the shoe than those that only travel in a straight line. Moreover, horses that live and work in a consistent terrain may maintain better foot function and health if they are left barefoot rather than shod.

Many factors influence the decisions that farriers make in terms of how the foot is prepared and what shoeing application is used, if any. Natural balance hoof care protocols are designed to help farriers make those decisions using simple guidelines that are directed by the individual horse and each individual foot.

DISTORTIONS OF THE HOOF

Most of the original information behind natural balance was gleaned from the study and observation of feral horses and how they maintained their feet in their own natural environment.[3] Although there is much that we can take from the self-maintained feet of horses living in the wild, there are environmental and use considerations that must be factored in when applying that information to domestic horses.

In the wild, hoof wall wear depends upon the terrain. For example, in a very hard, dry, and abrasive terrain, the ground does not yield to the hoof wall. Rather, the outer hoof wall yields to the ground and becomes worn all the way around the foot to the level of the sole (Figure 11.56). In soft, sandy terrain, the ground yields to the hoof wall, so hoof wall wear occurs differently and more slowly. In this case, the wall is allowed to grow beyond the level of the sole. The further the wall grows beyond the level of the sole, the more susceptible it becomes to bending and breaking. Without the strength and support of the sole, the wall becomes vulnerable and eventually bends and breaks off systematically until it is again down to the level of the sole. Therefore, self-maintenance does occur in a softer environment; it is just a longer and less kind process.

Horses that live in both a soft and wet environment have an even more difficult time with self-maintenance. The hoof wall is less brittle due to the moisture, so breakage does not occur until the leverage becomes great. In these conditions, bacterial tracts or abscesses may be the vehicles that mother nature uses to initiate the maintenance process. In other words, a curled, badly distorted heel and bar can develop a bacterial tract that works its way to the sole corium and eventually causes a heel abscess. If the heel abscess is severe enough, it can eliminate the entire heel, thereby eliminating the distortion. It is not kind or aesthetically pleasing but effective for carrying out the necessary maintenance.

In addition to terrain factors, horses in the wild do not routinely turn circles repeatedly or at high speeds. They tend to travel at low to moderate speeds and in straight lines for the most part, yet the outer edge of



Figure 11.56. Lateral view of a feral horse's foot that lives in a dry, abrasive environment. Notice the rounded outer hoof wall in the front half of the foot.

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their wall is rolled from one quarter, around the toe to the other quarter (Figure 11.56). Even with these conditions and activities, self-maintained horses have reduced some leverage from a medial and lateral perspective. In the domestic equine world, we not only add the weight of a rider and tack, but we also ask horses to turn in repetitive circles at high speeds. With that, we see many soft tissue strains and injuries on the medial and lateral aspects of the distal joints.

Perhaps it is not possible to use the self-maintained horse as a model for the domestic equine. Maybe the parameters established in feral horse's feet still place too much stress and leverage on the distal joints of the domestic horse. Perhaps we need to relieve even more strain and leverage in domestic horses based on the increased loads and demands that they incur.

Most of the current natural balance guidelines and protocols have been adapted more specifically to the domestic equine through further anecdotal and field research and observations over the last 15 years. The Equine Lameness Prevention Organization (ELPO) is one group that has taken the basics of natural balance guidelines and have further developed and improved upon them over the last 10 years. Practical applications by a large number of farriers in both treating lameness and improving performance over the last decade have helped to fine-tune these methods of balancing feet as well. Through a better understanding of healthy foot function and anatomy, the natural balance and ELPO guidelines have become a more widely used approach to trimming and shoeing domestic horses.

One difference between self-maintained feral horses and our domestic horses is the fact that domestic horses do not typically have the terrain, space, or activity to maintain their own feet and are therefore susceptible to excess hoof growth and hoof distortions. Hoof distortions are generally the result of hoof structures (primarily hoof wall) that grow beyond the level of the sole in a direction that reduces support to the foot or cause increased leverage to the foot. For example, a heel that grows beyond the level of the functional sole and forward becomes unstable and unable to support the weight and force of the horse as the foot engages and loads into the ground. A toe that grows much beyond the level of the sole can become a flare, which further increases the leverage at the time of break-over. A frog that fails to contact the ground and has become narrow and atrophied from lack of use because the heels have been allowed to grow beyond the level of the sole fails to absorb concussion and dissipate energy at the time of ground contact and load.

It is apparent that hoof distortions have a direct influence on function, support, and strain on the internal components of the foot and distal limb.[6,7] When a foot is subjected to increased strain on the soft tissue in and around the distal limb, lameness and pathology are likely to follow. Therefore, it is the job of the veterinarian and farrier to recognize these common hoof distortions and eliminate them at the time of trimming or shoeing to improve or eliminate lameness. Moreover, if these distortions can be dealt with early, or even avoided, then the prevention of lameness in horses can become a more realistic goal.

NATURAL BALANCE HOOF CARE GUIDELINES

A general understanding of the term functional sole plane is needed before discussing the detailed protocol. References have been made previously to the amount of wall that grows or extends beyond the level of the functional or live sole. The functional sole plane is established by scraping or paring the chalky or flaky appearing sole out of the foot until the smooth, waxy-appearing surface is revealed. It is important that when exfoliating the sole, it is approached slowly and methodically so that the functional sole is not invaded or penetrated. If you go too far into the functional sole material, you will not only invade possible sensitive sole, but it also compromises the structures that will be used to balance the hoof wall and distal phalanx to the ground. The live functional sole has been found (with few exceptions) to be an equal thickness beneath the medial and lateral aspect of the distal phalanx.[5] Therefore, if the goal is to balance the distal phalanx medially to laterally parallel to the ground surface, the functional sole is a very reliable guide.

NATURAL BALANCE EVALUATION, EXFOLIATION, AND MAPPING PROTOCOL

Recognizing Hoof Distortions

The first step in the natural balance hoof mapping protocol is to visually recognize hoof distortions in the heels, bars, frog, and toe (Figure 11.57). Heels that are curled, rolled over, crushed, or end dorsal to the widest part of the frog are distorted. Typically, the further dorsal the heels end to the back of the frog, the more distorted and less functional they have become. Similarly, the more curled, laid over, and fractured the bars are, the more distorted and less functional they are. The bars typically terminate into the frog commissures near the widest part of the foot (WPOTF). If the heels are allowed to grow forward, the bars must curve and bend, because the termination point of the bar cannot be moved forward, at least not at the corium level. Like hoof wall and sole material, bars can become overgrown. The overgrowth at the surface can grow forward of the WPOTF and in some cases, such as laminitis, can grow completely around the frog apex. However, actual bar termination maintains its position near the WPOTF.

A frog that has become atrophied and narrowed, and has the central sulcus closed up is often lacking in function and has become distorted. At the same time, if the frog apex has become narrow and pointed, it is likely stretched and distorted as well. Furthermore, if the wall is allowed to grow much beyond the level of the sole, it will start to bend, flare, and become stretched forward. A thickened outer dorsal wall, inner wall, and white zone of the foot indicate that the extra leverage is stretching the components. It may be necessary to look closely at the dorsal wall to see a slight deviation below the coronary band, within the proximal 1 inch of the dorsal hoof wall. As a rule, noticeable flares start about halfway down the hoof wall.

Identifying distortions and evaluating the degree of distortion in all aspects of the foot helps determine the overall health of the foot. Having guidelines to overcome these distortions can help prevent lameness and improve performance. The ELPO has further developed this concept of

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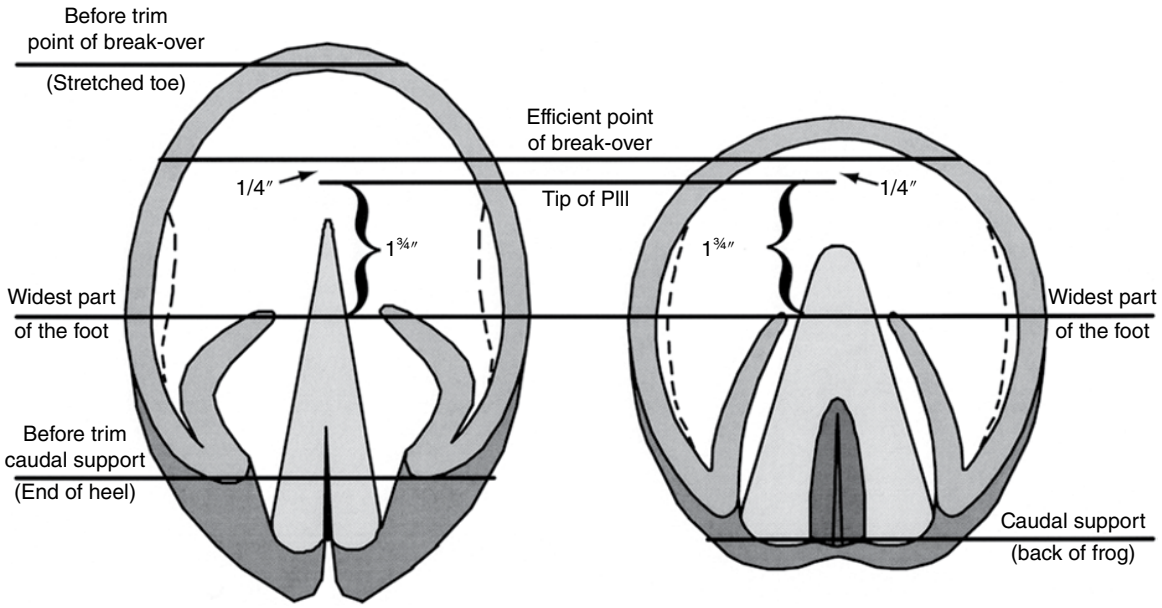


Figure 11.57. Drawings of two solar views. The solar view on the left is a foot with common hoof distortions. The solar view on the right is a non-distorted foot. Lines are drawn on the pictures to illustrate the point of break-over and where it should be on each foot, as well as the widest part of the foot (WPOTF) and caudal most aspect of support (either heels of the foot or back of the frog, depending on which structure contacts the ground first and primarily).

Table 11.3. ELPO hoof evaluation grading protocol.

Grade	Description/abnormalities
0	Considered to be a perfectly natural, normal foot, free of hoof distortions, that is expected to be functioning at its optimum efficiency. Hoof structures with this grade would also be representative of a foot that either requires no maintenance or has just been trimmed and/or shod and again is free of hoof distortions
1	Indicative of a natural normal foot that is at the end of a trimming/shoeing period and requires basic maintenance. Minor hoof distortions seen are the result of normal growth and with basic maintenance will be returned to a grade 0 status. If a #1 status is achieved after trimming/shoeing, then this grade would be representative of hoof structures that possesses only minor hoof distortions that would still allow the foot to function efficiently
2	Indicative of hoof distortions that can start to affect proper foot function. Although these are commonly seen at the end of a shoeing cycle, this rating is suggestive of distortions that generally were not fully dealt with at the beginning of the shoeing/trimming cycle. Feet and structures in this condition can start to negatively affect performance, but may not be recognized as problems by everyone
3	Indicative of hoof distortions that can cause minor to moderate lameness issues. Foot function is often being compromised and common gait faults such as stumbling, forging, and landing toe first are prevalent, and signs of DIP joint pathology may be recognized and even diagnosed by veterinarians. These types of distortions challenge the soft tissues around the DIP joint
4	Indicative of moderate to severe hoof distortions that are often associated with serious lameness issues. These feet have been subject to long-term hoof distortion, and often, irreversible damage can occur. Foot function can be at least somewhat restored with shoes, pads, and detailed hoof trimming
5	Indicative of the most severe hoof distortions that contribute to both soft tissue and bony damage. Horses are often severely lame or debilitated as a result of the distortions. These types of distortions are sometimes irreversible, but can be improved with the use of various prosthetics and more detailed hoof preparation

distortion evaluation by classifying or grading these structures that most commonly distort the foot (Table 11.3 and Figure 11.58).[1] The goal of the ELPO Hoof Evaluation Protocol is to accurately and consistently, in accordance with established standards, be able to determine the amount and/or location of hoof distortions in individual equine feet. Although an overall rating for each foot may be achieved, individual attention to primary hoof structures is the key. Through the systematic evaluation of the external hoof anatomy, a more accurate and meaningful evaluation of the foot can be achieved, as well as a determination of the overall health and soundness of the horse.[1]

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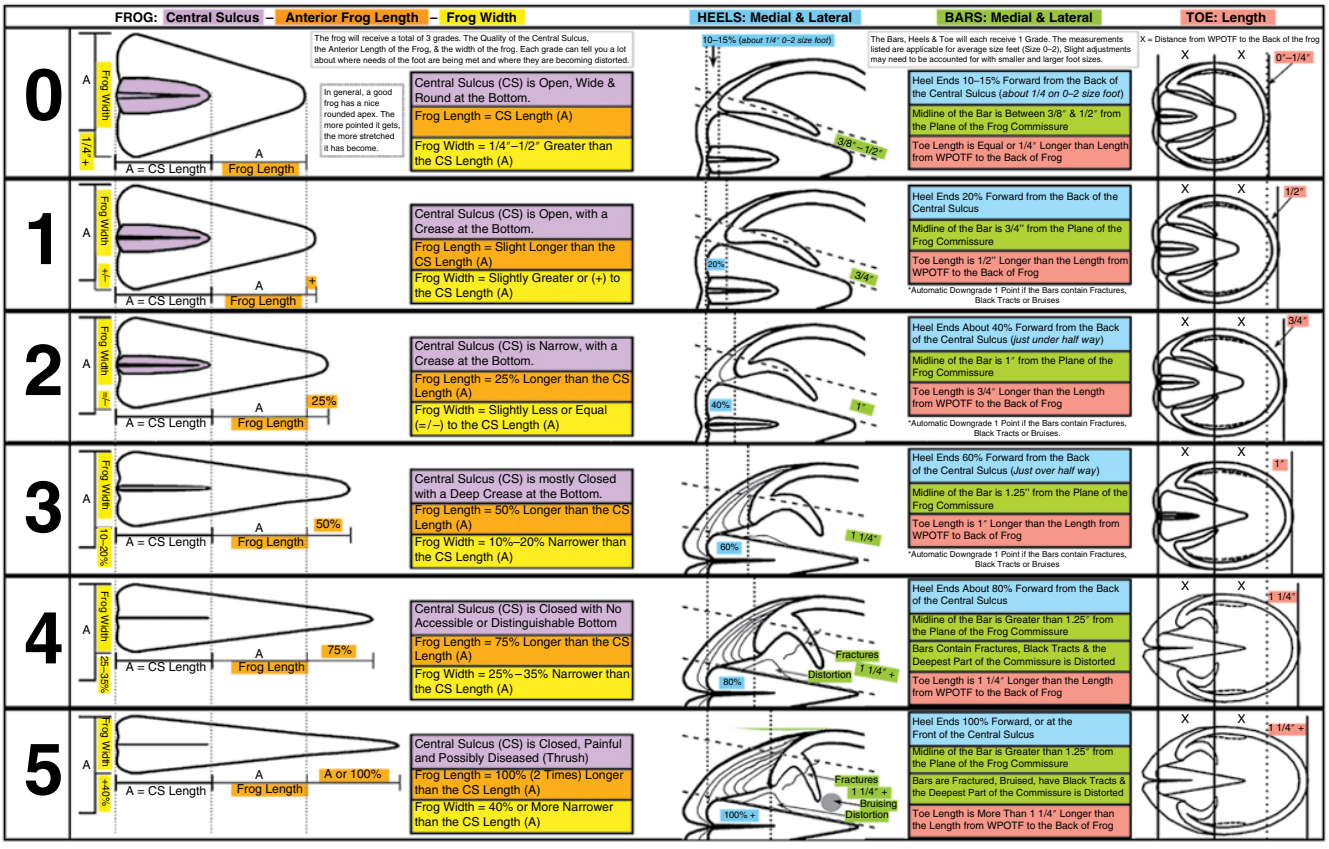


Figure 11.58. Detailed description of the grading protocol that is used for the frog, bars and heel, and the toe of the equine foot by the ELPO. (Disclosure: The information presented in this evaluation protocol are only general guidelines designed for equine professionals as a means to have a standardized formula for recognizing common hoof distortions that exist in many feet with basically "normal" conformations. This evaluation protocol is not intended to diagnose any lameness, nor is it intended as instructional guidelines for trimming or shoeing. For details on hoof care guidelines, look at the "ELPO Hoof Trimming & Shoeing Protocols" offered by the Equine Lameness Prevention Organization, Inc. [www.LamenessPrevention.org].)

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The primary hoof structures that are specifically evaluated are the frog, bars, heels, and toe and are evaluated from the viewpoint of the sole. A basic overview of the grading parameters is contained in Table 11.3, and a detailed description of the grading protocol for each structure is available through the ELPO and illustrated in Figure 11.58.

Exfoliating the Foot

When leaving horses barefoot, the amount of exfoliating sole material to remove varies from one environment to another. To assess how much sole exists, first exfoliate and uncover the true frog apex where it blends in with the sole. This provides an idea as to how much nonfunctional sole exists. As a general rule, very little exfoliating in the toe region is tolerated by horses that live in hot, dry climates. Slightly more exfoliation can be done in soft, sod environments where horses get moderate amounts of moisture. In very wet or swampy environments, most feet need very little exfoliation, and most of the time simply wire brushing the bottom of the foot handles most of the exfoliation needs.

Usually when horses are left barefoot, it is a good idea to be conservative when exfoliating the sole. However, it is fairly safe to exfoliate the sole from the WPOTF back, so it is important to find the waxy-appearing functional sole in this region on each side of the foot. This will later be the guide for establishing medial/lateral (M/L) balance.

When applying shoes to horses, it is important to be more specific about removing the majority of exfoliating sole materials to get a clear picture of the functional sole plane. Nevertheless, starting in the back half of the foot is always recommended because it is a relatively safe zone in which to work. Once a clear picture of the functional sole is established in the back half of the foot, move forward and exfoliate the sole in the toe region. Be extremely specific and careful when exfoliating the sole in the toe quarter (pillar) region of the foot. This is the primary guide when trimming the wall to balance the distal phalanx medially and laterally.

Mapping the Foot

The goal of mapping the foot is to gain an appreciation for the location of the distal phalanx and DIP joint within the hoof capsule. As part of their assessment to balance the foot, farriers have always tried to locate or picture the position of the distal phalanx within the hoof capsule but have largely had varying results when using the hoof capsule itself as a guide. The hoof wall can become distorted and more easily lose its relationship to the distal phalanx. However, more static references on the bottom of the foot help establish where the distal phalanx and DIP joint are relative to the hoof capsule.

Once the foot is exfoliated, a primary goal is to locate the WPOTF; there are three methods for locating this position:

Measure back approximately 1 inch from the true, exfoliated frog apex (on a medium size foot #00 to #2), and make a mark (Figure 11.59A).

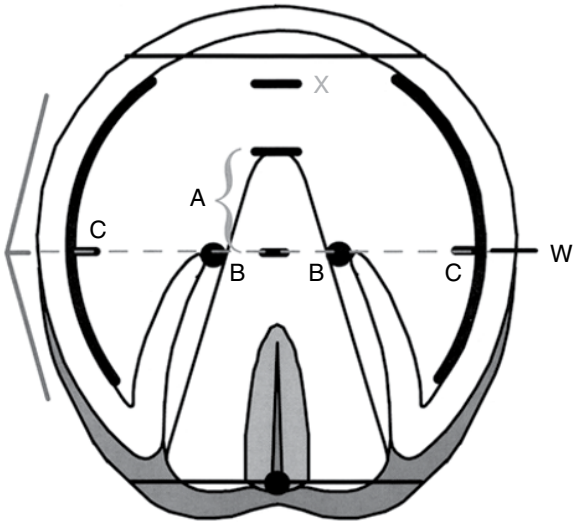


Figure 11.59. Drawings of the solar view of a foot that has the lines and marks used to locate (map) the widest part of the foot, which is generally the center of articulation. (A) Measurement back from the true frog apex. (B) The termination point of the bars into the frog commissures. (C) The actual widest part of the sole. (W) The widest part of the foot using the best 2 out of 3 marks. (X) The tip of the distal phalanx as determined by a given distance forward of the WPOTF.

Follow the bars to their termination point into the frog commissures. There is usually a slight bump or swelling in the commissures to indicate this location. Make marks at these termination points (Figure 11.59B).

Scribe a line at the sole–wall junction in the quarters of the foot on each side. The peak or apex of this arc that has been drawn should be the widest part of the sole. Make a mark at the apex on each side of the foot (Figure 11.59C).

If all three locations line up, then draw a line connecting all of the marks that have been made (Figure 11.59W). If only two of the three locations line up, use those two and disregard the third set of marks.

Radiograph studies have shown that from the marked WPOTF, the dorsal tip of the distal phalanx is a consistent and predictable distance ahead of this mark.[2] On foot size #1 (5-inch-wide foot), measure 1.75 inches ahead of the WPOTF and make a mark. This is approximately the tip of the distal phalanx (Figure 11.59X). For foot sizes smaller and larger, the distance measured forward varies by no more than 1/8 inch/foot size. For example, a #00 foot is approximately 1.5 inches, while a number #2 foot is approximately 1.875 inches. These distances are accurate to within ±1/8 inch.

As a standard starting point for the location of the point of break-over, measure forward 1/4 inch from the tip of the distal phalanx mark (Figure 11.57). In general, this position for the point of break-over (not the front of the shoe) seems to be effective for relieving excess strain and leverage on the DIP joint and excess tension on the deep digital flexor tendon at the time of break-over.[4]

Once the WPOTF and the point of break-over is established, the before-trimming and after-trimming ratios can be evaluated to compare how the foot has been and will be balanced around the WPOTF

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(Figure 11.57). The WPOTF is generally below the center of articulation (±1/4 inch), depending on the angle of the distal phalanx.[5] If the distance from the WPOTF to the "before trim" heels is shorter than the distance from the WPOTF to the "before trim" point of break-over, then there are most likely hoof capsule distortions that can negatively affect the performance and soundness of the horse (Figure 11.57). Changing the ground surface ratios around the WPOTF or center of the foot so that there is equal or slightly more mass behind the WPOTF has been shown to improve the function of the foot and soundness of the horse.

Again, the ELPO has taken the lead in developing the mapping protocol further to help improve accuracy and consistency.

NATURAL BALANCE BAREFOOT TRIMMING

The following natural balance hoof trimming procedure is used only on horses that will be left barefoot. The hoof preparation protocols for horses that are to be shod are slightly different and will be covered later.

Once the foot is exfoliated and mapped, it is time to trim the hoof wall to eliminate any distortions and achieve D/P and M/L balance. Begin by trimming the back two-thirds of the foot. Start just behind the toe quarter (pillar) region and follow the contour of the exfoliated sole, staying about 1/8 to 1/4 inch above the line of the sole (Figure 11.60). Next, trim the wall around the toe. Again, start behind the pillar and cut from one toe quarter around the toe to the other toe quarter. Stay at least 1/8 inch above the level of the sole all the way around the toe.

Once the rough cut has been made with nippers, a rasp is used to file the heels down to a flat landing that joins with a portion of the bar to form a solid base. The finished heel should be at or slightly lower than the ground surface of the frog and should end close to the back of the frog. Both the medial and lateral heel should have a similar curvature and end at the same position relative to the back of the frog and at the same height above the level of the functional sole plane. If the frog is atrophied, do not rasp the heel down so low that the functional sole plane in the heel region is invaded. With more use, the frog will eventually fill out and become a larger presence in the back of the foot.

With the heel prepared, a rasp is used to flatten the wall in the toe quarters so there is approximately 1/16 inch of gap between the sole and the ground surface of the wall. Make sure the gap is the same on both the medial and lateral sides. Once the toe is balanced with respect to the sole, bevel (or roll) the wall at a 10°–15° angle from the break-over line forward (Figure 11.60). It is OK to touch some of the sole ahead of this point, because it is not sensitive. The objective is to produce a slight pivot point at the point of break-over that was marked earlier.

After the break-over roll is produced, a flattened area of wall about 3/4 inch to 1 inch in length should be left in the toe quarter region on each side of the foot. Behind that flattened area, relieve the wall in the quarters slightly to avoid flaring and chipping (Figure 11.60).

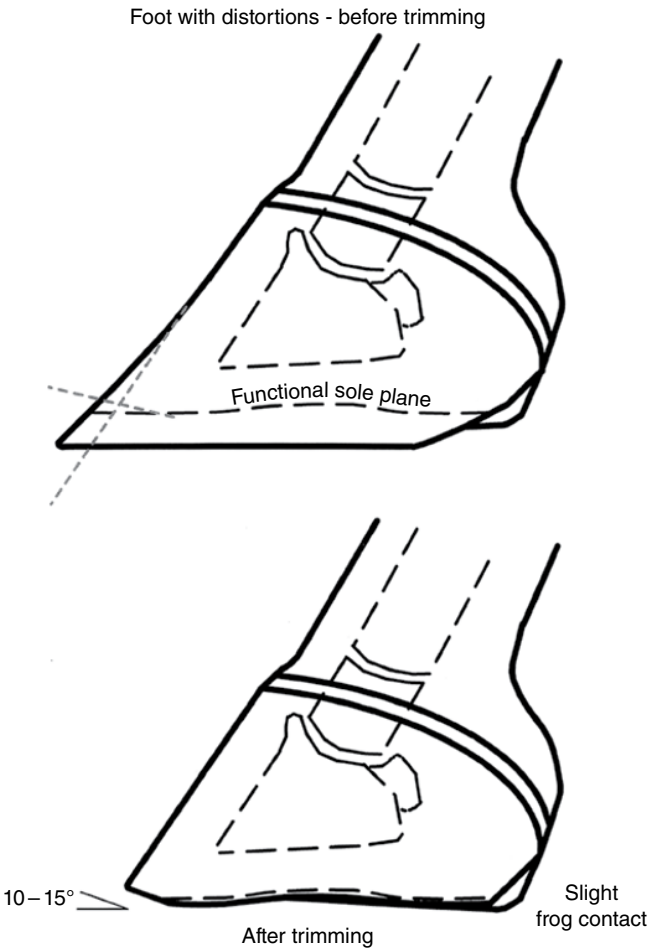


Figure 11.60. Drawings of two lateral views. The top illustration is a foot with common distortions before trimming. The dashed line near the bottom of that foot is the functional or live sole contour used as a trimming guideline. The lighter, shorter dashed lines indicate how the flare is removed and how the toe is to be rolled (beveled). The bottom foot is an illustration of a natural balance barefoot trimmed foot. The wall has been prepared close to the level of the sole, the frog is in slight contact with the ground, and the toe has been rolled at a 10°–15° angle to reduce leverage on the internal structures.

The final step in the barefoot trim is to dress the outer hoof wall to eliminate flares and produce a uniform wall thickness all the way around the foot (Figure 11.60). To eliminate sharp edges and avoid chipping, angle the rasp at a 45° angle to the wall and chamfer the distal border of the wall all the way around the foot.

NATURAL BALANCE SHOEING

The following procedure is used for preparing feet that will have shoes applied to them.

Once the foot has been properly exfoliated and mapped as outlined previously, it is ready to be trimmed (Figure 11.61). Begin with nippers and trim the wall in the back half of the foot first. Unlike barefoot trimming, the exact contour of the sole is not followed through the quarters. Instead, start with the nipper blades just behind the toe quarters and trim toward the heel, leaving a gap of

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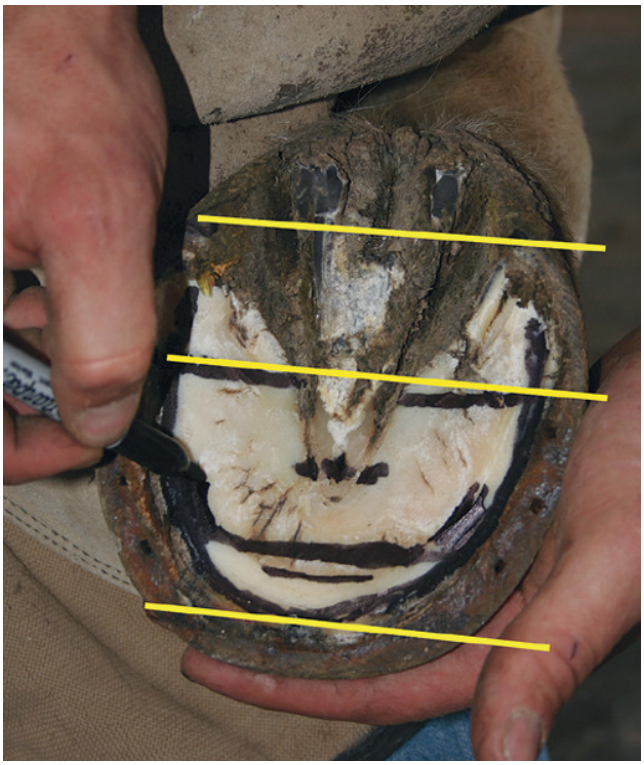


Figure 11.61. Sole view of a foot that has been exfoliated and mapped and is ready to be trimmed for shoes. Yellow lines have been drawn at the end of the heels, widest part of the foot, and at the "before trim" point of break-over.

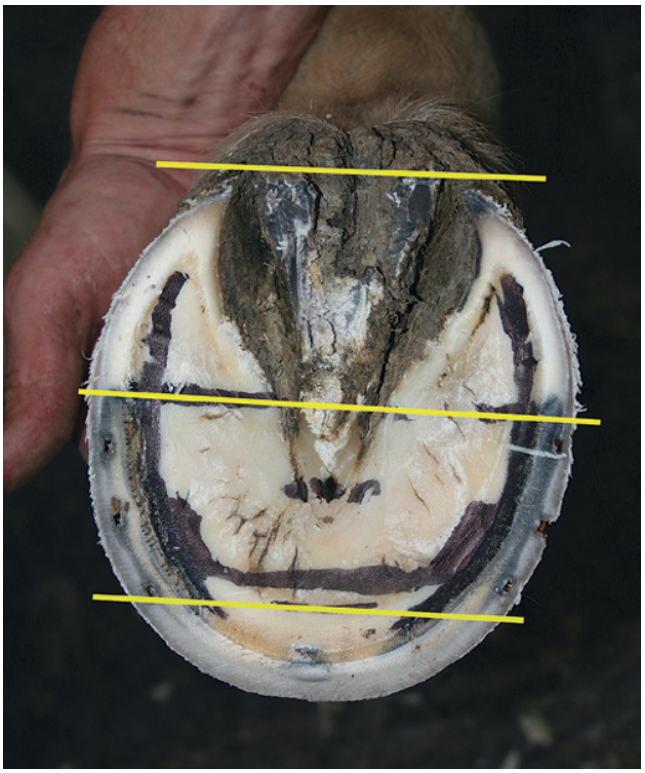


Figure 11.62. Solar view of a foot that has been exfoliated, mapped, and trimmed and is ready for a shoe to be applied. Lines have been drawn at the end of the heels, widest part of the foot, and the position that the point of break-over of the shoe will be placed.

approximately 1/4 inch above the sole in the quarter region. Do the same on each side of the foot.

With the initial heel trim finished, start in the toe quarter region just behind the previous cut and trim the wall from toe quarter to toe quarter. Make sure to leave at least 1/8 inch of wall above the level of the sole all the way around.

Once the rough-in trim is complete, use a rasp to file the heels so they are about 1/8 inch above the level of the functional sole in that region. Both the medial and lateral heel should have a similar curvature and have the same gap from the sole to the wall. The heels should also end evenly near the back of the frog. In cases of severely underrun and contracted heels, it may be necessary to rasp the heels down to the level of the sole to produce a good supportive landing and encourage better heel conformation. However, this should not need to be done every time the horse is shod. Once the heel growth becomes stronger and at a better angle, this more aggressive heel trimming should be stopped.

Once the heels are prepared exactly where desired, it is time to move to the toe. File the wall in the toe region so that there is a gap of approximately 1/16 inch from the exfoliated sole to the ground surface of the wall in the toe quarter region on each side. Although some horses are OK if the wall is rasped level with the sole, for safety measures it is recommended to leave a bit of wall above the sole. Either way, the gap should be exactly the same on both the medial and lateral sides, as this determines the balance of the distal phalanx to the ground once the shoe is applied.

When the heels and toe have been accurately prepared, proceed to rasping the wall in the quarters so that the finished surface is perfectly flat to accept the flat shoe (Figure 11.62).

The final step before applying the shoe is to dress or groom all flares on the outer hoof wall to obtain a uniform wall thickness all the way around the foot (Figures 11.62 and 11.63). By dressing the wall prior to fitting the shoe, the chances of fitting the shoe to hoof capsule distortions are minimized.

Shoe fitting and design is an important part of dealing with lameness issues and improving performance. Basic shoe design for Natural Balance® applications should fit the following criteria:

1. Fairly symmetrical foot surface mass around the widest part of the shoe—the length of foot surface of the shoe should be the same ahead of and behind the widest part of the shoe.
2. Some leverage reduction considerations should be built into the toe of the shoe (i.e. rolled toe). In most lameness and performance situations, the roll should continue around the toe quarters for medial and lateral leverage considerations (Figure 11.64).
3. Wear plates in the toe of the shoe should be avoided so that the point of break-over is allowed to wear back into the shoe as the foot grows longer.

Shoe fitting is extremely important, so good forging skills are always necessary. When sizing the shoe, the heels of the shoe must extend to the back of the frog, and the point of break-over of the shoe should align

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Figure 11.63. Lateral view of a foot in which the dorsal hoof wall has been groomed to a flat plane from the coronary band to the ground surface. All noticeable flares have been removed and uniform wall thickness has been achieved (as seen in Figure 11.62).

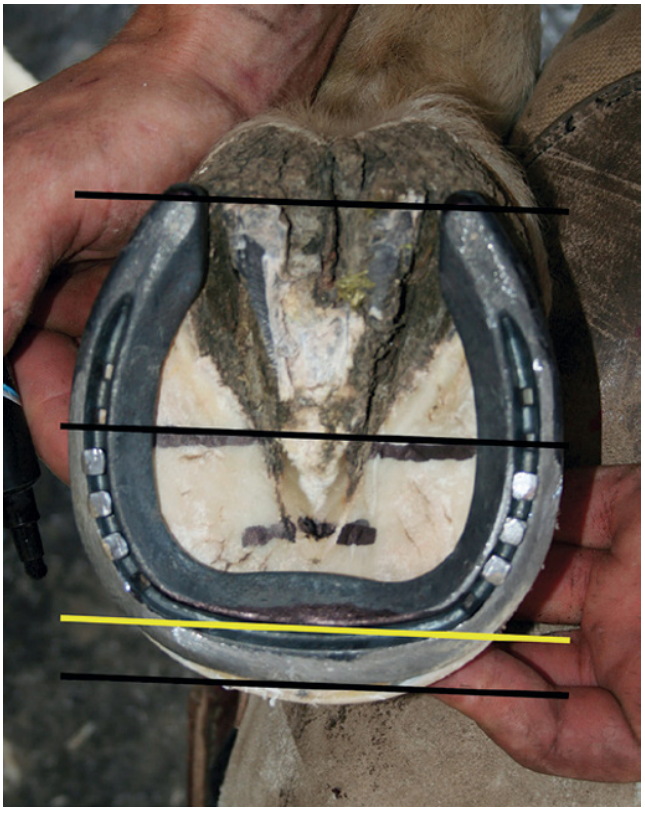


Figure 11.64. Solar view of a foot that has a Natural Balance® shoe applied to it. Black lines have been drawn at the back of the heels, the widest part of the foot, and the front of the shoe to illustrate that the foot surface of the shoe is divided up 50/50 around the widest part of the foot. The yellow line indicates the point of break-over of the shoe.

closely with the break-over line marked on the foot. The foot surface of the shoe should be divided equally around the WPOTF (Figures 11.64 and 11.65). The toe quarters of the shoe should fit to the groomed toe quarters of the foot, and the widest part of the shoe must align with the WPOTF.

When shaping the heels, they should have a gradual bend that follows the contour of the hoof, with the shoe becoming slightly wider than the heels as they progress from the widest part back. The finished heel of the shoe should be around 1/8 inch wider than the heel at the buttress.

The general shape of the shoe should be round on the foot surface with a slight boldness to the toe. However, the toe quarters of the shoe should maintain a width that is basically the same as the heels, meaning the toe quarters and heels should form a box (Figure 11.64).

Nailing on shoes and finishing the foot is basically the same as in any good conventional shoeing job. A quality nailing pattern with three or possibly four nails/side is generally required. Once the nails are clinched, grooming the wall is minimal, because the flares have been dressed prior to fitting the shoe. If there is any amount of toe that sticks out over the front of the shoe, simply position the rasp at a 30° angle and undercut the toe slightly (Figure 11.65).

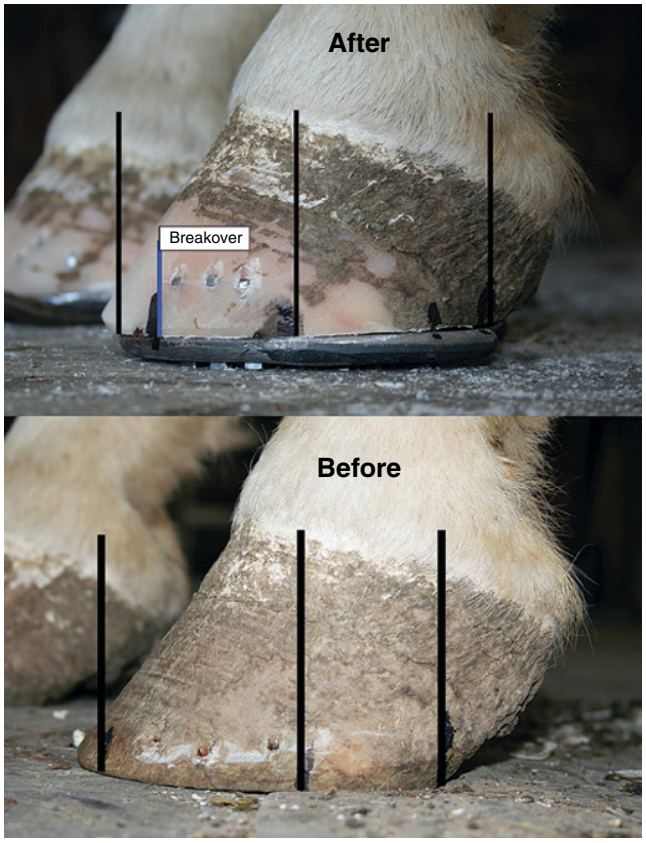


Figure 11.65. Lateral view of the same foot. Before trimming (bottom) and after trimming and shoeing (top) using natural balance guidelines. The black lines on the top image have been drawn at the front of the shoe, widest part of the foot, and back of the heels. The blue line is at the point of break-over of the shoe. The black lines on the bottom picture are at the "before trim" point of break-over, widest part of the foot, and the "before trim" back of the heels. The top picture illustrates better balance after trimming and shoeing around the widest part of the foot, which is generally the center of articulation of the DIP joint.

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The angle of the undercut should be similar to the angle of the roll in the shoe. The amount of toe that extends ahead of the shoe depends on the amount of hoof capsule distortion that exists. Normally within three or four shoeing periods, the amount of toe over the front of the shoe will be minimal, as the wall regains a better parallel relationship with the dorsal surface of the distal phalanx.

SUMMARY

Natural balance is a term used within the hoof care industry with both positive and negative connotations. Many of the negatives or failures associated with Natural Balance shoes have in large part been the result of inappropriate or misapplied hoof preparation techniques and/or incorrect shoe placement. The fact is that poorly prepared feet or an inability of a hoof care provider to read and understand the foot, regardless of the shoe applied, is a key factor in the ability to keep horses sound. Understanding the basic structures of the foot and how to locate them inside the hoof capsule and how the foot grows, and developing the ability to see and evaluate when the foot is distorted or overgrown will improve our success at preparing feet to maintain soundness. As the ELPO has continued to improve and develop the natural balance guidelines for mapping, barefoot trimming, and shoeing, the ability to not only get horses sound but keep them sound and prevent future lameness has greatly improved.

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12

CHAPTER

Miscellaneous Musculoskeletal Conditions

MUSCULOSKELETAL EMERGENCIES

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INTRODUCTION

Musculoskeletal (MSK) emergencies such as fractures, tendon/ligament lacerations, and septic synovial structures can occur in any type of horse and may be career ending or potentially life threatening if not treated appropriately. Forty percent of horse owners responding to a survey in the United Kingdom reported that their horse had suffered an injury within the previous 12 months and 54% of those injuries were wounds.[39] It is important to recognize significant MSK injuries early to provide the horse with the best opportunity for recovery. Every veterinarian should be prepared to stabilize a fracture or unstable limb and to quickly identify a laceration that involves a tendon, ligament, or synovial structure. Being prepared for these emergencies can help ease the stress of the situation and can lead to successful assessment and management of the injury. Prompt attention and treatment can often mean the difference between survival and euthanasia of a patient.

Assessment of the Patient

Regardless of the type of MSK emergency, the first and most important step should always be to stabilize the overall systemic condition of the horse. Many of these patients are in significant pain, often anxious, and potentially in shock. Depending on the type of injury, the patient may have also suffered extreme blood loss. Patient assessment and stabilization should include a thorough physical exam and potentially a packed cell volume (PCV) and total protein (TP) measurements. Based on these preliminary findings, pain management with nonsteroidal anti-inflammatory drugs (NSAIDs) and/or opioids should be utilized. Sedation may be used to calm the patient. Phenothiazine tranquilizers, such as acepromazine, should be avoided in horses with hypotension because they may cause an exacerbation of the hypotension.[34] Tetanus prophylaxis is also indicated in cases where wounds are present if the horse is not up to date or has an unknown vaccination status. Once the patient is calm and systemically stable, further evaluation of the injury can be pursued.

FRACTURE MANAGEMENT

Fractures frequently require emergency first aid treatment (Table 12.1). Significant blood loss is rare in equine fractures but can occur with some femoral fractures or pelvic fractures[52] and occasionally proximal tibial fractures.[36] Antimicrobial therapy is typically not necessary for initial equine fracture management. It should be instituted however if the skin has been penetrated by the fracture or if there is an open wound associated with the fracture. Whenever possible, gross debris should be removed from the wound/puncture site prior to placing in a bandage.

Clinical Signs

The most common clinical sign associated with a fracture is an acute non-weight-bearing lameness. Frequently this is associated with a traumatic event that occurred during competition. Swelling often follows the injury, and palpable or audible crepitus is frequently

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Table 12.1. Recommended contents of fracture first aid kit for horses.

Material	Purpose	Examples
Rigid material	To stabilize the fracture	PVC splints (varying lengths), aluminum rods, fiberglass cast material, wooden boards
Cotton	Padding for under the splint or cast Robert-Jones bandage	Roll cotton Sheet cotton Combine roll
Brown gauze Vetrap	Compress the bandage/padding	
Sterile gauze	Cover any open wounds	Kerlix AMD
Razor and/or portable clippers	To remove the hair from around any open wounds	
Elastic tape	Secure the top and bottom of the bandage/padding	Elasticon
Nonelastic tape	Secure the splint to the bandage/padding	Athletic tape Duct tape
Pre-made splints	Stabilize lower limb fractures	Kimzey splint
Sedation	Ease anxiety Decrease activity	Xylazine, detomidine Butorphanol Acepromazine
Pain management	Reduce inflammation, pain, swelling	Phenylbutazone Flunixin meglumine
Antibiotics	To initiate if there is a wound or if the fracture has punctured the skin	Parenteral and oral options Dictated by case and veterinarian
Antibiotic ointment	Topical dressing for open wounds	Silver sulfadiazine Triple antibiotic ointment

PVC = polyvinyl chloride.

present. Unfortunately, not all cases are this straightforward especially when the incident that led to the fracture may not have been witnessed or minimal swelling is apparent such as with fractures of the axial skeleton.

Diagnosis

Most fractures in horses can be easily localized and diagnosed with radiographs. If the patient is quiet and comfortable, a full radiographic study should be taken before limb immobilization as the splint may decrease image quality. If the patient is in shock or if the fracture is completely unstable, limb immobilization should be performed prior to image acquisition. A complete radiographic study will usually provide important information on prognosis that allows for better preparedness by the referral center. An initial prognosis can often be developed based on two simple criteria: complete or incomplete fracture and open or closed fracture.[36]

Radiographs may not be diagnostic for fractures of the axial skeleton or in the case of nondisplaced or incomplete fractures where the fractures are too subtle to be seen radiographically. Nondisplaced or incomplete fractures will present with acute onset of lameness, but identification of the fracture lines can be very challenging. If the injury is the result of trauma (i.e. kick from another horse), skin trauma will indicate the area to radiograph. Frequently, multiple radiographs will be needed to identify the fracture line, and even more commonly, the fracture lines may not appear for 14–21 days (Figure 12.1).

Identification of fractures of the axial skeleton can be even more challenging. Nuclear scintigraphy can be useful but is obviously not available in the field nor at all veterinary hospitals. Ultrasound can be very helpful to identify steps in bone contour and is frequently more accessible. Geburek et al.[15] found 73% agreement between ultrasonography and standing radiography for pelvic–femoral disorders.

Fracture Stabilization

Next to patient assessment, fracture stabilization is the most important step in the emergency first aid treatment of fractures. Horses are not readily ambulatory on 3 limbs and often become very anxious when they are unable to place weight on a fractured limb, and any attempt to weight-bear can result in further injury. Appropriate fracture stabilization can mean the difference between euthanasia and successful repair and

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recovery. First aid measures should be directed toward minimizing further damage to the fractured limb and maintaining it in a position and condition that will facilitate repair. The primary goals of fracture management should be as follows[4]:

1. Prevent damage to neural and vascular elements
2. Keep the fractured bone from penetrating the skin and becoming and open fracture or protecting the limb from contamination through any existing skin opening
3. Stabilize the limb to relieve anxiety that accompanies an uncontrolled limb in a horse
4. Minimize any further damage to the fractured bone ends and surrounding soft tissues

An important purpose of fracture stabilization is to prevent the development of an open fracture. Loss of intact skin coverage over a fracture is thought to predispose to infection, especially if internal fixation is performed. Equine skin is thin and readily penetrated by sharp bone fragments, and there is very little soft tissue support, such as muscle, below the carpus and tarsus. In general, fractures of the distal (phalanges) and proximal (humerus, ulna, and femur) aspects of the horse's limbs rarely become open, suggesting that fractures involving the metacarpus/metatarsus, radius, and tibia are the most critical to proper immobilization during transport. Immobilization or splinting techniques depend upon the fracture location and the forces applied to the fracture site.[52] A good rule of thumb is that the joint above and below the fracture site should be immobilized. In no circumstance should the splint end at the same level as the fracture because the splint will act as a fulcrum and further disrupt the fracture.[52]

For immobilization purposes, the forelimb and hindlimb of the horse can be divided into four functional segments based on anatomy and biomechanical forces (Figure 12.2).[4,52]

Phalanges and Distal Metacarpus

The phalanges and distal metacarpus are probably the most common sites for fractures to occur in horses, and biomechanically they are dominated by the angle of the fetlock joint[4] with minimal medial/lateral forces. Therefore, proper splinting techniques should attempt to counteract the bending force at the fetlock. This is achieved by creating dorsal cortical alignment (Figure 12.3). A compression bandage combined with a dorsally placed splint or a fiberglass cast applied with the limb maintained in a straight line from the carpus to the hoof provides the optimal dorsal cortical alignment (Figure 12.3). Large diameter polyvinyl chloride (PVC) pipe cut into thirds along its length is a common material used for splints. The curve of the PVC pipe cradles the bandage and the material is lightweight. Minimal padding (light bandage) should be used beneath the splint because fracture fragments can move within a large bulky bandage, and the bulk can sometimes make it difficult for the horse to ambulate efficiently. The splint is applied to the dorsal aspect of the limb from the carpus to the hoof and secured with nonelastic tape. A bandage cast is another alternative—fiberglass casting material placed circumferentially over a lightly padded bandage striving to maintain dorsal cortical alignment. To facilitate application of the splint or bandage cast, an assistant should hold the forelimb proximal to the carpus and let the limb hang (Figure 12.4). Commercially available splints such as the Kimzey Leg Saver (Kimzey Welding Works, Woodland, CA; Figure 12.5) are also available and can be utilized for distal limb fractures.

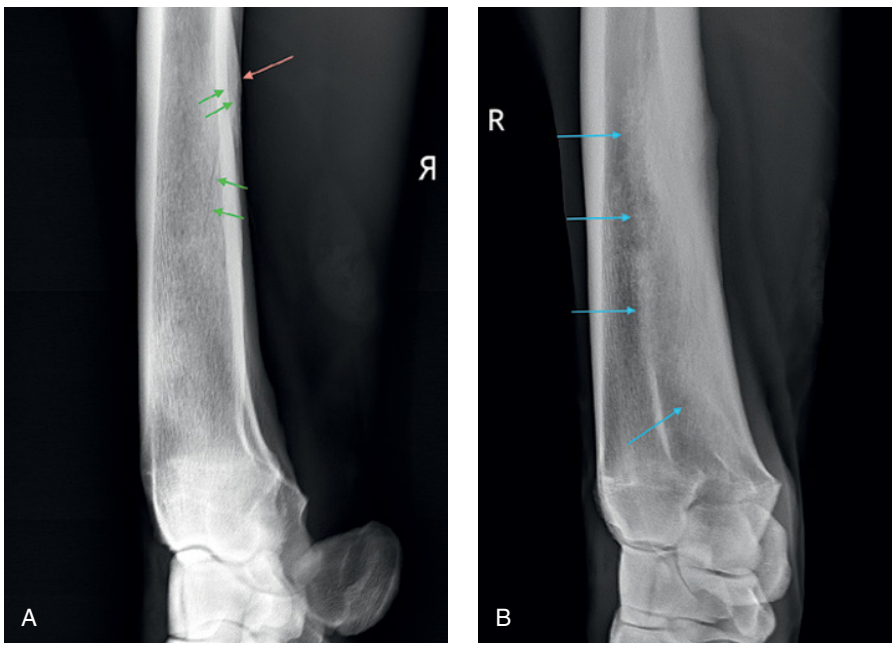


Figure 12.1. Incomplete, nondisplaced fracture of the radius. Fracture lines were not identified until nearly 4 weeks after the initial injury (hoof strike from another horse; red and green arrows in A). Most notable change was the amount of intramedullary sclerosis (blue arrows in B).

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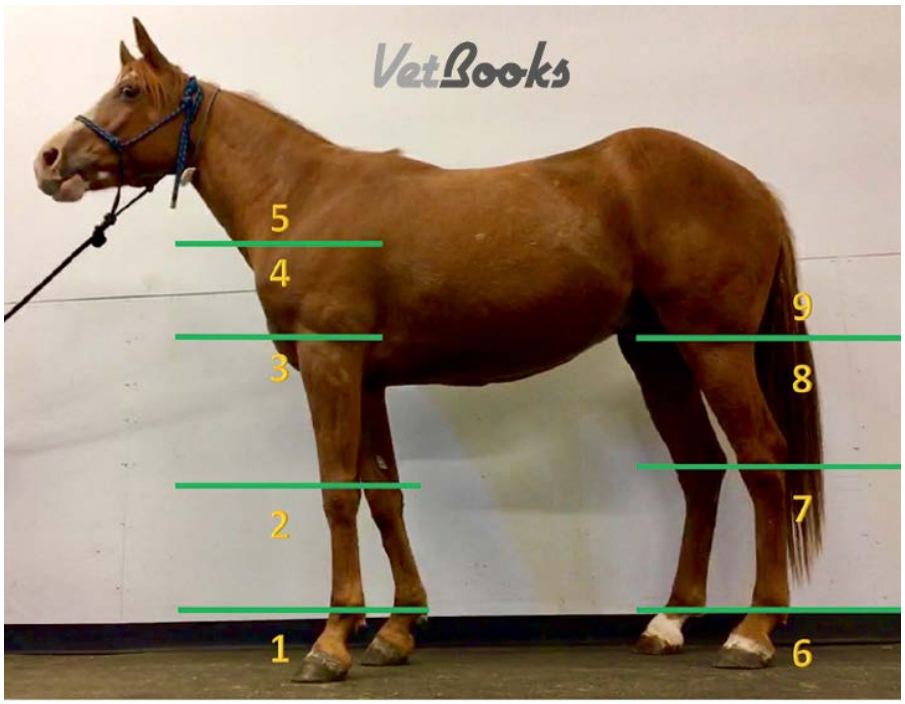


Figure 12.2. Functional divisions of the horse's limbs that can be used as a guide for appropriate application of external support to stabilize fractures for transport. They can be divided into five regions in the forelimb and four regions in the hindlimb. Forelimb: (1) dorsal splint, (2) compression bandage with caudal and lateral splint, (3) compression bandage with extended lateral splint, (4) caudal splint to the level of the elbow to lock the carpus in extension, (5) no immobilization necessary, (6) plantar splint, (7) compression bandage with plantar and lateral splint, (8) compression bandage with extended lateral splint, (9) no immobilization necessary.

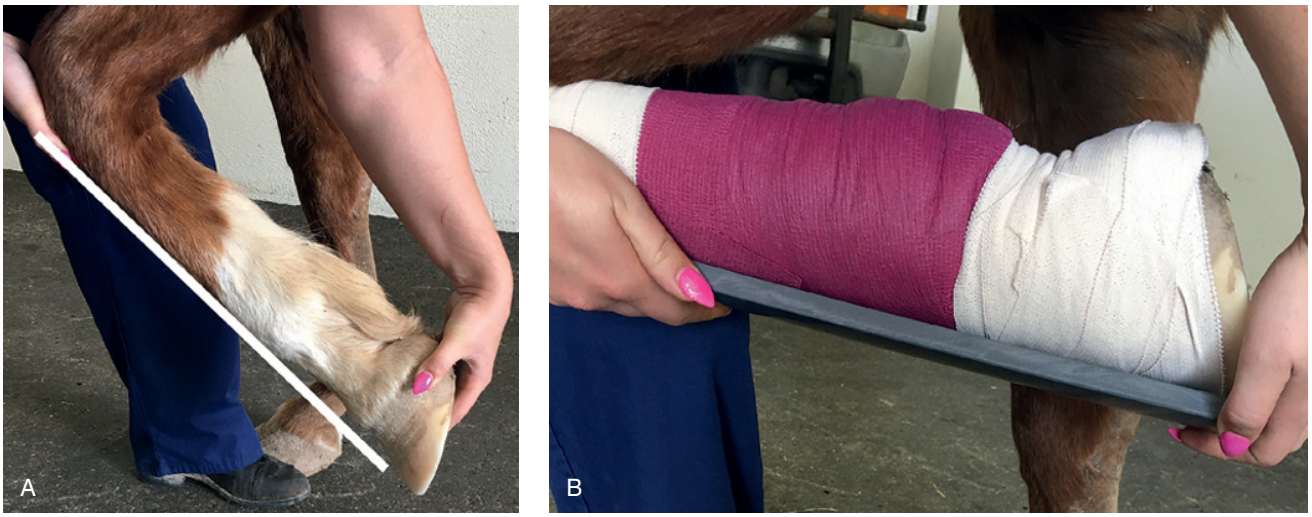


Figure 12.3. Dorsal cortical alignment is represented by the white line on the left image (A). This can be achieved with PCV placed on the dorsal aspect of the limb over a compression bandage (B). This stabilization is ideal for phalangeal fractures. The splint should be held in place with nonelastic tape.

Mid-forelimb (Mid-metacarpus to Distal Radius)

Fractures of this region include subluxation or luxation of the carpus as well as fractures of the proximal metacarpus and distal radius. Goals of immobilization are to establish a straight bony column, stabilize the joints above and below the fracture, and stabilize the limb in multiple planes. This requires two splints, one placed caudally and one placed laterally (Figure 12.6).[3,4] The underlying compression bandage should be applied in several layers, with each layer of padding (cotton) no more than 1 inch in thickness and compressed with elastic or brown gauze to increase its stiffness. Each layer is applied more tightly than the previous one.[14] The bandage should contain only enough padding to help reduce swelling in the limb and protect the limb from the splint material. These fractures have a high risk of displacing further during transportation if the bandage becomes loose due to excessive padding. The bandage and splints should extend from the ground to the elbow. The splints should be at 90° to each other to help prevent movement of the fracture during transport and should be tightly secured to the bandage using nonelastic white tape. If PVC material is not available, any lightweight, rigid material such as wood, aluminum, or flat steel may be used effectively for splinting. Fractures in this location have the advantage of having portions of the forelimb above and below the

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Figure 12.4. If a splint is not available, cast material may be placed over a compression bandage to create dorsal cortical alignment and immobilization of a distal limb fracture. An assistant can hold the limb at the back of the carpus and with the toe in extension in a non-weight-bearing position for application.



Figure 12.5. The Kimzey Leg Saver splint is a commercially available device used to stabilize phalangeal fractures or breakdown injuries in the distal limb of racehorses.

fracture site, which can facilitate fracture stabilization. However, because of the sparse soft tissue coverage of the metacarpal region, closed fractures in this location can easily become open if not adequately splinted. This is especially true for foals because their thin skin provides little resistance to bone penetration.

Middle and Proximal Radius

Fractures in the middle and proximal radius result in abduction of the limb distal to the fracture by the upper musculature of the forearm.[3,4] Abduction of the limb distal to the fracture can lead to the proximal fracture fragment penetrating the skin on the medial side of the antebrachium because of the lack of soft tissue coverage. Prevention of abduction of the distal limb is the goal when immobilizing fractures in this location. This is best achieved by applying a Robert-Jones bandage similar to that used with mid-forelimb fractures, but the lateral splint is extended up the lateral aspect of the shoulder, scapula, and chest and taped securely to the proximal forelimb at the level of the axilla. A wide board (15–20 cm) or metal rod appears to work better than PVC for this lateral splint because it can lay flat against the muscles of the upper limb, but PVC can also be used (Figure 12.7). The upper extension of the lateral splint should lie as tight against the shoulder and scapula as possible so that it prevents abduction of the distal limb during ambulation. A PVC splint that extends from the ground to the elbow should also be placed on the caudal aspect of the limb to provide additional stability of the fracture.

Proximal to the Elbow

The humerus, ulna, and scapula are well protected with muscles, which inherently stabilize and protect fractures of these bones. However, complete fractures of these bones disable the triceps muscle apparatus, making it impossible for the horse to fix the elbow in extension for weight-bearing. Restoring the triceps apparatus reduces the anxiety of affected animals and enables them to use the limb for balance during transport. A full-limb cotton bandage with a caudally applied PVC splint keeps the carpus extended and helps restore triceps muscle function (Figure 12.8). Some horses with ulna fractures bear considerable weight on the limb after splinting, but walking may be difficult. Not all fractures in these locations require stabilization. Foals may not have the upper forelimb strength to move the limb with a splint in place, and therefore a splint may be contraindicated. Caution should be taken when dealing with ulnar fractures to not end the caudal splint at the level of fracture as it can result in pressure over the sharp fracture fragments and the development of an open fracture.

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Figure 12.6. Placement for a caudal and lateral splint for a mid-forelimb fractures. Both splints should extend from the ground to the proximal radius/ulna. They can then be held in place by nonelastic tape or casting material.

Phalanges and Distal Metatarsus

Fractures in the distal hindlimb can be managed with a single splint, similarly to those in the forelimb except that the splint is best placed on the plantar surface of the limb. A splint/cast combination may be beneficial to provide more support, but a splint held in position with nonelastic tape should be sufficient for transport (Figure 12.9). Dorsally applied splints on the hindlimb appear to be less useful than in the forelimb. Due to the reciprocal apparatus, the distal limb is in flexion when non-weight-bearing, making it difficult to achieve true dorsal cortical alignment, and dorsally placed splints tend to break more readily. Also due to the reciprocal apparatus, application of a bandage cast in the hindlimb can be difficult because of inability to keep the limb steady and in proper alignment while the cast material hardens (Figure 12.10). The Kimzey Leg Saver splint may be used to stabilize fractures in this location, similar to the forelimb.

Middle and Proximal Metatarsus

A Robert-Jones bandage with PVC splints applied laterally and caudally using the calcaneus as a caudal extension of the metatarsus provides adequate support for fractures in this location.[3,4] The Robert-Jones bandage should be less extensive than in the forelimb because it is difficult to secure the splints to the limb if the bandage on the distal aspect of the metatarsus is too bulky. Robert-Jones bandages consist of multiple layers of padding each compressed with brown gauze or elastic tape, and each layer should be applied more tightly than the previous one.[14]

Tarsus and Tibia

Fractures in the tarsus and tibia are particularly difficult to adequately stabilize because of the reciprocal apparatus and its effect on joint motion in the tarsus and stifle.[3,4] Fractures in this location tend to collapse when the stifle flexes, and the tarsus remains in a fixed position. The main principle for stabilization is similar to that of the radius, which primarily involves preventing abduction of the distal aspect of the limb (Figure 12.11). There is very little muscle coverage of the medial aspect of the tibia, and this is the site that is prone to becoming open. To prevent abduction, a single laterally placed splint that is bent to follow the angulation of the limb and extends proximally above the stifle joint works well. The splint is most effective if it is bent back on itself to mirror the hindlimb contour back to the ground, creating a double splint. The splint is applied over a full-limb Robert-Jones bandage and is best made of lightweight metal such as aluminum (Figure 12.12) that can be bent into the proper position, similar to the lateral support of a Schroeder-Thomas splint. An alternative to the metal splint is a wide board (15–20 cm) that extends from the ground to the ilium and is applied to the lateral aspect of the bandage.

Transportation

Appropriate immobilization is very important in the management of fractures, but modes of transportation should be evaluated and discussed prior to splint application. Horses can still be ambulatory with distal limb splints, but often the splints required for more proximal fractures can make ambulation difficult and loading into a trailer even more difficult. Ideally, horses should be loaded into a trailer with a ramp to minimize the effort. When possible, horses should be traveled facing forward for hindlimb fractures and facing backward for forelimb fractures. This concept should be ignored if turning the horse around in the trailer will cause more manipulation of the fracture than to simply allow them to travel facing forward. The use of dividers is also recommended as the horse can brace against them.

MUSCULOSKELETAL WOUND MANAGEMENT

Horses are prone to deep lacerations of their distal limbs due to their flight response, kicking defense, and high speeds. Horses may jump sharp objects or fences or often pull excessively if their lower limb is trapped, inciting significant injury. These lacerations frequently involve tendons and ligaments as well as synovial structures. Early recognition of synovial involvement and/or tendon/ligament injury is paramount to the management of these cases.

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Figure 12.7. Stabilization for middle and proximal radius fractures. The tall lateral splint prevents abduction of the distal limb. A bandage and caudal splint are applied (A), and then a lateral splint extending proximal to the shoulder is applied (B). The tall lateral splint prevents abduction of the distal limb. A flat board or metal splint is best, but PVC can be used. *Source*: Courtesy of Dr. Jeremy Hubert.

Preventing Infection in Traumatic Wounds

The ideal treatment for infection is to prevent or limit infection in the first place. Lameness resulting from a traumatic injury is common, and the sequelae of infection can have a devastating consequence. The initial management of such injuries often determines the outcome. Limb wounds require a thorough examination to determine the structures involved and the degree of injury. Prior to invasive palpation, wounds should be clipped and cleaned in an aseptic manner. A sterile lubricant may be applied to the wound to prevent hair from getting into the wound while being clipped.

Povidone iodine or chlorhexidine-based scrubs remove most debris from the area, minimizing further contamination.[53] Copious lavage with a similar povidone iodine (0.1%) or chlorhexidine (0.05%) solution under moderate pressure (10–15 psi) removes bacteria but does not drive them into the damaged tissue; this is accomplished with a 35- or 60-mL syringe and an 18-gauge needle.[60]

Debridement can be done with a scalpel, removing superficial tissue until all notably contaminated or denuded tissue has been removed. Exposed bone usually needs superficial debridement as well (Figure 12.13). Debridement has been shown to be important in the management of traumatic wounds. Tissue that is contaminated beyond 10^5 microorganisms/g of tissue is likely to become infected.[18,44] Contamination with soil can result in a 25-fold greater infection rate,[42] and specific soil factors can result in as few as 100 microorganisms/g causing infection.[43] Once debrided, the wound can be explored with a sterile instrument such as a probe or with sterile gloved hands (Figure 12.14). Thorough exploration and a solid knowledge of anatomy will help determine the important structures involved.

TENDON AND LIGAMENT LACERATIONS

Clinical Signs

Complete severance of tendons of the distal limb may result in characteristic gait deficits. If the extensor tendons are severed, the lower foot may move more freely,

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Figure 12.8. Caudal splint used to lock the carpus in extension and restore the triceps apparatus. *Source:* Courtesy of Dr. Valerie Moorman.



Figure 12.10. Example of an improperly applied bandage cast on the hindlimb to stabilize a horse with a condylar fracture.



Figure 12.9. Use of a board (A) as a plantar splint for stabilization of a distal limb fracture in the hindlimb (B).

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Figure 12.11. Insufficient lateral stabilization of this distal tarsal luxation (A, B) resulted in further displacement during transportation (A).



Figure 12.12. An aluminum rod has been shaped to extend from the ground up over the stifle and hip in this horse with a tibial fracture. Similar to radial fractures in the forelimb, the splint prevents abduction of the limb distal to the fracture, further stabilizing fractures of the tibia. A wide board may be used instead of the aluminum rod. *Source:* Courtesy of Dr. Chris Ray.



Figure 12.13. The dorsal metatarsus is a common location for extensor tendon lacerations, and many injuries also involve the metatarsus. This wound will need debridement of the contaminated tissue and sharp debridement of the dorsal cannon bone. *Source:* Courtesy of Dr. Gary Baxter.

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Figure 12.14. This relatively minor wound on the medial aspect of the tarsus was associated with fractures of the sustentaculum tali, and a probe went directly to the bone and entered the tarsal sheath. *Source*: Courtesy of Dr. Gary Baxter.

and the horse may intermittently knuckle forward at the fetlock due to inability to extend the digit during limb placement. Lacerations or rupture of the flexor tendons results in visible changes to limb conformation during weight-bearing (Figure 12.15). Loss of just the superficial digital flexor tendon (SDFT) usually causes the fetlock to drop with normal foot placement. Loss of the deep digital flexor tendon (DDFT) will result in elevation of the toe during weight-bearing (Figure 12.16). Complete severance of both the SDFT and DDFT will likely cause a hyperextension of the fetlock and the toe coming off the ground during limb placement. If the suspensory ligament is also severed, the fetlock drops almost to the ground. Lacerations of collateral ligaments and sesamoidean ligaments do not always result in a characteristic gait but can be detected during manipulation of the limb while non-weight-bearing. Horses are often not lame while weight-bearing with extensor tendon injuries.[31] More lameness is present with lacerated flexor tendons.[9] The degree of lameness for lacerated flexor tendons, and ligaments can be highly variable and can be dependent upon the extent of other injuries.

Diagnosis

Digital palpation of the wound can help reveal the extent of the laceration to the tendons and ligaments in many cases (Figure 12.17). Wounds should be prepared aseptically prior to digital exploration, and sterile gloves

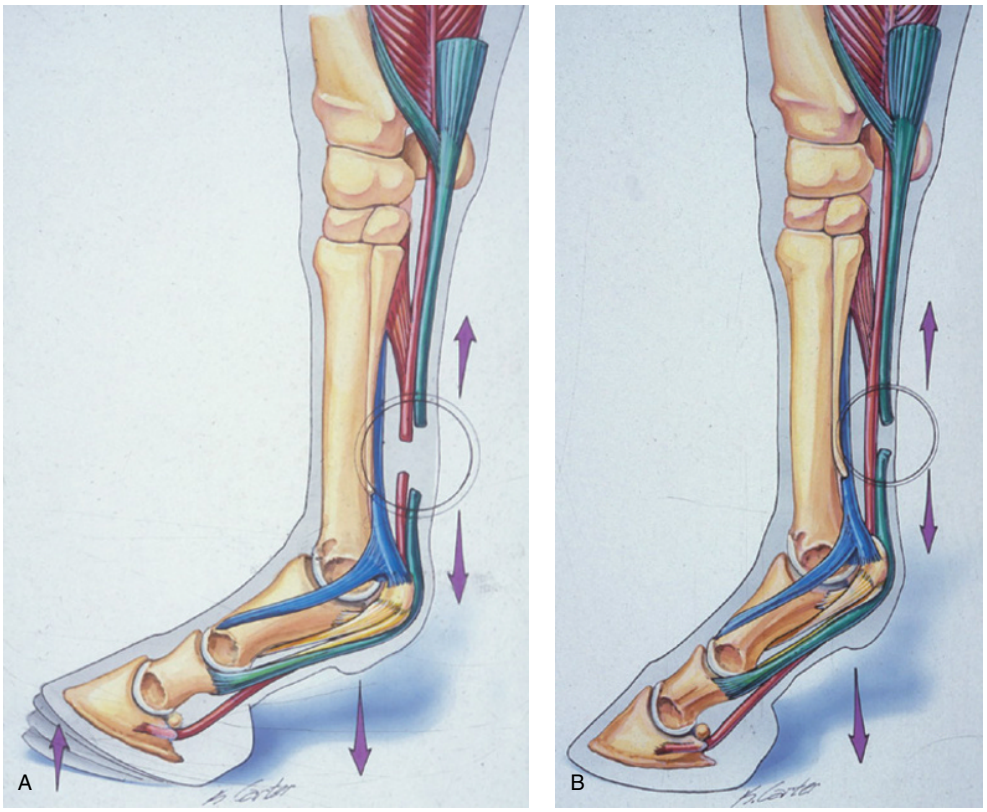


Figure 12.15. Illustration of the biomechanical effects of lacerations of both the SDFT and DDFT (A) and just the SDFT (B) in horses with flexor tendon lacerations. Arrows indicate distractive forces on the tendon ends, dropping of the fetlock, and elevation of the toe. *Source*: Courtesy of Dr. Gary Baxter.

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should be worn. Care should be taken to not push foreign material deeper into the wound and to not further separate soft tissues, because vital structures such as an artery, tendon sheath, or joint could be damaged or entered. Extensor tendon injuries are often avulsion-type trauma with loss of skin over the dorsal aspect of the cannon bone. Secondary bone trauma with the development of surface sequestra of the cannon bone often accompanies these severe injuries (Figure 12.18).

Ligament lacerations are commonly accompanied by tendon lacerations. Rarely is a suspensory ligament or sesamoidean ligament lacerated without the overlying tendon also being affected. Lacerations of collateral ligaments can occur alone although they are often accompanied with a lacerated synovial structure. Manipulation of the joint in all directions may be necessary to determine if ligaments are damaged, and imaging (ultrasound or a stressed radiographic view of the joint) may be required to make the diagnosis. Discrete palpation determines whether bone has been exposed or if neighboring tendons are also lacerated. Ultrasound examination can help define partial ligament tears and possibly locate foreign material in the wound.

Treatment

Routine first aid management of all wounds is recommended initially. Immobilization of limbs with lacerated DDFT and SDFT to support the fetlock and prevent further injury should be performed similar as with fractures of the distal limb. This also applies to ruptured tendons and/or ligaments.

For complete extensor tendon lacerations, and if indicated by duration of injury and other criteria, the optimal approach is to reappose the tendon ends, close the wound, and provide support to the fetlock, such as a caudal splint to prevent knuckling, for at least the first 4–6 weeks of healing. This approach will result in the most rapid healing and return to function and best cosmetic outcome. However, many lacerations involving the extensor tendon cannot be closed due to the severity of the trauma, and reapposition of the extensor tendon ends is not required for successful outcome. Conservative management of the wound and extensor tendon laceration with second intention healing can result in complete return to athletic use. Weight-bearing can be permitted for extensor tendon lacerations, so splints, soft casts, or resin-reinforced bandages can suffice to provide fetlock support. The primary goal of immobilization is to prevent dorsal knuckling of the fetlock. Removal of sequestra from the dorsal cannon bone may need to be performed to permit healing in severe injuries.

In general, flexor tendon lacerations are a much more serious injury with greater implications regarding soundness than lacerations of the extensor tendons and are therefore more challenging to treat. For flexor tendon lacerations, the optimal approach is wound debridement, tendon apposition with suture, wound closure, and cast application for 6 weeks.[12] Double locking loop, 6-strand Savage, or the three-loop pulley suture patterns have been described for repair of the flexor tendons. Recent literature has found that the 6-strand Savage has improved strength and resistance when compared with the 3-loop pulley.[11] Occasionally the tendon ends cannot be reapposed for a variety of reasons. Jordana et al. found that tenorrhaphy did not influence the survival of cases or their return to use.[21] Wound debridement and skin closure can still be performed because the tendons will heal with gap healing, provided they are adequately immobilized with a half-limb cast. For wounds that cannot be closed, use of boots and immobilizing splints (Kimzey splint) can achieve acceptable results, but management of these wounds is much more difficult than if casts are used.[9] If the digital sheath is concurrently involved with the laceration, tenoscopic exploratory of the digital sheath or lavage of the sheath should be performed to improve the prognosis.[13] Closure of the tendon laceration together with the digital sheath is indicated in most cases unless gross contamination is present.

Antibiotic therapy is dictated by the extent and duration of contamination. Systemic antibiotics should be considered for all wounds. More intensive antibiotic therapies are strongly recommended in more involved cases. These antibiotic options are discussed in more detail later in the chapter when discussing treatment of contaminated synovial structures.

Prognosis for Tendon and Ligament Lacerations

Extensor tendon lacerations have a good to excellent prognosis (better than 70%)[31] for return to full function with minimal intervention. Complications include prolonged healing and recurrent treatment of wounds and excessive granulation tissue and scar. Horses that are left untreated or turned out without fetlock support can develop permanent flexural deformity of the fetlock.

Flexor tendon lacerations have a good prognosis for pasture or breeding soundness and a fair prognosis for athletic soundness. Foland et al. found that 59% of horses with flexor tendon lacerations returned to riding soundness and that 57% of horses with both SDFT and DDFT involvement returned to some level of riding.[12] Jordana et al. reported 55% of horses with tendon and/or ligament lacerations returned to work.[21] This was lowered significantly by the number of structures affected. Complications include restrictive fibrosis, infection, fetlock hyperextension, and chronic delayed healing due to tendon sheath involvement. Management of flexor tendon lacerations is an expensive proposition due to the long-term care and continual shoe support out to 6 months after surgery. Details on the surgical technique can be found in other texts.[9]

MUSCULOSKELETAL INFECTIONS

Clinical Signs

MSK infections often begin with a traumatic wound, although iatrogenic synovial sepsis following joint/tendon sheath/bursa injections can also occur. In the early hours after injury, wounds involving synovial structures typically do not result in significant lameness. Initial clinical signs are associated with the severity of the

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Figure 12.16. Characteristic elevation of the toe during weight-bearing following laceration or rupture of the deep digital flexor tendon.



Figure 12.17. Typical location and appearance of a laceration of the flexor tendons in the mid-metatarsal region. The SDFT can be seen outside the wound and digital palpation revealed a lacerated DDFT as well. *Source*: Courtesy of Dr. Gary Baxter.

wound and typically involve swelling, lameness associated with soft tissue and/or bone injury, and potentially hypovolemia due to blood loss. The onset of synovial sepsis coincides with significant worsening of lameness. Osteomyelitis will usually develop much later, but it should be considered with any wound that has bone involvement (Figure 12.19).



Figure 12.18. Sequestrum of the dorsal proximal third metatarsal bone. This radiograph was taken 6 weeks after a traumatic wound. The wound has since healed aside from a small draining tract. The radiopaque marker within a gauze sponge is seen over the site of the sequestrum. *Source*: Courtesy of Dr. Deb Straker.

Diagnosis

If synovial cavities are suspected to be involved, additional diagnostics such as radiography and ultrasonography may be necessary. This should be done prior to synoviocentesis whenever possible. Early introduction of a needle may result in gas gaining entry into the cavity, which potentially complicates a radiographic or ultrasonographic diagnosis. Synoviocentesis combined with cytological evaluation of the fluid will provide additional information regarding synovial involvement (Table 12.2). When performing synoviocentesis, the needle should be placed at a site remote from the wound using sterile technique to minimize the risk of iatrogenic contamination. Caution should be taken if a cellulitis is present. It is often advisable in these horses to bandage the limb and initiate antimicrobial therapy prior to performing synoviocentesis.

If a synovial sample cannot be obtained, sterile isotonic fluid can be injected into the joint to facilitate aspiration of fluid.[54] The urea concentration in the synovial fluid should be compared with that in the serum as this will determine the amount of dilution that occurred and aid in determining the relative synovial fluid parameters.[54] After aspirating synovial fluid for culture and cytological examination, the synovial structure should be distended with sterile fluid to determine if it communicates with the nearby wound.

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Traumatic wounds that involve a bony surface or chronic synovial sepsis are most likely to result in osteomyelitis or osteitis. Osteomyelitis refers to the inflammatory or infective process of the cortex and marrow components of bone.[17] If only cortical bone is infected, it is classified as osteitis.[17] Penetrating wound tracts that lead to bone should be treated aggressively. The presence of osteomyelitis/osteitis is significantly associated with nonsurvival in cases with synovial sepsis (Figure 12.20).[57,61] Radiography is commonly used to diagnose osteomyelitis/osteitis, but unfortunately, 30–50% of bone demineralization must occur before it can be observed on plain radiographs. This may require 14–21 days, resulting in very low sensitivity and a delayed diagnosis. magnetic resonance imaging (MRI) and scintigraphy are the modalities of choice for suspected cases of human osteomyelitis and septic arthritis and may also provide for an earlier diagnosis in horses.[20] Ultrasonography also can be effective in helping to diagnose osteomyelitis by imaging fluid pockets in and around the infected bone.[41]



Figure 12.19. This traumatic wound was associated with an open comminuted fracture of the fourth metatarsus. Wound debridement and lavage are important to prevent osteomyelitis in these horses. *Source*: Courtesy of Dr. Gary Baxter.

Treatment

Acute wounds involving synovial structures, including joints, bursae, and tendon sheaths, often directly introduce bacteria and other contaminants into the synovial space. The clinician's primary concern is removing the bacteria from the space before infection can become established. Early recognition and treatment of a synovial penetrating wound are imperative to reduce the risk of developing septic arthritis. One study reported that horses treated within 24 hours had a lower risk of developing septic arthritis compared with those treated after 24 hours.[16] Wereszka et al.[58] found that horses with septic tenosynovitis were significantly more likely to survive if treated during the first day after clinical signs of synovial infection were observed than horses that did not receive treatment within 10 days after clinical signs of infection appeared. However, more recent publications reported that the duration of time until treatment may not affect survival[33,57] or the ability to return to athletic function.[57] Logically, it would still be advised that the quicker or earlier these cases are treated, the better the success (Figure 12.21).

Any wound that communicates with a synovial structure or bone should be considered contaminated. Therefore, early treatment with antimicrobials may help decrease the progression to synovial sepsis and/or osteomyelitis/osteitis. Antimicrobials can be administered parenterally, regionally, locally, or a combination of all. Additional therapies for preventing infection in a synovial cavity include some form of synovial lavage, drainage or endoscopic exploration, and wound debridement with or without closure. Small wounds with minimal contamination can be managed conservatively, but the clinical response should be monitored closely, and any deterioration should be met with more aggressive treatment.

Systemic antibiotics can be initiated for any infected wound, regardless of the structures, to provide a broad spectrum of activity against common microbes. These antibiotics are maintained until sensitivity of the cultured organism is obtained or concerns about sepsis have diminished. Most commonly, a combination of penicillin (22,000 IU/kg every 6 hours IV, every 12 hours

Table 12.2. Synovial fluid parameters used to aid diagnosis of synovial sepsis.

Fluid analysis parameter	Normal joint	Traumatic synovitis	Septic synovitis
Total protein (g/L)	18±3 Less than 20	20–40	>40
Total nucleated cell count (× 10^9/L)	<1.0 <3.5 (tendon sheath)	<10.0	>30.0
Neutrophils (%)	<10	<10	>80

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Figure 12.20. Radiographic evidence of osteomyelitis of the lateral condyle of the distal third metacarpal bone secondary to a wound and chronic synovial sepsis. There is significant soft tissue swelling, gas pockets, and bone lysis. Note the hole drilled in MCIII where intraosseous perfusion had been performed.



Figure 12.21. This wound on the dorsal aspect of the fetlock healed by second intention and trapped infection within the fetlock joint. Chronic synovial infections such as these are more difficult to resolve than acute injuries. *Source:* Courtesy of Dr. Gary Baxter.

IM) and gentamicin (6.6 mg/kg every 24 hours IV) is used initially. Oral antibiotics can also be utilized (trimethoprim-sulfa, 15–30 mg/kg orally every 12 hours; doxycycline 10 mg/kg orally every 12 hours).

Enrofloxacin is relatively commonly used when indicated; it is broad spectrum and often effective when aminoglycosides are ineffective. However, there is concern that articular cartilage damage may occur with its use in foals and younger horses.[19] Chloramphenicol also has a wide spectrum of activity and has good tissue penetration. The concern associated with this drug is the risk to human health due to the possibility of aplastic anemia in exposed people.[5] Vancomycin has been used successfully in cases of methicillin-resistant staphylococcal infections.[38] Due to concerns about antimicrobial resistance, it is only recommended to use this drug when culture and sensitivity results warrant its use.

If osteomyelitis is suspected, aggressive debridement is warranted. The infected bone should be exposed, curetted, and effectively removed. Osteomyelitis can be challenging to treat and often requires a multifaceted antimicrobial plan.

For both synovial sepsis and osteomyelitis, systemic antibiotic therapy can be supported with regional antibiotics. Regional perfusion by intravenous or intraosseous techniques can be utilized. In some cases, when the osteomyelitis is closely associated with a joint, such as in some cases of septic physitis, it may be prudent to treat the joint with IA antibiotics as well. Research has shown that IA administration of antibiotics provides adequate concentrations in the subchondral bone.[59] Antibiotic-impregnated materials such as polymethyl methacrylate (PMMA) or sponges will provide prolonged release of antibiotics locally.

For wounds that are obviously infected but do not appear to involve bone or synovial structures, principles similar to preventing infection of a wound are followed. The wound can be cleaned and then debrided. Once the extent and nature of the wound has been established, a decision can be made to allow for secondary intentional healing or to close the wound primarily. In general, infected wounds are chronic and primary closure usually is not attempted. Instead, once adequate cleaning and debridement of the wound have been performed, the wound should be bandaged with an appropriate dressing.

Regional Antimicrobial Delivery Systems

Intrasynovial Antimicrobials

The administration of intrasynovial antibiotics is a cost-effective way to attain high concentrations of drugs (above minimum inhibitory concentration [MIC] values) in synovial fluid for at least 24 hours. Antimicrobials commonly are injected into a synovial cavity on a daily basis, or they can be deposited after a synovial lavage. They also can be delivered to the joint using a constant infusion system.[25] Initial studies using a single dose of 150 mg of gentamicin provided concentrations above the

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MIC for common pathogens for 24 hours.[27] A 150-mg dose of ceftiofur also has been shown to provide adequate concentrations for more than 24 hours.[32]

Amikacin has recently become one of the most widely used intrasynovial antibiotics; it has also been shown to be effective against a broader spectrum of bacteria than gentamicin and has concentrations above MIC values for common pathogens for up to 34 hours.[51] A dose of 250–500 mg is commonly administered. Other antibiotics that have been used are cefazolin, timentin, methicillin, and imipenem cilastatin.[2,48]

Regional Limb Perfusions

Regional limb perfusions have become the accepted method of providing increased tissue concentrations of antibiotics for the treatment of synovial and osseous infection, as well as for preventing the establishment of infection. The two main methods of delivery are intravenous (IVRLP) or intraosseous (IORLP). Both involve the application of a tourniquet proximal to the infected tissues, and the antibiotic is infused distal to the tourniquet (Figure 12.22B). A distal tourniquet should also be utilized if the infected tissues are proximal on the limb.[50] Several studies have shown that antimicrobial concentrations reach up to 100 times the MIC values for common pathogens.[6,35,40,46,47,59]

The technique is simple to perform and can be done under field conditions in the standing sedated horse or under general anesthesia. A pneumatic tourniquet is preferentially used (Figure 12.22A), and wide rubber tourniquets (i.e. Eschmarch) are acceptable, but narrow rubber tubing is not.[10,26] To assist in applying pressure to the digital vessels in the standing horse, gauze rolls can be applied against these vessels under the tourniquet. The perfusate should include an antibiotic diluted in a balanced polyionic solution. The most common antibiotic used is amikacin; also used are gentamicin, penicillin, ampicillin, cephalosporins, enrofloxacin, and vancomycin. Doses vary considerably, but in most clinical situations 1000–2000 mg of amikacin is used in adult horses diluted to a volume of 30–60 mL.[8,24,37] A smaller volume (30 mL) is recommended in foals.[22] For IVRLP, a 20- to 25-gauge butterfly catheter is used, the vein accessed, and the solution slowly infused. Application of the tourniquet for 15 minutes was found to be adequate to achieve maximum concentrations of amikacin in the coffin joint following IVRLP.[24] One study using a contrast medium showed maximal dye uptake in the soft tissues at 30 minutes.[23]

For intraosseous perfusion, a hole is drilled in the cortical bone of the third metacarpal/metatarsal bone for distal limb treatments. A 3.2- to 5.5-mm cannulated bone



Figure 12.22. Intravenous regional limb perfusion using a pneumatic tourniquet. The tourniquet is placed above the site of the lesion and a vessel is selected to infuse the antimicrobials. (A) The palmar vein. (B) The saphenous vein. Rolls of gauze can be applied over the vessels for extra pressure; these can be seen under the tourniquet in figures A and B. *Source*: Courtesy of Dr. Jeremy Hubert.

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screw with a Luer lock head is placed in the drilled and tapped hole. Alternatively, the end of a catheter extension set fits securely into the drilled hole. The remainder of the process is very similar: a tourniquet is placed proximal to the third metacarpal/metatarsal bone. The infusate, of similar volume and concentration as with IVRLP, is slowly injected avoiding excess pressure. The tourniquet should be left in place for 20–30 minutes following the injection. Intraosseous perfusion may be favored over IVRLP in cases that lack easy venous access or the presence of osteomyelitis. Data suggests osteomyelitic lesions may respond better to intraosseous perfusion.[29]

Antibiotic-Impregnated Materials

Antibiotics can be effectively delivered by slow release from impregnated materials. These products are left *in situ* to release the drug over an extended period of time, resulting in high local tissue concentrations. The most common depot material used for local antibiotic delivery in horses is PMMA, or bone cement.[37] It can be formed into beads, and the beads are incorporated in a strand of nonabsorbable material for ease of later removal, although they can be left in place unless they become a source of clinical concern themselves. Elution rates of incorporated antibiotics are highly variable and depend upon a number of factors. Based on the most common pathogens encountered in equine orthopedic infection, 0.5–1 g of amikacin added to 10 g of PMMA and made into smaller cylindrical beads provides the ideal vehicle for drug elution in most clinical scenarios.

SPECIFIC TREATMENT STRATEGIES FOR MUSCULOSKELETAL INFECTIONS

Lavage and Drainage for Synovial Cavities

Through-and-Through Needle Lavage

A simple, quick way to lavage a synovial cavity is to place large-bore needles (14 or 16 g) into the joint at multiple sites and lavage a solution such as normal saline solution through the joint, alternating the ingress needle (Figure 12.23).[1] The horse must remain sedated and the area desensitized to facilitate the procedure. Fourteen-gauge needles are placed into the cavity at appropriate locations, and a fluid line is attached under a pressure system. A recent cadaver study using microspheres found that the greatest recovery of microspheres occurred with the first liter of lavage fluid through the tarsocrural joint.[28] This would suggest that the volume of fluid used is less important than the number or placement of needles/portals. A solution of 10% DMSO (110 mL in a 1-L bag of normal saline) can also be considered. Though a greater recovery of microspheres was found following needle lavage than following arthroscopic lavage,[28] needle lavage is usually not effective in more chronic infections because clots of fibrin and proliferative synovium block the needles. Furthermore, needle lavage cannot be used to assess further damage to the joint and is likely best suited for acute simple punctures of synovial cavities.[45]



Figure 12.23. Through-and-through needle joint lavage is performed using hypodermic needles. The ingress needles are alternated during the lavage, and all synovial pouches should be lavaged if possible.

Arthroscopic/Endoscopic Lavage

Infected synovial cavities are usually best lavaged and debrided through arthroscopic/endoscopic visualization. Foreign material, fibrin, and bone fragmentation if present can be seen and removed, and the joint itself can be debrided accordingly. A more accurate prognosis can also be provided.[61] Lesions to the cartilage, tendons, and bone can be identified and treated appropriately. Partial synovectomy can be performed to aid in debridement of the joint by allowing better visualization of the entire space. Most joints, bursae, and tendon sheaths are accessible for an arthroscopic/endoscopic lavage, and this is the preferential treatment for any synovial injury that is more than 24 hours old.[2] Wounds that have large puncture holes are not suitable for this technique, however, because this precludes adequate distension and visualization of the joint.

After arthroscopic lavage, the arthroscopic portals can be left unsutured to allow for continued passive drainage and repeat lavage in the standing horse. One clinical study showed good results using open wound drainage.[49] It may be especially helpful in chronic synovial wounds. The portals are allowed to close by second intention, but close attention to bandaging is critical.

A variation to this technique is to place an ingress system within the synovial cavity with a smaller egress arthrotomy. This permits repeated lavage and instillation of antimicrobials into the cavity with continued passive drainage. This technique is commonly used for tendon sheath infections. Soft fenestrated drains, such as a Jackson Pratt drain (Jackson Pratt Hubless, American Hospital Supply, Chicago), are placed within a cavity with the drain distad and sutured in place. This same system can be used for constant infusion systems of antimicrobials.[30] A balloon reservoir containing antimicrobials can be attached to the system, allowing a constant infusion.[25] One concern of this system is the potential

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for development of a superimposed infection if the system is not kept very clean with sterile bandaging and skin preparations.

Prognosis for Musculoskeletal Infections

With appropriate treatment, horses with MSK infections have a good prognosis. Cousty et al.[7] reported 90.3% of horses (foals and adults) with septic synovial structures had a favorable outcome (no lameness, mild synovial effusion, and return to function). Another report found 84% of horses with infection of a synovial structure survived to discharge and 54% returned to athletic function.[57] Factors that had a negative impact on prognosis were increased intrasynovial concentration of TP at the time of admission[33] and positive bacterial culture of synovial fluid.[56] Radiological evidence of osseous involvement in cases of synovial sepsis has been associated with a poorer long-term outcome.[55]

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THE POORLY PERFORMING HORSE

Elizabeth J. Davidson

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INTRODUCTION

Evaluation of the poor performer is one of the most challenging aspects facing the equine clinician. Because all body systems in the horse must function in a coordinated and harmonious way for good athletic performance, it bears to reason that the number of potential performance limiting abnormalities is extensive. When clinical signs are obvious, for example, a loud abnormal respiratory noise during exercise, selecting the appropriate diagnostic tools to accurately identify the causative abnormality is straightforward. However, many poorly performing horses present with subtle, inconsistent abnormalities only during exercise, which greatly complicates the diagnostic process.

The type of clinical complaints often reflects the horse's specific equestrian sport.[2,3] For example, a Thoroughbred racehorse may present for lugging out down the homestretch, whereas a Warmblood dressage horse may fall out of rhythm during canter pirouettes. Although it is not necessary for the examining veterinarian to be an accomplished rider or trainer, a solid working knowledge of the horse's discipline is important. It can be difficult to assess a horse with poor-quality extended trot without prior knowledge of what a good extended trot should look like. Additional examples of complaints based on athletic endeavors are given in Table 12.3.[2,3]

Evaluation of these difficult cases is time consuming and requires comprehensive assessment including an accurate history, a detailed clinical examination, complementary diagnostic tools, and specialized techniques.

SIGNALMENT AND HISTORY

Poor performance evaluation starts with obtaining the horse's signalment including the horse's occupation. Some underlying conditions are more common in young horses, while others are more common in older horses. For example, developmental bone diseases such as osteochondrosis frequently hinder young horse athleticism, while wear-and-tear conditions such as degenerative joint disease afflict the older horse. Other conditions such as exercise-induced pulmonary hemorrhage are associated with exercise intensity and affect a variety of horse breeds and ages. The horse's occupation may also influence injury type. For example, stress fractures are common in the racing performance horse, while hindlimb suspensory desmitis is common in dressage horses. It is also important to recognize that to some extent the horse's signalment affects its innate ability to perform and lack of ability is often misinterpreted as poor performance by owners/trainers. In older horses, age-related physiological changes decrease their athletic capacity. Their exercising VO_2max is approximately 25% less than the younger horse[8]; old horses just cannot run as fast as young horses. In the untested young horse, a lack of talent from physical or mental incapacity may be misperceived as poor performance; good breeding does not ensure good performance. The horse's breed and occupation are also important factors in performance. Certain breed characteristics are more desirable for specific equestrian events. For example, Quarter horses typically have a shorter length of stride combined with low head carriage, which makes them well suited for western performance; however, these characteristics are less suitable for upper-level dressage work. When there is a mismatch between horse type and its intended use, the owner's complaint of poor performance may be due to the horse's natural inability to perform desired gait qualities based on its conformation rather than an underlying medical condition, i.e. the wrong horse for the job.

The value in obtaining a thorough and accurate history cannot be overstated. This includes not only a detailed description of presenting complaint(s) and the duration but also past and present performance history. Information from the owner/trainer can provide valuable insight into the potential abnormalities affecting their horse since it relays the magnitude of the problem(s) and whether the horse was ever a good performer. Response to medications and/or exercise modifications and previous lameness are also important historical data. If possible, the veterinarian should determine the answers to the following questions:

1. What is the training schedule and have there been changes? Rapid increases in training intensity may be "too much too soon." For example, the horse being reintroduced to exercise after lay-up for injury may require additional time for sufficient healing or be suffering from physical fatigue due to lack of fitness. Young and/or unproven athletic horses that are objecting to recent increases may have reached their mental or physical limit.
2. Has there been a traumatic event? Recent falls or trailer accident may indicate an underlying musculoskeletal injury.
3. Under what circumstances does the problem occur? Are there consistent factors that are more likely to induce the complaint? The owner should try to determine exactly when the problem occurs and if it is related to a specific reason. For example, the horse trips during downward transition from canter to trot.
4. Is the problem consistent? One of the biggest obstacles when evaluating the poor performer is the inability to reliably reproduce the abnormal clinical signs. This is especially true when the inciting factors are unknown or the owner has empirically rested the horse prior to veterinary evaluation. Ideally, the horse should be fit and abnormalities consistent prior to evaluation.

Table 12.3. Types of poor performance problems.[2,3]

Horse occupation	Clinical problems	Potential cause(s)
Jumpers (hunters, eventers, show jumpers)	Refuses to jump and/or knocks down rails	MS pain or rider error
	Drifts to one side over fences	MS pain, lameness
	Prefers to land on 1 lead	FL lameness, less commonly HL lameness
	Switching leads in the hind end	HL lameness or occasionally neurologic
	Takes a small or ½ canter stride immediately prior to fence ("chips in")	MS pain or rider error
	Rushes fences	MS pain, ±behavioral in anxious horses
	Switches lead in between or immediately prior to fence	HL lameness or rider miscue
	Difficulty with related distances between combination fences	MS pain
	Loss/lack of power	Bilateral HL lameness or sacroiliac pain
	Change in jumping style, usually loss of bascule or jumps flat	Back pain, FL lameness, HL lameness, or combination
Team roping	**Header**	
	Lunges across arena when pulling the steer for heeler to rope	HL lameness, such as distal hock pain
	Acts up in roping box (rears, spins, looks away from steer)	MS pain or behavioral if the horse is tired and has had too many steers roped on him
	Refuses to run fast to the steer	Bilateral FL or HL lameness
	Heeler	
	Refuses to stay in stop position and/or bounces out of stop position	HL lameness such as distal tarsal joint pain
	Stops at an angle instead of square to the steer	FL lameness
	Poor turning after header ropes the steer horns	MS pain
Tie-down and breakaway	Does not stay in the stop or quits working the rope	MS pain, ±HL pain
	Drifts to the side, backs up at an angle after stopping	If the horse drifts right, left HL MS pain
	Decreased running speed	FL lameness
Barrel racing	Runs past the first barrel or takes wide turns	FL lameness
	Decreased speed	MS pain or EIPH
	Refuses to enter arena or acts up (rears, spins)	MS pain or behavioral
Cutting	Does not stay down when stopping or changing directions to mirror cow movement	HL pain such as distal hock joint or medial femorotibial joint pain
	Difficult moving to the right or the left	MS pain; desmitis, sacroiliac pain, and/or thoracolumbar myositis
Dressage	Resists or kicks out at rider aids	Usually MS pain
	Behind the bit	Bilateral limb lameness or bit problems
	Lack of impulsion	HL lameness or sacroiliac pain
	Open mouth, teeth grinding	MS pain or gastric ulcers
	Inability to perform medium or extended trot, may break into canter	FL or HL lameness
	Difficult for lateral movements	FL or HL lameness
	Becomes disunited at the canter	HL lameness or neurological disease or lack of fitness
	Late flying lead changes or difficult changing leads	HL lameness
	Heavy on one rein, crookedness, or reluctance to take even bit contact	FL lameness
	Bucking, rearing, bolting	MS pain, either lameness or back/sacroiliac or both
	Saddle slip	HL lameness
Reining	Bouncing, sideways, or rough stops	HL lameness such as distal tarsal pain or proximal suspensory
	Backing sideways	MS pain
	Choppy, frantic circles	FL or HL lameness
	Over bent when circling	MS pain
	Late or early lead changes	HL lameness
	Spins off center, loss of pivot foot position	HL lameness
	Does not pivot on hind end in rollbacks	HL lameness
	Wrings tail, flips head, mouthing the bit	Lameness or back pain
Endurance	Shortened or choppy gait on hard ground	Sore feet or coronitis or traumatic laminitis
	Stiff or rigid across the back	Bilateral lameness or back pain or exertional myopathy
	Easily fatigued	Chronic lameness or lack of fitness or respiratory disease
	Unbalanced gaits	MS pain

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Table 12.3. (Continued)

Horse occupation	Clinical problems	Potential cause(s)
Racehorse: Thoroughbred	Lug in or out, especially during final stretch	Lameness
	Decline in speed	Lameness, respiratory, or cardiac abnormalities
	Does not cool out normally	Chronic lameness or respiratory disease
	Plaiting (hindlimbs travel close together)	HL lameness commonly upper limb
	Stiff hindlimb gait	HL lameness or back pain or both
	Not using the hindlimbs or weak behind or slipping/falling out behind	Lameness or neurologic deficits
	Shorts choppy trot	Bilateral lameness
	Gait improves with exercise	Bilateral lameness such as distal hock pain
Racehorse: Standardbred	Drifts in or out	Lameness, most horses bear away from pain site
	Decline in last quarter mile speed	MS pain or respiratory disorder or atrial fibrillation
	Makes breaks in gait	MS pain
	Interference	Compensatory limb lameness
	Will not take a hold of the bit	Bilateral lameness or fatigue
	Worse on turns	Carpal, fetlock, or stifle lameness
	Off when jogging but improved when racing	FL pain such as carpal lameness
	Stiffness and soreness when walking to the track	Bilateral FL or HL lameness

MS = musculoskeletal, FL = forelimb, HL = hindlimb.

5. What is the current management of the horse? Have there been changes? Modification in the horse's diet such as switching to a higher protein feed or from grass to alfalfa may impact its performance especially in high strung horses. Older horses with low-grade chronic osteoarthritis might be less tolerant of long periods of stall confinement.
6. Has the horse received any medications? If so, have the clinical signs changed? Improvement in performance with nonsteroidal anti-inflammatory therapy, such as phenylbutazone (2.2 mg/kg) twice a day for 7–14 days, is strong indication that the problem is pain related. This information helps to rule out behavioral or other non-orthopedic issues.
7. Has an alternative rider been tried? Changes in performance may be related to the rider's skill. Professional riders may be able to improve the overall quality of the horse's performance, while less experienced riders may be unable to consistently give the horse the proper rider commands or miscue or be unable to navigate specific patterns or jumping courses with proficiency. Unbalanced riders or heavy riders may induce hindlimb and/or back problems.
8. Have there been recent tack changes? Horses may object to a poor fitting saddle or saddle pad. A new bit or mouthpiece may be too harsh for the individual horse's mouth.
9. Has the horse ever performed well? This is key information when trying to sort out if a horse simply lacks physical and mental capacity for its intended occupation. This is a common scenario with owners that have a limited budget and buy young untrained or minimally trained horses or off-the-track Thoroughbreds because they are inexpensive. Despite the horse's potential based on breeding or conformation, unproven performers may not be able to live up to their owner's expectations.

CLINICAL EXAMINATION

The most common cause of poor performance in the athletic horse is musculoskeletal injury. Although overt lameness is easily recognized and an accepted cause for poor performance, mild or subtle lameness is often overlooked or not identified as a potential cause of decreased performance by their owners. In fact, a high proportion of "owner-sound" horses are lame when assessed by a skilled lameness diagnostician.[5,7] Therefore, comprehensive orthopedic evaluation is recommended in any poorly performing horse even without a history of lameness.

Examination starts with thorough physical examination including inspection of body for symmetry (or asymmetry), limb palpation, assessment of hoof conformation and horseshoe, static joint flexion testing, and palpation of the horse's topline. Gait assessment, i.e. the lameness examination, should be performed in a systemic manner preferably with the horse exercising on hard and soft surfaces. Subtle lameness may be more apparent on a specific surface or in small tight circles. This is particularly important for the horse with bilateral limb lameness since they often appear to be sound in straight lines.

Assessment of the horse's gaits while lunging at the walk, trot, and canter is also indicated. Head/neck position, overall body flexibility, willingness to exercise, and balance of gaits should be keenly observed. Low-grade musculoskeletal pain often manifests as subtle uneven gait or a preferred gait in one direction. Horses with cervical pain may preferentially exercise with its head/neck tilted to one side or hold the neck in a straightforward neutral position with limited bend at the poll. Horses with mild hindlimb lameness may canter with its hind end to the inside or outside of the circle, usually drifting away from the lame limb. Horses with bilateral hindlimb lameness and/or sacroiliac may exhibit overall lack of impulsion, stiff/stilted joint movement when

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cantering, or simultaneous hindlimb push off at the canter ("bunny hopping"). Assessment of gait change after implementation of tack can also be helpful. Fit of the saddle and bridle and their suitability can be assessed. Systematic application of one or more pieces of tack, side reins at various degrees of tension, or weighted surcingle may induce axial skeletal pain and/or exacerbate lameness.

Since many poor performance abnormalities are only apparent or exacerbated with a rider, the next step is the ridden evaluation. This is particularly true in sport horses when the abnormalities occur only during specific movements, sport-specific gaits, and/or changes of pace such as canter-to-trot transitions. In these horses, ridden exercise is critical. Traditionally, gait abnormalities noted with the application of a saddle and rider have been attributed to back pain. Affected horse may exhibit shortened length of stride, lack of hindlimb impulsion, reluctance to work, and/or a stiff back. However, these clinical signs are not pathognomonic for back pain. Lame horses, especially those with bilateral hindlimb lameness, also exhibit the same abnormal gait characteristics. Lame horses adapt their gaits by decreasing extension of the thoracolumbar-sacral region.[6] This resultant trunk stiffness is often perceived as back pain by the rider even though the underlying pain and decreased back flexibility are due to limb lameness. Nervous or anxious horses may also exhibit short choppy gaits and stiffen or tense their backs until they are more comfortable with their surroundings.

The addition of a rider's weight increases the load applied to the horse's limbs, which many explain why many horses with subtle lameness, especially hindlimb lameness, are only visibly apparent when ridden.[1] Complaints of horses being more comfortable when trotting in one direction or posting on one diagonal may be associated with unilateral hindlimb lameness. Peak forces are higher on the sitting trot diagonal compared with the rising trot diagonal,[9] and hindlimb lameness is often worse when the rider sits on the diagonal of the lame leg.[4] In addition to uneven load stresses on the limbs, rising trot also creates asymmetrical stresses on the back. Increased lameness and/or back stiffness is frequently more obvious during sitting trot vs. trotting in the jumping position. Horses with back pain or bilateral hindlimb lameness may be resistant to collected movements or other maneuvers that require the rider to sit deeply in the saddle.

Based on what the owner feels when riding, they often formulate assumptions regarding the underlying cause of the horse's clinical signs. However, rider preconceptions should be interpreted cautiously since they may be incorrect. For example, difficulty bending correctly in a circle or the horse being heavy in the bridle is often perceived as neck pain by the rider. Although horses with cervical pain can exhibit these abnormalities, more often these gait abnormalities are due to forelimb lameness. In response to pain, horses with mild forelimb lameness will shift their head and neck to one side prior to overt limping and obvious head/neck nod. For riders, this uneven head and neck position results in increased rein pressure or heaviness in one hand. Horses with mild bilateral symmetrical forelimb lameness may feel like they are "heavy on the forehand" to the rider. Saddle slip where the saddle consistently slips to one side of the horse back has been blamed on ill-fitting saddles; however, current research indicates that lameness influences the presence of saddle slip, with most saddles slipped to the side of the lame hindlimb.[3,7] In these horses, comprehensive evaluation including keen observation by the attending veterinary is paramount to distinguish inciting causes from their resultant gait abnormality.

DIAGNOSTICS

Diagnostic analgesia can help determine if the horse's poor performance is due to musculoskeletal pain vs. behavioral, training, or other problems. In addition, nerve and joint blocks can elucidate the authentic site of underlying pain. Neck, back, and pelvic abnormalities are commonly incriminated by their riders; however, the presenting clinical signs in these horses such as body stiffness, resistance to rider aids, and unwillingness to bend are also exhibited in horses with limb lameness. In these horses, resolution of the abnormal gait following palmar digital analgesia is definitive evidence that the clinical signs are due to foot pain and not due to axial skeleton pain. In horses with bilateral symmetrical lameness, simultaneous blocking of both limbs may be diagnostic. For example, in Thoroughbred racehorse with short choppy hindlimb gait, blocking the distal plantar metatarsal nerves in both hindlimbs may be indicative of plantar osteochondral disease of the distal third metatarsus. Alternatively, response to joint therapy may be used for diagnosis. However, not all horses with painful joints will respond favorably to intra-articular medications, and limited or no improvement in clinical signs does not necessarily eliminate those sites as the source of problem.

Comprehensive nuclear scintigraphic evaluation can be beneficial in the poor performer. It is particularly useful in the racehorse with intermittent lameness, appeared to be "sore all over," and those with mild bilateral gait abnormalities. Common abnormalities include distal palmar/plantar fetlock pathology, bilateral upper limb stress fractures, and pelvic fractures. Scintigraphic imaging in riding horses may also be a valuable diagnostic aid. Axial skeletal disease, sacroiliac abnormalities, and rib lesions can be identified with this modality. Albeit frustrating, a "negative" bone scan does not necessarily indicate the horse does not have a musculoskeletal problem. Horses with stifle synovitis and soft tissue injuries such as proximal suspensory desmitis frequently do not have associated areas of increased radiopharmaceutical uptake.

There are many other reasons for a horse to experience decline in performance, and additional investigations may be necessary. Subtle neurological problems can have a dramatic effect on gait. For horses that trip or exhibit disunited gaits or incoordination, a complete neurological evaluation is indicated including cervical imaging and cerebrospinal fluid analysis. Testing for muscle conditions such as polysaccharide storage myopathy or exertional rhabdomyolysis may also be beneficial. Abnormalities of the cardiac and respiratory systems are also common in poor performers. Previously

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restricted to treadmill evaluation, upper airway endoscopy and telemetric electrocardiogram can be readily performed in the field, a considerable advantage for the riding horse. Numerous other cytological, serological, and metabolic testing may also be necessary.

TREATMENT

After diagnosis(es) has(have) been established, appropriate treatment strategies can be employed. Given the magnitude of potential causes of poor performance, concerted and exhaustive efforts in the evaluation and diagnostic process may be necessary for diagnosis. However challenging, an accurate diagnosis is paramount since the best therapeutic practices are targeted at the causative underlying injury site(s). Whole horse treatments such as repeated or frequent systemic anti-inflammatory treatment and indiscriminate joint injections should be avoided since they may prolong diagnosis, resulting in further deterioration of injury and decreased likelihood of return to performance.

In summary, the poorly performing horse is a diagnostic and time-consuming challenge. These horses often require a comprehensive multisystem evaluation. Musculoskeletal abnormalities prevail, and most horses that are lame are often sore in more than one limb and/or have axial skeleton pain. In the riding horse, any part of the horse–rider–saddle combination can be problematic and should be investigated thoroughly. Some horses do not have underlying medical abnormalities and simply lack the physical ability to achieve owner's expectation for desired athletic events. Recognition of the underlying cause(s) of poor performance is crucial for successful management and return to athletic function.

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EVALUATION OF PROPER SADDLE FIT

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INTRODUCTION

Proper saddle fit and use is a critical component of optimum athletic performance and the prevention of back pain or discomfort in horses. Unfortunately, it is not always clear what is meant by "good saddle fit," and objective parameters for assessing saddle fit and function during dynamic activities are lacking and often difficult.[2] In an English study of sport horses, 18% had poor fitting saddles defined to be in uneven panel contact, and 33% had unbalanced saddles with either cranial or caudal angulation of the tree.[4] Even for qualified saddle fitters, agreement has been reported to only be fair (kappa = 0.32) for measures of overall saddle fit among members of the Society of Master Saddlers.[5] Saddles are often fit for the seat of the rider; however, the correct width or length of the saddle for a particular horse, adequate padding, and stirrup attachment or positioning, which influence a rider's leg positioning and balance on the horse, are often overlooked.[9] Considerable interactions between the rider, saddle, and horse's back influence the rider's comfort and effectiveness in the saddle and the movement patterns of the horse's back and saddle.[8] Poorly fitting or improperly used saddles and saddle pads often contribute to back pain and poor performance. It is imperative to understand the basic principles of assessing proper saddle fit and the effects of proper vs. poorly fitting saddles on both the rider and the horse.

CLINICAL SIGNS OF POOR SADDLE FIT

Signs of asymmetric rider positioning in the saddle include uneven wear patterns on the seat from asymmetric rider contact with the seat bones (tuber ischii) or asymmetrical leather wear over the stirrup irons or stitching under the rider's legs. Horses with back pain related to poor saddle fit may display adverse or abnormal behavioral signs such as ear pinning, teeth grinding, head tossing, tail swishing, unwillingness to stand still during saddle or saddle pad placement, or biting or kicking while being saddled or while tightening the girth.[7] Asymmetric sweat patterns on the horse or saddle pad, asymmetric dirt patterns on the saddle pad, regional hair loss in areas of saddle contact, local edema, and open sores are all acute signs of poor saddle fit. Chronic signs of poorly fitting saddles include white hairs or granulomas and muscle atrophy in the saddle region.

Ill-fitting saddles can cause horses to hollow their backs (i.e. induced lordosis) and resent mounting. Affected horses often have reduced spinal mobility and are slow to warm up or relax during initial riding but may be completely normal during lunging or work in the round pen due to unweighted exercise. Regular bucking or rearing, resistance to work that worsens with time, difficulty with collection or maintaining impulsion, and resentment or ignoring training aids also may be indicative of poor saddle fit issues related to ridden exercise. Reduced protraction of the thoracic limb (i.e. shortened stride), stumbling or tripping, or obvious lameness can be produced by poorly fitting saddles. Upper pelvic limb lameness and lack of impulsion also may be exacerbated by increased saddle pressure.[4]

SADDLE EXAMINATION

To evaluate proper saddle fit, the saddle is assessed for manufacturer defects or signs of wear, and the rider–saddle and saddle–horse interfaces are evaluated during static and dynamic examinations. The ideal saddle provides uniform contact and pressure across all regions of the back during both static and dynamic assessments.[2] There should be no elevated or depressed areas that predispose to increased pressure and no left-to-right or craniocaudal asymmetries. The most common saddle fitting faults include bridging, increased pressure over the withers at the front of the saddle, high overall pressure, and asymmetric or localized pressure points.[10]

Examination of the saddle begins with assessing saddle construction and wear. The seat is evaluated for asymmetric wear, which often indicates that the rider is uneven in the saddle. Uneven flaps and stirrup bar placement are manufacturing defects, and an affected saddle should be returned for replacement. Worn or dried out leather straps must be replaced due to safety concerns.

Because the tree forms the foundation of the saddle, any saddle with a broken or asymmetric tree must be repaired or discarded. Saddles with broken trees have reduced stability and support, allowing the saddle to deform and causing localized increased pressure. Broken and twisted trees are evaluated visually by inspecting the symmetry. Excessive flexibility is assessed by applying craniocaudal forces to the bars or by applying lateral compressive forces to the head of the tree. The ventral surface of the panels is inspected for adequate flocking, left-to-right angular symmetry, and craniocaudal symmetry in the flocking. Horses with asymmetric withers often affect the symmetry of the cranial portion of the panels. The caudal edge of the panels should be wide and flat to maximize comfort for the horse, rather than narrow or pointed, which can cause localized pressure at the back edge of the saddle. In Western saddles, the fleece along the ventral surface should be palpated carefully with a flat hand to identify any protruding screws, nails, or plant burrs that can produce localized pressure or wounds. The width of the gullet should be two to three fingers wide along the entire length to allow positioning and movement of the dorsal spinous processes.[6]

STATIC EXAMINATION OF SADDLE FIT

The static examination of saddle fit on the horse's back is accomplished by visual inspection and palpation of the saddle–horse interface in the standing horse. A useful analogy for assessing saddle fit and proper saddle pad

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use is that the saddle should fit the horse's back like a well-made shoe and the saddle pad functions like a sock.

In a well-fitting saddle, the addition of a thin saddle pad should result in optimal saddle and saddle pad function and should not produce increased pressure or back pain. However, a proper fitting saddle used with an excessive number or increased thickness of saddle pads will likely produce localized pressure and back problems. In poor fitting saddles, the addition of a thin pad will likely not improve saddle fit. Likewise, the use of several saddle pads to compensate for a poor fitting saddle (such as a poor fitting shoe with several socks) will likely exaggerate saddle pressure and produce back pain. That said, wide fitting saddles may benefit from the use of several pads as a temporary solution to mismatched saddle horse sizes.

The static examination of saddle fit begins with the owner placing the saddle on the horse's back. The saddle is initially evaluated without any pads or girth to limit influence by other confounding variables. The owner is instructed to place and position the saddle on the horse's back in the usual manner. This provides an opportunity to qualitatively assess the horse's response and behavior to saddle placement and to evaluate saddle fit and positioning. Horses that pin their ears or attempt to bite or kick with saddle placement likely have or have had issues with poor fitting saddles and secondary back pain. As noted above, the tree provides the framework for the saddle and acts to transfer the rider's weight to the back of the horse. Therefore, the length, width, angles, and twist (or shape) of the tree ideally must match the contour and symmetry of the individual horse's back. The flare at the front of the tree should fit over the withers and allow free scapular movements. The width and angle of the tree must match the lateral slope of the withers.

In English saddles, the cranial edge of the saddle should rest at the caudal edge of the scapula. In Western saddles, the saddle normally covers the dorsal aspect of the scapulae, but the skirt should flare cranially to allow caudal rotation of the scapula during thoracic limb protraction. New saddles often have very stiff and inflexible skirts that may occasionally rub on the dorsal scapula cranially or lumbar region caudally. The length of the saddle should be proportionate to the length of the horse's back and the size of the rider. Saddles that are too short localize pressure and likely cause back pain. Saddles that are too long tend to bridge across the horse's back and localize pressure at the withers and caudal saddle, with little or no contact in the middle of the saddle where most of the rider's weight is located. Saddle bridging is a common problem in lordotic horses; this can be temporarily managed by placing additional padding or shims to fill in the noncontact areas at the center of the saddle to provide uniform saddle contact. Proper skirt length in Western saddles should provide 4–6 inches of clearance in front of the tuber coxae. Long skirts can cause friction rubs on the cranial aspect of the tuber coxae and restrict lateral bending of the trunk.

Stability of the saddle on the horse's back can be estimated by applying cranial-to-caudal, side-to-side, and diagonal rocking motions to the saddle. Properly fitting saddles are very stable and allow no displacement of the saddle. Wide saddles or the use of too many saddle pads causes instability and excessive displacement of the saddle during these induced movements.

In properly fitting and positioned saddles, the pommel and cantle of most saddles are horizontally level, and the deepest portion of the seat should be located near the center of the seat.[2] Saddles that slope downward in the front cause the rider's weight to be shifted over the withers. In contrast, caudally sloping seats cause the rider's weight to be shifted caudally, away from the horse's center of gravity. The position of the girth or cinch should correspond to the narrowest portion of the chest; otherwise, the saddle tends to constantly slide forward or backward, producing back pain and altering performance.

Properly fitting saddles should have two to three fingers wide (4–6 cm) of vertical clearance between the pommel gullet and the dorsal withers (Figure 12.24). Narrow saddles tend to sit higher and produce a taller saddle–wither gap, whereas wide saddles come close to resting on the dorsal spinous processes due to lack of lateral contact and support over the withers (Figure 12.25). Gliding a flat hand under the front edge of a well-fitting saddle and over the withers should provide a sensation of a smooth, uniform, soft contact bilaterally (Figure 12.26). The angle of the head of the tree should coincide with the lateral slope of the withers. Asymmetric trees or scapulae can produce left-to-right differences in saddle contact over the lateral wither region. Gliding a flat hand under the panels or skirt of a well-fitting saddle should also provide a smooth, uniform contact bilaterally, which closely follows the contour of the horse's back. The flocking in English saddles should be symmetric, of uniform firmness, and evenly distributed with no prominences or depressions. Overstuffed panels form round, firm cylinders that do not readily conform to the shape of the horse's back. Underflocked panels are flat and provide no cushion. The panels at the back edge of an English saddle should be flat and in contact with the horse's back. Some saddles have pronounced upward curving panels that cause a substantial reduction in the surface area over which to distribute a rider's weight. The gullet can be visualized from behind the horse and should provide ample clearance for the dorsal spinous processes.



Figure 12.24. Proper spacing between the saddle and dorsal aspect of the withers, with a saddle pad in place.

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Figure 12.25. Poorly fitting Western saddle with too close of contact between the pommel gullet and the dorsal aspect of the withers.



Figure 12.26. Evaluation of smooth, uniform contact between the front portion of the panel and the underlying dorsal aspect of the scapula. The contact pressure should be symmetrical from left to right.

STATIC EXAMINATION OF SADDLE PADS

Static evaluation of the combined saddle and saddle pad fit is accomplished by repeating the above examination with the saddle pad in place. If more than 1 saddle pad is typically used by a rider, then pads or blankets are sequentially added or exchanged during the examination to assess the influence of select saddle pads or saddle pad combinations on proper saddle fit. The size, shape, thickness, and material of the saddle pad should correspond to the intended use of the horse. Saddle pads should also be selected for specific temperature, moisture absorption, friction reduction, shock absorption or cushion, and uniform pressure distribution characteristics.

Saddle pads for a short-term dressage test have different requirements than saddle pads used for racing or during extended ridden activities (e.g. a 100-mile endurance ride). One method for indirectly assessing saddle fit and pressure is to ask the rider to ride the horse in a clean, white saddle pad prior to saddle fit assessment. Properly fitting saddles produce symmetric and uniform dirt or sweat patterns on the saddle pad in the shape of a butterfly. Regions of increased dirt and lack of dirt are noted bilaterally, which can help determine where the saddle fit should be examined more closely.

The size and shape of the saddle and saddle pad should be compared. Half pads or pads that are too small can cause increased localized pressure along their margins, especially if the edges are not tapered. Again, like wearing socks in a well-fitting shoe, it is important to have no foreign objects, wrinkles, or step defects in the saddle pads, which can cause discomfort and localized pressure. Saddle pads should always be pulled up into the gullet of the saddle in an effort to reduce friction and pressure over the dorsal withers. Saddle pads should also be evaluated for excessive wear, cleanliness, and moisture.

DYNAMIC EXAMINATION OF SADDLE FIT

Dynamic examination of saddle fit and saddle pad use involves assessing both horse and saddle movement during unridden and ridden exercise. The horse initially should be assessed for pain reactions or abnormal behavior in response to saddle placement and tightening of the girth. Saddle movement and the influence of saddle fit and saddle pads can then be assessed during locomotion in hand, on the lunge, or in a round pen.

Proper saddle fit should allow fluid movements of the thoracic limb without restricting any flexion/extension or lateral bending motion of the thoracolumbar spine. Proper saddle fit provides seamless interactions between the rider and the horse during dynamic activities and intended use. Poorly fitting saddles often cause horses to guard their back due to pain or muscle hypertonicity.[7] Scapula or spinal movement asymmetries can produce obvious one-sided or uneven saddle movement or saddle pad migration.

Evaluation of uniform sweat or dirt patterns on the saddle pad after both unridden and ridden exercise can provide insight into saddle fit and pressure distribution underneath the saddle. Recent research has used pressure-sensitive mats to obtain pressure distribution patterns and areas of peak pressures under both normally and abnormally fitting saddles.[1,3,9] The effects of saddle pad thickness and weight distribution of the rider in the saddle can also be quantified and monitored over time. Evaluation and knowledge of saddle and saddle pad construction and fit as well as rider–saddle–horse interactions is important in assessing causes of poor performance and back pain in horses. A saddle that does not fit the rider may alter the rider's position and balance and precipitate back pain for the rider.[2]

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PREPURCHASE EXAMINATION

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The prepurchase examination (PPE) is the most comprehensive examination a veterinarian will perform on a horse. It is not only an examination of overall health and soundness of a horse, but the information obtained is used to help make decisions about the ability and capability of performing an intended purpose for the prospective buyer(s).[24] However, the purpose of the PPE is not to evaluate or guarantee a horse's future behavior, trainability, or athletic ability.[26] The horse's future performance cannot be predicted or guaranteed by the PPE; it can only attempt to ascertain any current or potential problems that may affect future soundness.[11] The PPE is a collection of information used by the buyer to help make an informed reliable decision for purchase. The veterinarian's focus should be to be a facilitator in the transaction while always representing the buyer's best interests. Therefore, the veterinarian's role in performing the PPE is to evaluate and advise on the serviceability of a horse for a prospective buyer's intended purpose and to provide guidance on how any existing conditions may affect the future performance of the horse.[15,21] Evaluating and determining the suitability of a horse is generally considered the task of the prospective buyer, agent, and/or trainer.[21]

The veterinarian should be well informed as to the responsibilities and potential risks of performing PPEs. If the veterinarian is not comfortable with all aspects of performing and interpreting the PPE, it would be in their best interest to refer the examination to a colleague that has these skills. Performing the examination is not necessarily the most complicated component of the process as much as the interpretation, documentation, and reporting of the examination findings—crossing the t's and dotting the i's, as they say. The PPE is one of the most common claims brought against equine veterinarians for malpractice or veterinary negligence.[13] Issues recognized in some claims have included (1) owner and agent pressures resulting in compromise to the veterinarians normal procedures and standards, (2) overinterpretation of radiographs or laboratory results when an incomplete history was obtained or an inadequate physical examination was performed, (3) misunderstanding on the part of the prospective buyer as to the PPE not providing warranty to the transaction, and (4) incomplete or inaccurate information obtained from agent or trainer directly involved in the transaction.[13]

There are numerous parties involved in the PPE. At minimum there is the prospective buyer, seller, and examining veterinarian. In many cases there may also be an agent for the prospective buyer and agent for the seller or trainers, and the buyer may also have a trusted veterinarian that they wish to have involved. Throughout the process it should remain clear to everyone that the prospective buyer is the owner of information gained from the PPE. If a legal agent is representing the prospective buyer, then communications may be between the examining veterinarian and the agent; however the prospective buyer must be kept informed and receive the final PPE report. It is important to realize that all parties involved in a potential sale have a role in the process and that their roles should be acknowledged, respected, and addressed in one shape or form.[26]

The prospective buyer is the person financially obligated to the PPE and responsible for making the decision to purchase or to decline purchase. The relationship between the veterinarian and prospective buyer should be established early and clearly stated in the record. Pre-exam communication with the prospective buyer should outline the structure of the PPE, available ancillary testing, cost of services, limitations of the PPE, any financial limitations, and any and all conflicts of interest. The veterinarian is working for the prospective buyer; the prospective buyer is the owner of all information gained from the PPE.[11,15,21,23,26] If the PPE does not end in a change of ownership, it should be made clear to the prospective buyer that the findings of the PPE are privileged information and should not be discussed with anyone beyond the parties involved so as not to hinder any future potential sales.

The seller is the legal guardian of the horse being examined. This individual plays a critical role, in that anything done to the horse during the examination (i.e. riding under saddle, pulling blood for potential drug screening, sedation for imaging, etc.) can only be performed following consent from the seller. The seller is also the source of any pertinent history or medical records that should be collected prior to performing the PPE. If the seller is not present at the time of the PPE, signed consent forms or witnessed consent should be obtained prior to performing the PPE.[2,15,21,26] Although the PPE contract is between the prospective buyer and the veterinarian, the seller should remain well informed throughout the examination. Keeping in mind that the information gained from the PPE is the property of the veterinarian and the prospective buyer, consent from the prospective buyer must be granted before any sharing of materials.[15,21,26]

The veterinarian is the individual actually performing the PPE and plays a tremendous role in the collection, interpretation, and dissemination of PPE results. Another important role of the veterinarian is to recognize and alleviate any areas of conflict of interest. According to the American Association of Equine Practitioners (AAEP) Professional Conduct and Ethics Committee, the most common potential conflict of interest that is encountered in practice is when the seller or the buyer and seller are clients of the practice asked to perform the PPE.[4] It has commonly been recommended that a veterinarian should refuse to perform a PPE on a horse owned by their client. In many instances this can be difficult, and oftentimes both the seller and buyer are comfortable having the established veterinarian perform the examination. The AAEP Professional Conduct and Ethics Committee has addressed this issue and states

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that, if in this situation, there must be full disclosure to all parties that this relationship exists and that the seller give full disclosure of all medical records and history. All conflicts of interest and written agreements from all parties should be documented. The veterinarian should not act as the prospective buyer's agent and should not hold any responsibility for the prospective buyer's decision to complete or reject the transaction.[25]

The agent for the buyer or trainer may be separate individuals or one in the same. It is important to establish their role and legal powers prior to initiating the PPE. The agent or trainer will play a role in evaluating the suitability of the horse for their particular client and the horse's intended purpose. Werner recently published his approach to the PPE and outlined service maps that he established to help guide the examination and define the roles of all the individuals' involved in a successful PPE.[26] The reader is referred to this manuscript for further details on the PPE.

STRUCTURE OF THE PREPURCHASE EXAMINATION

The PPE should be broken down into different steps to make the examination progress more efficient and reduce the potential for deletions or inaccuracies in the examination.

Step One: Pre-examination

This step should be performed prior to the PPE appointment. A request should be made to obtain previous medical records and all pertinent history (performance career, current level of work, etc.) either from the seller or through permission from the seller, from the seller's primary veterinarian.[24,26] It is also during this step that all potential conflicts of interest be acknowledged, discussed, accepted, and documented. At this stage, if there is any refusal of information from the seller, or the veterinarian perceives any significant reason to doubt the seller's integrity, the veterinarian should document any conflicts and refuse to perform the examination due to a conflict of interest.[23,26] If the veterinarian has prior knowledge of the horse including any existing problems, they should insist that the seller gives permission to reveal all information to the prospective buyer. If permission is not granted, the veterinarian should graciously refuse the examination.[2]

Developing a PPE package of information that can be sent to the prospective buyer and seller prior to the PPE can save time on the day of the PPE and help to identify any potential conflicts prior to initiating the PPE. Prospective buyer and seller information should include all contact information (buyer, seller, agent, trainer), horse identification (name, breed, sex, age, markings, tattoos, freeze brands, and height), conformation of availability on the day of the PPE, and the appropriate prospective buyer/seller consent and waiver forms.[15,26] A detailed description of the PPE format can also be included. Once the information has been collected, the veterinarian can review the information in preparation for the appointment, and any conflicts can be resolved at this time.

Scheduling of the appointment time and location should also be carefully considered. Points of consideration should include the availability of all involved parties and competent help to perform the exam, scheduling conflicts of the examining veterinarian, location of the exam, and weather conditions.[23] If any of these conditions risk the quality and completeness of the examination, the veterinarian should consider postponing the examination. Documentation of any less than satisfactory conditions should be included in the final PPE report.[13] Additionally, the PPE can require considerable time to perform properly, and if not scheduled appropriately, the veterinarian can find himself/herself rushed and distracted from the task at hand. There are advantages to performing the PPE in the horse's home environment, but there cannot be distractions that will not allow for an accurate assessment of the horse. Space and footing is also very important especially when examining a performance horse. Working the horse on multiple level footings and having adequate space to exercise the horse on the lunge and under saddle are often imperative.[24]

Step Two: Review and Define PPE

Prior to initiating the PPE, the veterinarian should take the time to acknowledge all parties that are present and review the Step 1 information. Any questions or requests that the prospective buyer(s) might have pertaining to the examination should be answered at that time. The veterinarian should confirm the prospective buyer's expectations of the PPE and make it clear that although the PPE is a very complete and comprehensive examination, it may not identify all potential abnormalities.[11,21] It should be made clear that the PPE is a short-term contract lasting the day of the examination and not a warranty.[13,16] The ability of the veterinarian to predict the future soundness and suitability of the horse is impossible.[15] Recommendations for imaging or testing based on pre-exam information, signalment, intended purpose of the horse, and requests made by the prospective buyer can also be discussed at this stage. Additional specialty testing (i.e. reproductive ultrasound, upper airway endoscopy, advanced imaging, etc.) should also be outlined, and the indications for performing and costs associated with each test should be discussed. If a test is recommended or requested but not available at the examining practice, potential referral and its expense should be discussed.

Step Three: Prepurchase Examination

The PPE can be designed and carried out in various formats. A detailed description of the complete PPE will not be discussed, as this will vary greatly from case to case. Within any equine practice, a PPE may vary depending on the type of equine population, economics, and the needs of a particular client base. Regardless of the horse's value and the wants and needs of the client, it is important that the quality of the exam, communication, and reporting of findings be held to the utmost standard of care. The veterinarian should develop a systematic approach to perform each and every PPE using that system to help avoid overlooking portions of the exam.[11,21,24] It is common practice to use a dedicated worksheet that is designed specifically for the PPE and can be tailored to the veterinarian's methods. The worksheet should include patient identification (age, breed, sex, color, markings, tattoos, and brands), checkboxes and comment areas referring to general body condition, conformation, inspection of all body systems, soundness

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evaluation, general comments, communication comments, imaging interpretation, and comments concerning any specialty testing that was performed.[26] The worksheet should be maintained in the medical record. Use of a dedicated PPE worksheet will help to reduce the risk of an incomplete exam and inaccurate recall of findings in the formulation of the final report. There are a number of forms online that can be used as a guide for designing a PPE worksheet that is specific for the veterinarian's needs and wishes.

The basic PPE should include a visual inspection of the horse both in and out of the stall, during work, and following work. Any noted abnormal behavior or vices should be noted. This will give the veterinarian an idea of the horse's response to an unfamiliar environment and personnel, recovery from exercise, and any changes in stance or movement throughout the exam. Overall body condition and conformation should be closely evaluated and recorded.[2,11,15,21,24,26]

Physical Examination: An overall general examination of the horse should be performed using a muzzle to tail inspection of all body systems. It is also important to have knowledge of a horse's age based on dentition. Confirming that the horse's dentition corresponds to the age on record is important, as it is not uncommon to have marked discrepancy between the two in some cases.[21] Auscultation of the thorax and abdomen is performed during the initial exam. Auscultation of the lungs using a rebreathing bag is often performed following the dynamic exam. Palpation at this stage concentrates on skin health, identifying any lumps, bumps, or swellings, and careful inspection for common surgical scars (ventral midline, neurectomy, laryngeal, etc.). In the gray horse careful inspection for melanoma lesions at the base of the tail, in the perianal region, and in the region of the parotid salivary glands is important. If firm swellings are identified in the parotid region, consideration of upper airway endoscopy including inspection of the guttural pouches is advised. It is common to find melanotic plaques in the guttural pouches of gray horses with melanoma affecting this region.[5,7,27] In geldings and stallions it is advised to obtain permission to sedate the horse to allow close inspection of the penis and prepuce.[27] Small precancerous or cancerous lesions cannot be identified without close visual inspection. In mares, the seller should be questioned as to the mares' potential for pregnancy. If the response is unclear, it should be suggested to the prospective buyer that an examination for pregnancy be performed.[26]

The ophthalmic examination should be performed in a dark quiet space with an ophthalmoscope.[12] The goals of the ophthalmic examination should be to identify the presence of an abnormality, state if an abnormality will or will not have a significant effect on functional vision, and state whether the abnormality is likely or unlikely to progress over time.[12] The veterinarian should not document any quantitative comments concerning visual impairment or subjective statements with respect to the horse's suitability for riding with suspected visual impairment.[12] It is generally viewed as inappropriate and unnecessary to routinely dilate a horse's eyes during the PPE. The exception to this is if an abnormality is identified and the veterinarian, with the owner's permission, wishes to more fully examine the eye.[12]

Thorough evaluation of the cardiovascular system is important especially in sport horses. Accurate assessment of the heart and identification of heart murmurs can be challenging depending on the veterinarian's equipment and ability to detect subtle murmurs. Evaluating the horse before and after exercise can be useful; however the effect of exercise is a poor guide to the significance of a murmur.[17] A murmur may be physiologic or may be due to disease, not all of which affect a horse's performance negatively, depending on the nature of the murmur. Key factors to take into consideration include (1) the intended use of the horse, (2) safety to horse and rider, (3) the prospective buyer's understanding and willingness to accept risk, (4) the horse's past performance record, (5) age of the horse, (6) if the prospective buyer considering resale, and (7) insurability.[17] If a heart murmur is identified and the veterinarian cannot be confident that it is a physiologic murmur (i.e. second-degree atrioventricular block), further investigation utilizing echocardiography and electrocardiography is recommended.[17] If the intended use for the horse is breeding, many horses with moderate to severe cardiac disease are perfectly suitable for breeding purposes.[17]

Any abnormalities that are identified during this initial stage of the physical examination should be discussed with the prospective buyer and referral offered if the veterinarian is not familiar with the findings and uncomfortable with the interpretation.

Musculoskeletal Examination: Physical examination of the musculoskeletal system should follow a systematic approach and be detailed and methodical to avoid straying from the system. In the equine athlete it is very common to identify or palpate abnormal variations of normal that are considered blemishes (mild fetlock, tendon sheath, and tarsocrural effusion) and insignificant to the horse's future soundness. Recognizing obvious asymmetry between like effusive structures is of greater clinical significance than symmetry between structures.[15] For example, eliciting a mild response to deep palpation of the proximal suspensory in the front limbs of horses in work is a common finding; eliciting the same response in the hindlimbs should be of greater concern, depending on the degree of response.[20]

Step Four: Dynamic Examination

This portion of the examination is best performed over multiple level surfaces in an area with minimal distractions.[24] Having an assistant employed by the veterinarian who is familiar with PPEs and soundness evaluation is helpful and can help assure an efficient and uncomplicated examination. To accurately evaluate a horse's gait, the horse should be relaxed and trot freely on a loose lead. The basic PPE should include observing the horse at the walk and trot on the straightaway, at the trot and canter on the lunge, and after upper and lower flexion tests of all four limbs. The response to flexion tests is highly variable and should be interpreted with caution. A positive flexion test does not always correlate with the presence of lameness in a particular limb.[6] Many horses will trot off asymmetrically following flexion, but the response should resolve within 2–3 strides to be considered a negative response.[6,22] As long as all parties agree, and owner and rider consent is obtained, it is advised to also observe the horse under saddle.[11,15,26] It is recommended to use the horse's own tack to reduce any

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unwanted variables.[24] A percentage of horses will not display lameness until they are ridden.[6] This may bring subtle lameness to the surface that may not otherwise be visible. The horse can also be put under more strenuous demands giving the examiner the opportunity to perform a post-exercise physical examination.

A particularly difficult and oftentimes uncomfortable situation is recognizing and confidently documenting the presence of subtle neurologic deficits. During the static exam, evaluating for such signs as symmetric or asymmetric muscle atrophy, abnormal posture, or abnormal hoof wall wear can give clues to possible central, peripheral nerve, or muscle disease.[3] In the standing horse, the veterinarian can evaluate the cervicofacial/auricular and cutaneous trunci reflexes as well as tail tone, perineal, and anal tone reflexes.[9] The veterinarian should closely observe the horse during the dynamic exam for subtleties that might indicate proprioceptive deficits. Evaluation of stops and starts from the walk, transitions from the walk to trot and trot to the walk, tight turns, and backing the horse can help detect subtle neurologic deficits. The goal is to determine if the horse travels symmetrically, smoothly, and with appropriate stride length and foot flight, if the horse appears strong and consistently places the feet appropriately, and if the horse moves in a balanced and coordinated fashion.[3] It can be difficult to differentiate whether asymmetry and weakness are due to musculoskeletal pain and weakness or neurologic deficits.[3] Horses with musculoskeletal pain tend to be consistently irregular, whereas horses displaying neurologic gait deficits tend to be much less consistent and make variable mistakes when positioning their limbs.[18]

Step Five: Diagnostic Testing

There are a number of factors to be considered when requesting or recommending additional diagnostic testing. Knowledgeable buyers often come to the PPE requesting specific ancillary testing regardless of the examination findings. Less knowledgeable buyers may be advised by their primary veterinarian, trainer, or friends as to what type of ancillary testing to request. The examining veterinarian will usually discuss with the prospective buyer ancillary testing that he/she recommends based on the findings of the basic examination. Their recommendations are often based on a number of factors, such as age, breed, intended purpose of the horse, and abnormalities that were identified during the PPE. The decision to perform the recommended testing is up to the prospective buyer. If the prospective buyer refuses additional testing, the conversation, decision, and reason for the refusal should be documented in the final report.

Following completion of the basic PPE, any additional testing that was recommended or requested in Step 1 or 2 can be performed. Depending on the veterinarian's capabilities and facilities, additional testing that is commonly performed may include upper airway endoscopy, musculoskeletal ultrasound, and echocardiogram. Advanced imaging can be considered if a specific finding is to be investigated further. In the future nuclear scintigraphy and magnetic resonance imaging (MRI) may become common practice in PPEs.[10,14,15] Obtaining blood for the purpose of drug screening should also be addressed with the prospective buyer and the blood acquired prior to administering any sedation. Ancillary testing procedures must be interpreted relative to the physical exam findings and relevant to the history of the animal being examined.

There are no required standards for imaging in the PPE. The recommendations for imaging are based on a number of factors including the signalment of the horse, past and present performance, previous medical history, intended purpose for the horse, results of the initial PPE, communication with the prospective buyer, and budgetary constraints.

Radiography is the most utilized imaging modality in the PPE.[8] It is common for a prospective buyer to come to the exam with a list of requests for imaging, regardless of the exam findings. If left up to the recommendations of the veterinarian, the question of if or what to radiograph is generally based on the same above criteria. If the initial examination fails to identify any abnormalities, radiographic evaluation is used to scan for preexisting or potential future issues. The most thorough evaluation would include complete studies of the front feet, bilateral carpi, tarsi, and stifles, all four fetlocks, and cervical and thoracolumbar spine. This number of imaging studies would add considerable expense to the exam, and the veterinarian must keep in the mind radiation safety issues.

A few examples of radiographic studies that might be recommended by a veterinarian assuming relatively normal physical examination and dynamic examination are given below:

- 14-year-old Quarter horse gelding: Bilateral front feet navicular series, bilateral tarsi, and bilateral stifles
- 4-year-old Warmblood mare: All four fetlocks, bilateral tarsi, and bilateral stifles
- Young off-the-track Thoroughbred: Bilateral carpi, all four fetlocks

Recommendations for cervical spine and thoracolumbar radiographs should be discussed with the buyer if evidence of back pain and reduced range of motion in the neck are discovered during the examination particularly in jumpers and dressage horses.[14,15] Shoes should be removed if radiographs of the feet are to be taken preferably by someone that is skilled in properly removing horseshoes or by a farrier. Regardless of who removes the shoes obtaining the owner's signed consent is necessary, and if consent to remove the shoes is refused, a refusal statement should be signed and included in the report.[25]

All radiographs should be of high quality, well positioned, and labeled correctly. Including poor-quality radiographs in a PPE is inappropriate and a potential source of litigation.[13] If the radiographic studies are performed by technical personnel, the veterinarian should approve each image before including them and signing off on the final PPE report.

Abnormalities are commonly identified on PPE radiographs that do not correlate strongly with potential lameness. This can make interpretation and reporting difficult in the final report. In this instance, if the veterinarian is not comfortable interpreting the findings as it relates to continued soundness or potential unsoundness, seeking a second opinion is advised. The relative importance of any radiographic finding that is considered abnormal, or not

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within normal limits, must be put into perspective based upon clinical findings in order to place appropriate relevance on the finding. For example, a study that correlated radiographic findings and the presence of lameness in PPEs found 85.5%, 70.0%, 37.5%, and 22.7% of horses with grade 0–3 navicular bones, respectively, to be sound.[8] It is important to communicate to the prospective buyer that these findings can be identified in the absence of lameness. In contrast, it is important to communicate and document that the absence of radiographic abnormalities does not guarantee the absence of future lameness. These situations support the importance of obtaining the horse's past medical history and records, including previous imaging, for comparison. If previous radiographs are available, comparing studies can help determine the significance of an abnormal finding.[14] In cases where there are concerns with abnormalities identified on the physical examination or radiographs and the prospective buyer wishes to continue with the PPE, the veterinarian can recommend additional imaging (ultrasound, nuclear scintigraphy, MRI, etc.) to further evaluate the findings. Permission from the owner must be obtained before performing advanced imaging.

Ultrasound is used commonly in PPEs either as a request of the prospective buyer or for evaluation of an abnormal finding identified on the examination. Ultrasound is a necessary skill for the equine practitioner to have but a skill that is difficult to perfect. Having good-quality equipment capable of producing high-quality musculoskeletal images is also important. If the veterinarian is not comfortable performing or interpreting an ultrasound study, referral to a skilled ultrasonographer should be offered to the prospective buyer.

Because of the large variation in ultrasonographic appearance between horses and even within the same horse, ultrasound is probably best used for investigating areas of concern identified in the PPE or follow-up in horses with a history of having soft tissue injuries.[14] The metacarpal and metatarsal regions are the most common areas to have ultrasound performed in the PPE. It is common in the aged athlete to find subtle or chronic changes in, for instance, the proximal suspensory, in an otherwise sound horse. In a horse that has joint effusion and no radiographic abnormalities, ultrasound can be used to evaluate the surrounding soft tissues. Subtle bony changes may also be evaluated more effectively with ultrasound vs. radiographs. Similar to abnormal radiographs, these findings must be discussed with the prospective buyer along with their significance to future soundness or lameness. The reader is referred to the chapter on performing and interpreting ultrasound.

Nuclear scintigraphy may be considered when further investigation of a particular finding is of concern to the potential buyer(s). It is usually not useful as a screening tool. Depending on a horse's level of activity, scintigraphy commonly results in a number of areas of increased radiopharmaceutical uptake that are not associated with pathology or lameness.[14] This can cause confusion in communication with the prospective buyer, difficulty in interpretation and reporting, and potential termination of an otherwise good purchase. Examples for indications for recommending scintigraphy might include further investigation of radiographic abnormalities in the feet, or neck and back pain.[14,15]

Although the gold standard of equine imaging, MRI should not be used as a scanning tool in the PPE. As with nuclear scintigraphy, abnormal MRI findings, in a sound horse, can be potentially confusing to communicate to a prospective buyer. However, MRI for the purpose of investigating lameness gives the veterinarian much more detailed information to communicate the potential risk to the prospective buyer.[10] The limitations of MRI are its narrow area of focus, making it inadequate as a screening imaging modality, and, if standing MRI is not available, the need for general anesthesia. MRI is useful for investigation once an area of interest has been isolated.[14,15]

Step Six: Interpretation of Findings and Reporting

Once the examination is completed, the veterinarian is tasked with interpreting their findings and communicating the findings to the prospective buyer in a well-organized understandable format, both verbally and written.[1,2,11,13,15,16,21,23–26] The initial verbal report should be between the veterinarian and prospective buyer directly or via the phone. If an agent represents the prospective buyer, the communication is directed toward the agent with the understanding that the prospective buyer will receive a copy of the written report. With permission from the prospective buyer, findings of the PPE can be discussed with the seller.[15,16,24–26]

There are numerous recommendation and examples of what the final PPE report should contain and what the format should be. The AAEP has established guidelines to help the examining veterinarian formulate a report that is complete, concise, and accurate (Table 12.4). These are available at https://aaep.org/guidelines/reporting-purchase-examinations. While compliance with all of these guidelines helps to ensure a properly reported purchase examination, it remains the sole responsibility of the veterinarian to determine the extent and depth of each examination. The AAEP recognizes that for practical reasons, not all examinations permit or require veterinarians to adhere to each of these guidelines (Table 12.4).

SUMMARY

The PPE exam is the most complete comprehensive examination that the veterinarian will be requested to perform. The information gained from the PPE will be used to aid a prospective buyer in making a purchase decision. Obtaining the information (the PPE) and delivering that information in a way that the client understands and can use to make an informed decision is the sole responsibility of the veterinarian. The veterinarian should be well versed in performing and documenting PPEs and be aware of the potential risks. It is the veterinarian's responsibility to determine the horse's overall health and serviceability to perform the prospective buyer's intended purpose for the horse. The veterinarian should not hold any responsibility for determining the horse's suitability for the prospective buyer.

The key to avoiding an unpleasant and potentially litigious PPE is clear and open communication between all parties involved in the transaction. The owner is responsible for willingly supplying any and all performance and medical history. The prospective buyer is responsible for clearly communicating their goals of the PPE, their

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Table 12.4. AAEP guidelines for prepurchase report.

- All reports should be included in the medical record.
- The report should contain:
  - A description of the horse with sufficient specificity to fully identify it.
  - The time, date and place of the examination.
- The veterinarian should list all abnormal or undesirable findings discovered during the examination and give his or her qualified opinions as to the functional effect of these findings.
- The veterinarian should make no determination and express no opinions as to the suitability of the animal for the purpose intended. This issue is a business judgment that is solely the responsibility of the buyer that he or she should make on the basis of a variety of factors, only one of which is the report provided by the veterinarian.
- The veterinarian should record and retain in the medical record a description of all the procedures performed in connection with the purchase examination, but the examination procedures need not be listed in detail in the report.
- The veterinarian should qualify any finding and opinions expressed to the buyer with specific references to tests that were recommended but not performed on the horse (X-rays, endoscopy, blood, drug, EKG, rectal, nerve blocks, laboratory studies, etc.) at the request of the person for whom the examination was performed.
- The veterinarian should record and retain the name and address of parties involved with the examination (buyer, seller, agent, witness, etc.).
- A copy of the report and copies of all documents relevant to the examination should be retained by the veterinarian for a period of years not less than the statute of limitations applicable for the state in which the service was rendered. Local legal counsel can provide advice as to the appropriate period of retention.

The following is a list of recommended information that should also be included in the final PPE report in addition to the AAEP's guidelines[16,23,25]:

- List of all medical records and pertinent information received prior to, or at the time of the examination, and if known, a list of individuals given access to the information. Any refusal to supply medical records or pertinent information should be clearly documented along with the reason for refusal.
- Include a brief summary and risk assessment of any relevant past medical history.
- Brief synopsis of pertinent verbal and written communications that occurred during the examination.
- The horse should not "Pass" or "Fail" the PPE. When stating PPE findings using phrases such as "in my opinion," "I cannot predict the future," and "I do not know" should be used when appropriate.
- If a second opinion is obtained to clarify unfamiliar or debatable exam findings that opinion should be solicited from an expert in the area in question, and the second opinion report documented in writing and included in the report.
- Any signed consent forms or statements.

Also available at https://aaep.org/guidelines/reporting-purchase-examinations.

intended purpose for the horse, any limitations they might have, and any misunderstanding they might have with any aspect of the PPE. The veterinarian is responsible for clearly outlining the goals, structure, and limitations of the PPE, discussing all available ancillary testing and the indications for their use, outlining the cost of services, and generating the final report. It is important that all parties are willing to give full disclosure of any conflicts of interest prior to initiating the PPE.

Performing the exam under the ideal conditions as dictated by the veterinarian, in the appropriate settings, and using the right equipment will help reduce the risk of performing an inadequate and incomplete PPE. Performing a PPE at the local performance arena, in the middle of a thunderstorm, to accommodate the trainer's hectic schedule can result in inaccurate assessment of lameness and misinterpretation of radiographic findings. Having the knowledge and experience to perform a thorough PPE and interpret and report the results accurately will help reduce the risk of a poor uninformed purchase decision and future complications for the veterinarian. Reporting that a horse was healthy and serviceably sound only to find out later that squamous cell carcinoma of the penis was identified following purchase and stating that a horse was suitable for the intended purpose only to find out later that the horse refused a jump due to a lack of training, injuring the new owner, are situations that can be avoided by properly and carefully performing and documenting a PPE.

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STANCE AND GAIT ANOMALIES CAUSED BY NEUROLOGICAL DISEASE

Lutz S. Goehring

Neurological disease in horses is actually quite rare, particularly if we compare the frequency of occurrence to lower airway disease, lameness, or colic prevalence in the general horse population. Neurological disease results from dysfunctional signaling along neurons and/or of synapses in the central and peripheral nervous system (CNS and PNS, respectively). As the nervous system of the horse stands for interaction with its environment, major tasks of CNS and PNS are the control of body position, stance, and motion through their combined control of muscle activity. Therefore, many nervous system diseases will have significant influences on a horse's stance and gait. For the less experienced owner, any gait anomaly is rather an orthopedic lameness with a suspected pain component than a neurological problem. This is why the veterinarian needs to pay detailed attention to the presenting complaint by careful examination. The common denominator of a neurological gait is unpredictability of limb placement. A neurological gait is typically irregularly irregular, which makes it different of the repeatable, regularly irregular lameness.

From a neuroanatomical standpoint, neurological gait anomalies can be caused by diseases that affect the CNS and/or PNS, either rostral or caudal of the foramen magnum. Lesions in brain areas and therefore rostral to the foramen magnum can be within the vestibular system, the cerebellum, and/or the forebrain/basal nuclei/motor cortex. Lesions in the spinal cord, its segmental spinal nerves, and neuromuscular junctions, however, are causing most of the neurological gait anomalies in horses. In general, a lesion or lesions interfere with electrical or chemical signal transmission along nerve cells and their synapses, while a successful signal transmission is necessary to initiate, activate, fine-tune, terminate, and provide feedback to various nervous system structures for any coordinated activity. Depending on the noxious cause, it will result in single focal, multifocal, or diffuse lesions. Multifocal lesions can be regional and contained to a specific structure, or they can be disseminated. Only immune-mediated disease or specific toxins will affect a distinct structure, cell type, or cellular mechanism, resulting in a distinct deficit (e.g. toxins of *Clostridium botulinum* or *Clostridium tetani*).

For the veterinarian it is usually easy to recognize moderate to severe neurological gait anomalies. Minor gait changes are more difficult to be distinguished from lameness. Furthermore, neurological and musculoskeletal conditions may coexist independently or share a common pathophysiological background, and both conditions will contribute to an abnormal gait while confounding the diagnosis. Osteochondrosis is a perfect example of gait problems resulting from both musculoskeletal and neurological causes. It can lead to limb lameness through joint problems, while facet joint (osteo)chondropathy of the cervical vertebrae can result in compressive cervical spinal cord disease. See the section on cervical vertebral stenosis later in this chapter.

NEUROLOGICAL GAIT AND (NEUROANATOMICAL) LESION LOCATION

The first question veterinarians should ask themselves when a horse presents with suspected neurological gait anomalies is: "Is what we observe caused by neurological disease?" To make a certain diagnosis of neurological disease, it is important to take a thorough history and conduct physical and neurological exams, each and every time. Furthermore, a neurological examination should always be complete, including an assessment of the patient's level of consciousness, cranial nerve function testing, and brief examination of both eyes, and observe eating, drinking, and, ideally, defecation/urination prior to a gait evaluation.[1,10] A neurological gait is the result of upper (UMN) and/or lower (LMN) motor nerve malfunctioning, either directly or indirectly, and can be described in terms of three distinct features of anomaly: ataxia, dysmetria, and weakness. The neurological gait that is observed may be the result of a single, a combination of two, or all three descriptors. Each limb must be observed and evaluated for presence of these findings and graded using a predetermined scale.[5]

Ataxia—The word is derived from the Greek word for indiscipline or "lack of order." An ataxic gait is irregular and lacks predictability when it comes to limb protrusion and placement; it is best described as being "irregularly irregular." Ataxia results from a decreased perception of proprioceptive information ascending from the peripheral sensors, Golgi (tendon) organs in all limbs, trunk, and neck to the cerebellum. The cerebellum receives proprioceptive information also from the vestibular organ and vestibular nucleus in the brain stem. As a result of proprioceptive disruption, the horse can no longer correctly perceive its position, posture, or equilibrium, which leads to mal-interpretation in the (fore)brain/basal nuclei, resulting in delays/absence of movement initiation, maintenance, and control through upper motor neuron (UMN) tracts. Thus, ataxia is typically combined with dysmetria (see below), and ataxia is noticed in a horse if one of the following four components of the nervous system is affected: (1) spinal cord; (2) vestibular organ, vestibular nerve, or nucleus; (3) cerebellum; and (4) fore brain. Each component adds specific detail to the clinical presentation. Vestibular ataxia is usually accompanied by a head tilt and asymmetrical ataxia. Horses with one-sided vestibular ataxia show a head tilt toward the side of the lesion. These horses typically bend their neck and trunk in a concave fashion and tend to circle toward that side. In addition, nystagmus may be present. Cerebellar ataxia is characterized by a

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symmetrical ataxia and hypermetria in all four limbs, but without loss of strength. It is routinely accompanied by an intention tremor of the head, a base-wide stance, and absence of a menace response. Forebrain ataxia is accompanied by abnormal mentation and, sometimes, cranial nerve deficits. Spinal ataxia is the most frequently encountered form of ataxia in the horse. Its cause is a disruption of ascending proprioceptive flow of information toward the cerebellum. As a result, descending information cannot appropriately be fine-tuned, which influences initiation/maintenance/termination of motion through muscle tone and motor function in neck, trunk, or limbs. Alternatively, as the descending motor pathways are most likely damaged by the same processes as the ascending pathways, ataxia and dysmetria occur simultaneously. If fore- and hindlimbs are affected, this is either caused by a focal cervical or by multifocal lesions throughout the spinal cord.[1,2,5,10]

Dysmetria—Dysmetria collectively describes a state of rigidity, spasticity, and decreased or exuberant limb flexion. It is associated with UMN conduction disruption in the spinal cord, interneuron circuits, or cerebellar disease. UMN tracts have two major functions: some are involved in the initiation of movement, whereas others have an inhibitory effect on a peripheral reflex loop (e.g. a patellar reflex). Dysmetria is caused by a reduced inhibition of the peripheral reflex loop, and the uninhibited reflex is observed. A hypermetric gait is characterized by an increased range of motion and excessive joint flexion. A hypometric gait is typified by limb stiffness and reduced joint flexion, particularly of the tarsal and carpal joints.

Weakness or Paresis/Paralysis—Weakness or paresis is the decreased ability to initiate gait, maintain posture, or resist gravity. Paralysis is the inability to do all three. Paresis can be further divided into UMN and lower motor neuron (LMN) paresis. UMN paresis, or flexor weakness, presents with a delay in initiating movement, followed by longer and, typically, lower stride. LMN paresis is usually observed as an antigravity or flexor weakness, presenting as short-strided, poor swing-phase gait; trembling due to muscle fatigue (muscle fasciculations); and lowered neck carriage while standing. Muscle atrophy is more pronounced and often more localized in LMN than in UMN paresis; for both forms it may take 2–3 weeks before the muscle atrophy becomes noticeable. Toe dragging and abnormal hoof wear can be seen with either form of paresis. Weakness in all four limbs, but no ataxia or dysmetria, indicates diffusely affected neuromuscular units and is by definition an LMN weakness.[1,2,5,10]

FINDINGS FROM A NEUROLOGICAL EXAM THAT CAN HELP IN LOCALIZING A LESION

Level of Consciousness and Body Position

The normal horse is quiet or bright, alert, and responsive (QAR or BAR); it pays attention to its surroundings, recognizes visitors, and finds water and food. A horse puts weight on all four limbs, which are vertically placed underneath the body (aka the columns of a Greek temple). The exception to this is a standing horse with a locked stifle. In a QAR or BAR horse, the poll of the head should be above the level of the withers. The normal recumbent/resting horse is usually found in a sternal position or in a flat lateral position. Minimal disturbance is typically sufficient for arousal and getting up. A horse extends its forelimbs first, followed by an abrupt extension and lift-off with both hindlimbs. An abnormal horse is either stuporous or (semi)comatose; wanders in circles within its confined space; may appear blind, agitated, or violent; or has a seizure. All clinical signs relate to a problem rostral to the foramen magnum. An abnormal stance can be wide based (sawhorse stance) or narrow based (horse-on-a-ball or goat-on-a-rock stance). The sawhorse stance is typical for a horse with a cerebellar lesion, whereas the horse-on-a-ball stance is seen with diseases collectively affecting the LMN. Whenever a horse places its limbs randomly, once it comes to a standstill (hence, an asymmetrical limb placement), this may be a sign for a lack of ascending proprioceptive information or interpretation in the forebrain. A conscious horse that cannot rise from recumbency is probably affected by profound weakness, which can be (focal/multifocal) UMN or (diffuse) LMN weakness. Whether the horse can or cannot lift its neck from the ground, maintain a sternal position, or sits like a dog is a critical observation in determining where the lesion of the problem is located—the cranial or caudal neck, thoracolumbar spinal cord, multifocal, or diffusely involving gray and white matter of the spinal cord. Horses with a head tilt and vestibular ataxia often stand slightly wide based, and the body may be concavely bent toward the side of the lesion. If recumbent, a horse with a head tilt is more comfortable while lying on the affected side. These clinical signs worsen when the horse is blindfolded.

Head and Body Symmetry

The normal horse shows symmetry in the head position and the posture of the neck, limbs, and trunk. Symmetry also extends to facial expression and the carriage of the tail. A head tilt (a sign of central or peripheral vestibular damage), a dropped ear, and paralysis of facial muscles (facial nerve paralysis) are examples of head asymmetry. Regional muscle atrophy or complete unilateral atrophy is more likely a sign of UMN paresis. Defined muscle atrophy of a single muscle belly is best explained by peripheral nerve damage. However, generalized symmetrical muscle atrophy without signs of ataxia or dysmetria is the result of diffuse LMN disease if neurological and needs to be distinguished from a horse with chronic malnutrition.[1,2,5,10]

Manipulations in the Standing Horse

The normal horse is able to extend and flex its head and neck dorsally and ventrally. During lateral flexion of the head and neck, the horse's muzzle should touch its ribs at either side. Some horses with cervical vertebral stenotic myelopathy (CVSM) may be reluctant or resent lateral flexion due to pain in the intervertebral (facet) joints. Both sides of the horse's neck and back musculature, from front to back, should be probed with a blunt instrument such as the handle of a neurology hammer or

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a ballpoint pen. Mild stimulation of the chest and abdomen elicits a cutaneous trunci muscle response, but not in all horses. A two-pinch technique with forceps is used to elicit this local reflex first, and then a pain response may be noticed with a deeper pinch.

The dorsal processes of the thoracic and lumbar vertebrae must be palpated and (gently) percussed. No pain response or crepitus should be observed or palpated. With both hands placed over the withers, the horse is pushed away and pulled toward the examiner. A normal horse quickly activates the extensor or antigravity muscles in the forelimbs to maintain a solid stance. An abnormal horse can easily be pushed away or pulled toward the examiner because of LMN weakness or a lack of proprioception.

Hopping can be done by picking up one forelimb at a time; however, caution is advised in the obviously severely affected horse. The examiner faces the same direction as the horse, holds the lifted forelimb at the pasterns, and pushes the horse away with his/her shoulder. The normal horse will hop one jump at a time with a nice lift-off and landing. The abnormal horse will have a delayed lift-off and a short, rigid jump followed by a clumsy landing. "Hopping" allows to test several aspects of the nervous system: muscular strength, proprioception, and dysmetria.

Stimulating the axial musculature of the back usually elicits a ventrodorsal movement of the vertebral column, lordosis followed by kyphosis. The abnormal horse shows scoliosis or it appears stiff in the back. A normal horse has at least some degree of a tail tone and a perineal reflex. A completely flaccid tail is a sign of LMN weakness. A tail should not continuously be held off the body. This can be a clinical sign of tetanus or tetany, if the horse is unable to clamp its tail upon stimulation of the perineum.

The (standing) tail pull is a very important test during a neurological examination in the standing horse (Figure 12.27). It can help to distinguish between UMN and LMN weakness. The square standing horse is gradually pulled at its tail laterally until the examiner notices a contraction of the quadriceps muscle. The horse will be pulled fairly easily toward the examiner if there is lumbar LMN dysfunction.

A normal horse walks with a regular four-beat movement. Limb placement should be graceful and regular, and a solid surface allows the examiner to listen to hoof placement. The hindlimbs follow the forelimbs, and the hindlimbs typically overreach the imprints of the forelimbs.[10]

Key abnormalities in the neurological horse are revealed in unpredictable limb placement, a shortened stride length, toe dragging, pacing, and generalized rigidity or stiffness resulting in a truncal sway. All of these signs can result from ataxia, weakness, or dysmetria. These may be mild and unobservable during a straight-line exam when walking the horse on a flat hard surface. In such cases the signs can be exacerbated by circling or spinning the horse, backing it up for several steps, and walking it, with and without head elevation, up and down a slope. A horse with a neurologically affected gait may exhibit circumduction (an exaggerated movement of the outer hindlimb) or pivoting (planting a forefoot and turning around without lifting it) during spinning. During backing, the limbs of a normal horse should move diagonally, with simultaneous placement of the contralateral fore- and hindlimbs. During uphill and downhill walking, a normal horse places its limbs solidly and securely, without knuckling in the fetlocks or carpal/tarsal joints. Insecure placement of limbs is a sign of ataxia. Particularly when walking downhill, simultaneous elevation of the head exacerbates insecurity with limb placement. Neurologically abnormal horses also should be examined during a trot and, if not too severely affected, during canter. A good test for proprioception is to trot a horse and bring it to a sudden stop. A smooth collection of all limbs underneath the body will be observed in a normal horse.[1,2,5,10]

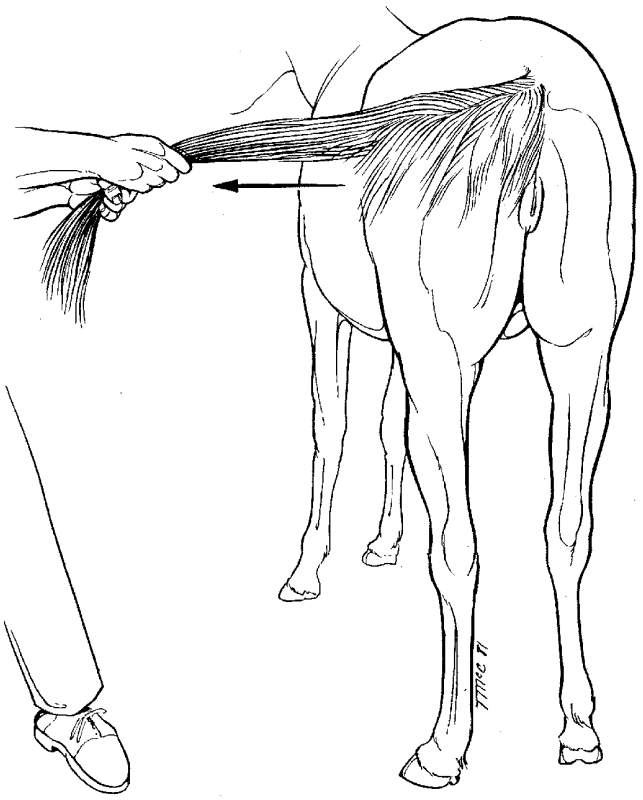


Figure 12.27. Tail pull (arrow) in a standing horse tests the strength of the quadriceps muscle and the reflex activity of the femoral nerve as a part of the lower motor neuron system. A gradually increasing pull on the tail toward the examiner should result in a quadriceps muscle contraction, and the hindquarter of the horse should not move significantly. For example, a horse with a cervical spinal cord lesion showing a grade 3 out of 4 ataxia of the hindlimbs still has a strong response to the standing tail pull. Pulling on this horse's tail during a walk will show incoordination or ataxia.

PITFALLS OF NEUROLOGICAL EXAMINATION

The clinical presentations discussed above suggest neurological damage that affects the gait. However, the signs also can arise from illnesses or problems unrelated to the nervous system. These should be considered when making the diagnosis, and possible explanations for suspect neurological findings are listed in Table 12.5.

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Table 12.5. Alternative explanations for presentations with a suspected neurological presentation.

Clinical finding	Alternative, non-nervous system-associated explanations
Generalized muscle atrophy	Starvation, emaciation, (intestinal) nutrient absorption disorder, paraneoplastic syndrome, chronic myopathies
Regional muscle atrophy	Lameness-associated disuse
Low head–neck carriage	Nuchal ligament desmitis or rupture, exhaustion, emaciation/starvation, fever, use of sedatives
Ataxia	Fever, sedation
Stiffness, short strided	Lameness, myopathy
Hindlimb hypermetria	Laminitis, *Chorioptes* spp. infestation, fibrotic myopathy
Recumbency	Exhaustion, colic, rhabdomyolysis, pain, laminitis

SELECT NEUROLOGICAL SYNDROMES AFFECTING THE GAIT

Cervical Vertebral Stenotic Myelopathy (CVSM)

CVSM is probably the most frequent cause of neurological gait abnormalities in performance horses. In general, CVSM is a condition of compression of the cervical spinal cord by malformed cervical vertebrae. This focal compression has its greatest impact on white matter proprioceptive and UMN tracts, resulting in neurologic deficits. Hallmark findings are generally a symmetrical ataxia and dysmetria in front limb and hindlimb. The layperson terminology refers to this condition as "wobbler," which should be avoided, as a "wobbler" could be any ataxic horse with numerous causes responsible for its clinical presentation. Cervical spinal cord compression leading to CVSM is a frequent cause of sudden ataxia in the tall breeds (Thoroughbred, Warmblood, etc.) and is typically recognized either in the young, fast-growing horse (6 months to 2–3 years of age) or in the middle-aged performance horse. Differential diagnoses include trauma (to the neck), NAD/EDM, EPM (Americas), and rarely neoplasia (lymphoma, melanoma). In the young horse, spinal cord compression can be a dynamic or static condition. Dynamic compression occurs due to vertebral instability and usually affects intervertebral sites C3–C4 and C4–C5. Dynamic compression is usually seen in younger horses associated with developmental orthopedic disease and osteochondrosis at the intervertebral facet joints. Mal-articulation of the facet joints can cause abnormal movement between two vertebrae resulting in spinal cord compression. Static compression causes continuous spinal cord compression usually due to vertebral malformation associated with osteoarthritis and bony remodeling that affects the more caudal cervical region. Both causes of compression can occur in the same animal, although static compression is more likely in the middle-aged performance horse. The diagnosis is usually made based on clinical findings together with cervical radiographic and myelographic confirmation of the site(s) of spinal cord compression. Advanced imaging (CT, contrast CT, and MRI) may be used instead of a myelogram depending on the suspected lesion location in the neck and the specific capabilities of the advanced imaging unit. Treatment options include conservative management with anti-inflammatories, exercise restriction, and dietary changes or surgical intervention to either remove the source of compression (dorsal laminectomy) or to stabilize the area(s) of spinal instability/compression. In general, surgery is the most widely reported treatment for CVSM and is often indicated to prevent repetitive trauma to the spinal cord. Excellent and more extensive reviews of CVSM can be found in these references.[7,8,12]

Botulism

Botulism is a feed-borne intoxication associated with *C. botulinum* toxin. If several horses are on a property, botulism usually manifests as a herd outbreak. Botulism toxin blocks the acetylcholine release at presynaptic neuromuscular junctions, postganglionic parasympathetic nerve endings, and peripheral ganglia. Posture and gait deficits are noted initially, with muscle fasciculations and a short-strided gait, followed by recumbency. Hallmark findings of botulism are generalized weakness, resulting in an abnormal gait and recumbency, and cranial (motor) nerve dysfunction (tongue tone, mastication, swallowing). Excellent reviews of botulism are available in other texts.[4,14]

Equine Motor Neuron Disease (EMND)

Equine motor neuron disease (EMND) is a rare, slowly progressive disease that usually affects only a single animal in a herd. Although its etiology is unknown, lack of antioxidants, particularly vitamin E, has been associated with its development. EMND causes a gradual and progressive loss of LMN of the entire spinal cord, leading to signs of symmetrical neurogenic muscle atrophy and weakness without ataxia. Horses with EMND are described as having a loss of muscle mass in combination with a good appetite, and cranial nerves are not affected.[3,11]

Equine Protozoal Myeloencephalitis (EPM)

Equine protozoal myeloencephalitis (EPM) is a disease of the Americas, most commonly caused by a protozoan organism, *Sarcocystis neurona* (and to a lesser degree by *Neospora hughesi*). The geographical restriction of *S. neurona* is linked to the habitat of its definitive host, the American opossum (*Didelphis virginiana*). *S. neurona* oocysts are produced in the opossums' gastrointestinal tract. After horses ingest the oocysts through contaminated feed, sporulation occurs, and asexual

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replication of *S. neurona* merozoites may follow. In a small but poorly defined number of horses, the protozoan enters the central nervous system, where replication can be prolonged and progressive, leading to multifocal disease of the brain and spinal cord. The most common presentation of EPM is asymmetrical gait anomaly, with a gradual worsening over weeks to months. Cranial nerve abnormalities are also common. EPM is well reviewed in several textbooks.[1,5,15]

West Nile Virus and Eastern/Western/Venezuelan Encephalitis Viruses

The implementation of a good vaccination program against West Nile virus and eastern, western, and Venezuelan encephalitis viruses means that disease should only be expected in poorly or nonvaccinated horses. Hallmark clinical findings of these viruses are active muscle fasciculations, tremors, and hyperesthesia. Cortex or general brain involvement appears to be more common with eastern, western, and Venezuelan encephalitis viruses, whereas the spinal cord and basal ganglia are frequent targets of West Nile virus.[9]

Equine Herpesvirus Type 1-Associated Myeloencephalopathy (EHM)

EHM is a rare syndrome that can follow infection with equine herpesvirus type 1. It causes multifocal ischemic damage, primarily to the spinal cord, usually in adult horses of the tall breeds. Only horses that develop a cell-associated viremia during infection are at risk of EHM. The clinical presentation varies with the number of ischemic infarcts. A low to moderate number of infarcts results in asymmetrical ataxia and both upper and lower motor weakness; a high number leads to coalescing disease with profound weakness, often resulting in tetraparalysis.[9,13]

Ryegrass Staggers

The clinical signs of ryegrass staggers present shortly after ingestion of a preformed toxin from an endophyte closely associated with perennial ryegrass (*Lolium perenne*), although a few other grasses are associated with a similar syndrome. The endophyte (*Neotyphodium lolii*) is in a symbiotic, lifelong relationship with the grass plant and may produce significant amounts of the toxin, lolitrem B, under specific growth-related and environmental conditions. The syndrome has a regional occurrence, reflecting its close association with (infected) perennial ryegrass occurrence and feeding. Lolitrem B affects the cerebellum and the inhibitory (inter)neurons in the spinal cord. The clinical signs are very typical: tremors, base-wide stance, oscillating eyeball, and a staggering, hypermetric gait. The signs worsen with exercise and persist as long as there is further toxin intake. Once toxin-free feed is provided, there is usually a fast recovery and return to full function.[6]

Stringhalt and Shivers

Although recognized as neuropathies, the etiology for and location of a neurological lesion for both stringhalt and shivers remain poorly understood. A hyperreflexia seems to be involved in both syndromes, and interneuronal disease would be most plausible. In stringhalt is a hyperreflexia of the lateral digital extensor muscle of the hindlimbs, typically hyperflexing one or both hindlimbs during a walk. Stringhalt is typically less prominent or absent at the trot or canter. Stringhalt is differentiated as a plant-associated form involving a toxin, in which case several horses sharing pasture may become affected simultaneously, and an idiopathic sporadic, single-animal form. A peripheral axonopathy may be found in combination with a neurogenic myopathy with the plant-associated form of stringhalt. Shivers, more common in draft and Warmblood breeds, is a hyperreflexia of the flexor muscles of the pelvic limbs, which becomes most noticeable when the horse is backed. Rarely, we see the movement pattern at the forelimbs. The affected hindlimb, often in combination with the tail base, shows jerky, trembling movements that are due to cyclic contraction followed by relaxation of the involved musculature.[5]

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